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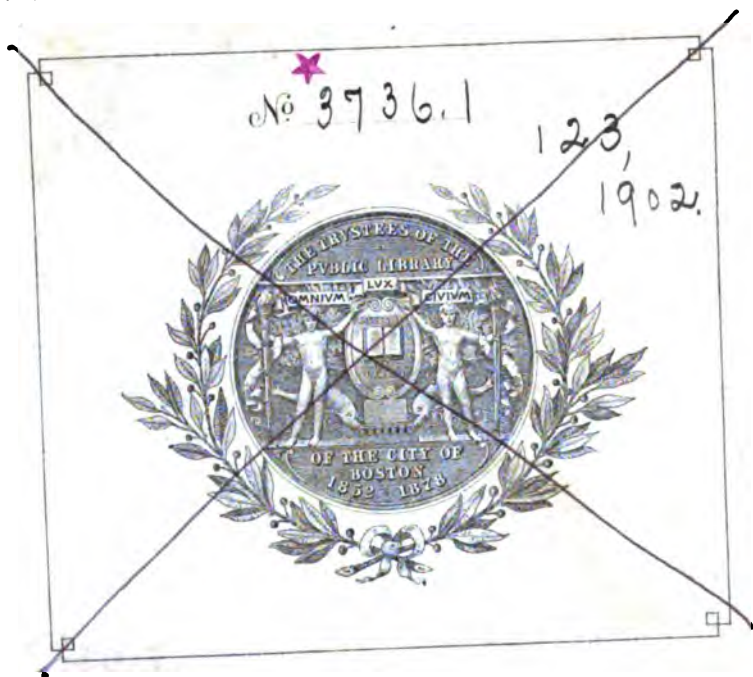
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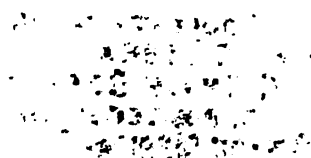
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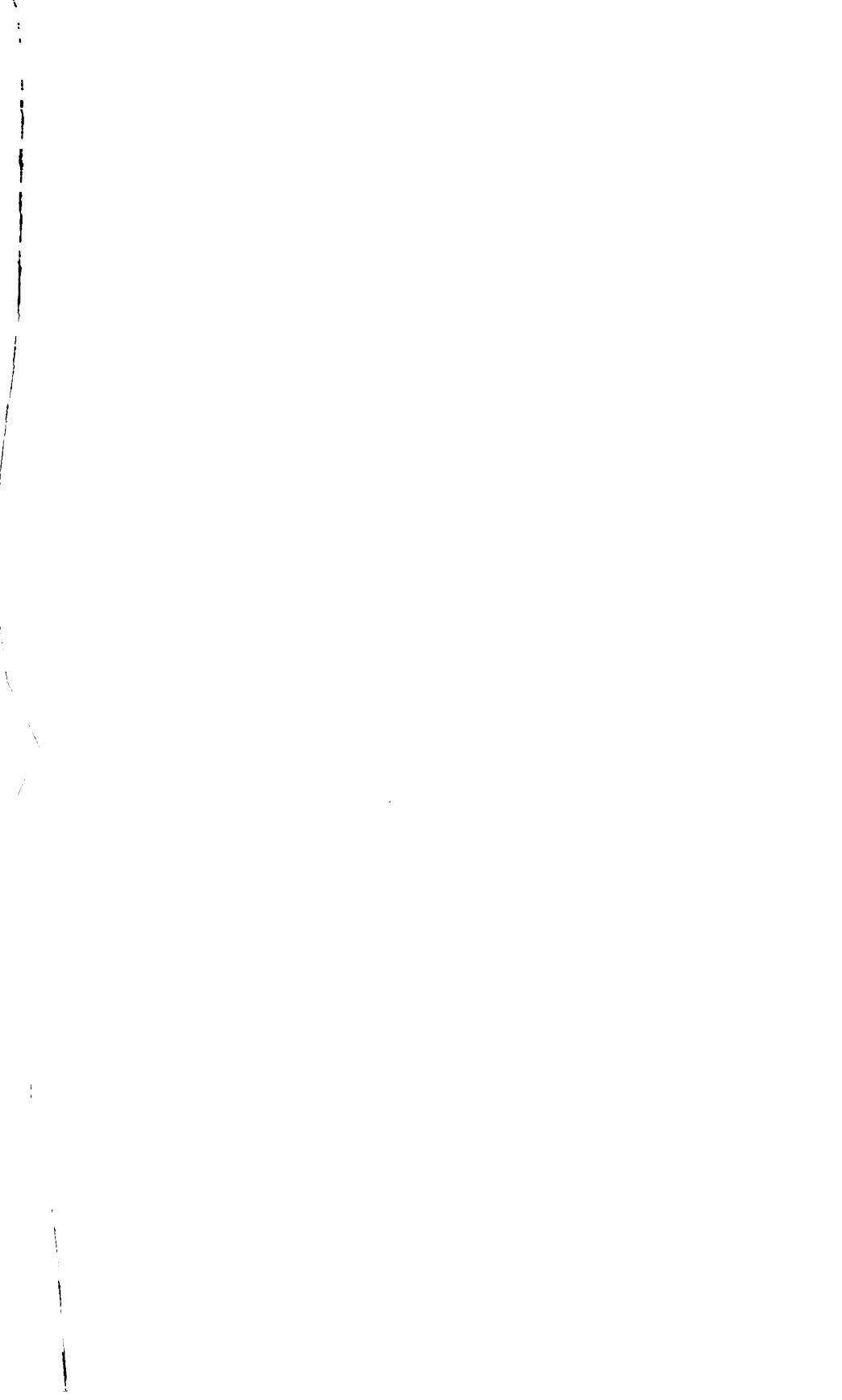
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JANUARY, 1902.

CASES ILLUSTRATING URETERAL SURGERY.

BY HENRY C. COE, M.D.,
OF NEW YORK.

CASE I.—Miss M. L., aged twenty-six years, entered the General Memorial Hospital with a uterine fibromyoma extending as high as the umbilicus. The tumor had grown rapidly during the previous six months, causing marked pressure symptoms. Operation, June 3, 1899. Both ovaries and tubes being normal were not removed. The tumor extended between the folds of the left broad ligament, displacing the uterine artery and ureter to such a degree that both were ligated and divided together before the accident was discovered. The proximal end of the ureter was freed from the ligature and was identified by the escape of urine. It was surrounded by gauze, the entire uterus was removed, and an examination of the ureter was made with a view to immediate repair of the injury. As the ends could be easily approximated, I decided to perform uretero-ureteral anastomosis by the lateral method advocated by Van Hook and Kelly—ligating the distal end and making a slit in the duct one quarter of an inch below the point of ligation, through which the upper end was drawn by traction sutures of fine silk. Seroserous sutures were introduced in the usual manner, and the united ends were covered with a flap of peritoneum. The peritoneal flaps were sutured and the subperitoneal space drained per vaginam. Time of operation about one hour. The convalescence was normal, with the exception of a rise in temperature on the third and nineteenth days (102° F.). The daily amount of urine varied from thirty to forty ounces until the sixth day, when a leakage was noted. This was neither constant nor considerable. The patient was kept in the hospital for six weeks, in the hope that the fistula would close. The vaginal wound healed so that the opening could not be identified. The dribbling occurred only when the patient was up and about, so that it was inferred that there was a small valve-like opening at the point of anastomosis, and not a complete separation of the ends of the ureter. Thirty ounces of urine were passed daily. I saw the patient at my

office a month later. She was in excellent health, and was not annoyed by the leakage, which was slight and did not occur at all during the night. She subsequently went abroad, and I have not seen her since, but her physician informed me a year after the operation that the fistula had entirely closed.

CASE II.—Mrs. R., aged fifty-nine years. Operation at the Memorial Hospital, June 17, 1900. Double intraligamentary dermoid cyst, with universal intestinal and intrapelvic adhesions. The uterus was so intimately attached to the growths that it was necessary to remove it. After freeing the cyst on the right side the uterine artery was ligated, the cervix divided, and the entire mass was reflected to the opposite side, in order to enucleate the growth in the left broad ligament. The left ureter was not identified until removal of the tumor, when it was found that it had been torn across. The stump of the cervix was removed and the ureter was examined. It was found that the distal end was so much dilated that the proximal end could easily be invaginated into it to the distance of an inch without danger of constriction. This was done, and the ends were secured with sero-serous sutures. It was impossible to secure a peritoneal flap, as the whole pelvis had been denuded of peritoneum. The repaired ureter now extended across the middle of the pelvic cavity without any support, so that the entire pelvis was packed with gauze, on which the ureter rested. While inserting the gauze, traction was inadvertently made upon the ureter and the ends pulled apart, so that it was necessary to do the work over again. Peritoneal flaps were secured with some difficulty, and the raw surface was shut off from the general cavity, with vaginal drainage. The patient was under ether an hour and a half and had considerable shock, but rallied quickly and made an uninterrupted recovery, passing a normal amount of urine from the outset. She was discharged four weeks after operation, and has since remained in good health.

CASE III.—Mrs. K., aged thirty-one years, was referred to me on account of a supposed cyst in the anterior vaginal wall. On examination I found a soft, doughy tumor, the size of a grape-fruit, occupying the vesico-uterine pouch, displacing the uterus backward and the bladder forward. It was easily palpated above the symphysis and extended downward almost to the meatus. The patient was quite anæmic, with a loud systolic bruit. Operation at the Memorial Hospital, March 7, 1900. An explorative incision was made into the supposed cyst, and a bloody fluid escaped. On enlarging the incision and introducing the finger an irregular cavity was felt, filled with soft, gelatinous masses, fully a handful of which were scooped out; there seemed to be no distinct capsule. The bleeding was so profuse that I was obliged to tampon the cavity and desist, as the patient became pulseless and was revived only with heroic treatment.

The microscopical diagnosis was myxo-angioma, with probably malignant degeneration (sarcoma).

Two weeks later the mass was only one-third its original size, and the patient was so much improved that I made a second attempt to remove it. A free incision was made over the growth, and it was enucleated by blunt dissection from above downward until the base of the bladder was reached, when it was dissected out with scissors. To my great chagrin, on examining the mass after removal I found attached to it a portion of the trigone, an inch square, and at least an

inch of the left ureter. I also found that the right ureter had been severed close to the bladder. Although the patient was again in collapse from loss of blood, so that rapid work was necessary, the wound in the bladder was closed with superficial and deep sutures, the end of the left ureter being turned into the bladder at a point where the tension would be least, and secured with deep and superficial sutures. The right ureter was much dilated and the tension such that I was unable to draw it down. The patient's condition was now so alarming that it seemed improbable that she would survive, so that the operation was suspended, the bed of the tumor being packed with gauze. She rallied quickly as before. A catheter was left *in situ*, and during the first week from thirty to thirty-five ounces of normal urine were discharged through it daily, with only occasional dribbling through the wound. Eventually most of the sutures gave way, and a large fistula two inches in diameter remained, involving the entire base of the bladder. I was unable to identify the end of either ureter. The patient left the hospital after six weeks, wearing a urinal.

She was in such a miserable condition that I really thought that it would have been better for her if she had not survived.

Two months later she re-entered the hospital, and I operated for repair of the fistula. It was found that the uretero-vesical anastomosis on the left side had been entirely successful. Fortunately, the end of the right ureter had become adherent at the edge of the fistula. A probe was introduced into it, and it was dissected off with the vesical mucosa, and thus turned into the bladder. The edges of the fistula were then split so that the everted mucosa could be rolled inward. The vaginal edge was denuded, the cervix uteri being utilized to fill in the upper part of the gap. Sutures were introduced in the usual manner, parallel with the long axis of the vagina, care being exercised not to include the end of the ureter. A catheter was left *in situ* for three days, and the patient subsequently passed her urine every four hours, retaining from four to six ounces. On the sixth day the nurse allowed her to go all night, so that twelve ounces accumulated in the bladder, with the inevitable result. A small fistula developed, which was easily closed a fortnight later.

The patient has since remained perfectly well, having no vesical symptoms whatever. She is a hard-working woman, and is in better health than she has been for years. When I examined her a month ago it was difficult to find the cicatrix.

Although my experience in ureteral surgery is too limited to permit any deductions with regard to operative technique, the cases reported seemed to be sufficiently instructive to justify me in briefly recording them. They at least serve to illustrate the fact that in complicated cases, especially with intraligamentary tumors, it is sometimes impossible to identify the displaced ureters before they are injured, and, therefore, that every suspicious cord, adhesion, or supposed bloodvessel should be carefully inspected before it is clamped or ligated, and again after it has been divided. When it is established beyond a doubt by the escape of urine that a ureter has been divided, nothing except the absolutely desperate condition of the patient should deter the sur-

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geon from at once attempting to repair the injury, instead of resorting to the unsurgical makeshift of suturing the end of the ureter in the abdominal wound, or to the serious procedure of removing a healthy kidney from a patient already depressed by a long and bloody operation.

The well-known difficulties attending secondary operations for the cure of ureteral fistulæ, even in the hands of experts, would seem to render it imperative that an attempt should invariably be made to secure immediate repair. As regards the method to be adopted in the individual case no fixed rules can be formulated. Each must be studied separately. The technique is now sufficiently familiar, at least theoretically, but the opportunities for its application are so rare that few surgeons have an opportunity to acquire such confidence and dexterity in dealing with this complication as with others which occur in connection with abdominal surgery.

CLINICAL HISTORY OF A CASE OF BLINDNESS FROM CONGENITAL DEFORMITY OF THE OCCIPUT.¹

BY CHARLES A. OLIVER, A.M., M.D.,
OF PHILADELPHIA.

ON October 20, 1899, through the courtesy of Mr. Frank R. Harrison, of East Liverpool, Ohio, a student in the third-year class of the Department of Medicine of the University of Pennsylvania, the writer was given the opportunity to study a case of blindness in a four-year-old girl.

The patient, the fourth of five children, was born at full term after an uncomplicated labor not requiring any aid. The first child was living and well. The second was stillborn at full term, the infant being badly macerated, and reported to have been dead for at least a month previous to its birth. The third, which was also a full-term child, lived but a few minutes. The fifth was born dead. In none of these labors except the first was there any physician in attendance. The mother, who was slightly undersized in height, had never been strong. The father, not a blood relation of his wife, died from renal disease some four years before the patient was studied. His habits had been bad, he having been in the habit of drinking at least three quarts of whiskey per week for years.

No family history of hereditary disease or congenital trouble could be elicited or determined, except that an aunt on the father's side was said to have died of tumor of the breast, which, from an incomplete history given, seemed most probably to be carcinomatous in character. The mother disclaimed the existence of any disease, though it was said

¹ Read before the October, 1901, meeting of the Section of Ophthalmology of the College of Physicians of Philadelphia.

that from time to time she exhibited a number of characteristic and well-pronounced hysterical stigmata, these being associated with slight rises of temperature, during which an elevation of a single degree was sufficient to produce an attack of delirium.

The mother stated that both of her children "had since birth slept with their eyes open," and that while sleeping a bloody froth from an unknown source often escaped from their mouths. She also asserted that the patient was born with a badly shaped head, and large, prominent eyes; but that the child could see some little until it was three years of age, when it became totally blind, and the protrusion of the eyeballs grew more pronounced. She had never noticed that the patient preferred to look to one side or to the other.

The only diseases that the patient had ever had were so-called "croup" and measles, these being diagnosed and treated by a competent physician. There was not any history of epilepsy or convulsions. The child's mental condition had always been good.

Careful physical examination failed to evidence anything wrong, except that the head was disproportionately small and quite deformed posteriorly, and that the eyes were prominent, divergent, and in constant motion. The occipital protuberance was almost wanting. The occipitoparietal suture was not well closed, the thickened serrated edges of the occipital and parietal bones curving outwardly. In front of the coronal suture the frontal prominence appeared, when laterally viewed, as if slightly raised into a dome-like elevation. The superciliary ridges were quite flattened. The orbital cavities seemed somewhat shallow, especially to their nasal sides. The root of the nose was broad, and the internal commissures of the eyes were too wide apart. Both the superior and the inferior dental arches, particularly the lower one, were shallow, while the chin showed a marked recession and apparent incompleteness of development. The nasal orifices were small. The mouth, which was almost constantly kept open, was employed for breathing purposes. Both pinna, as can be in measure seen in the reproductions of the photographs, showed a number of characteristic faults.

The eyelids, which were large and freely mobile, stretched across a pair of enlarged and prominent eyeballs that enjoyed free and full movement. The right eyeball was the larger and the more prominent. The palpebral fissures were very wide and abnormally long. Both globes had their antero-posterior axes directed outwardly, the right one being the more divergent. The eyes were in a state of constant rotary nystagmic movement. They could be pushed but slightly backward. There were not any evidences, either solid, fluctuating, or pulsatile, of orbital growth or tumor.

The pupils were round, that of the left eye being three and a half millimetres in size, and that of the right one three millimetres in diameter. Repeated examinations of the irides, which were normal, showed that they were freely and equally mobile to light-stimulus, carefully thrown from every part of the ordinary visual fields; while forced attempts, by which accommodative action should be brought into play, gave most excellent and prompt iridic reactions, and strong convergence, obtained by having the subject fix in the direction of its fingertip held against its nose, produced marked pupillary contraction.

No clonic movements of either iris sphincter could be seen, though

persistently searched for. Neither pupil dilated when the skin of the back of the neck was pinched. The elevator muscles of the upper lids and orbicularis muscles were freely active. The excursions of the two eyeballs, in spite of their prominence and divergence, were good in all of the ordinary physiological directions. The external portions of the two organs failed to present any areas of analgesia or anæsthesia. Local thermometric studies proved negative.

Thorough study of the interior of the eyeballs with the ophthalmoscope failed to show a single abnormality or sign or mark of any form of inflammation. The media were clear. The chorioids, the retina, and the optic nerve heads were healthy in every detail. Their contained blood-currents were well tinted, and a venous pulse in the larger retinal stems could be readily produced by pressure upon the globes. Intra-ocular tension in each eye was normal.

FIG. 1.



Careful examination showed the well-known catoptric images, the movements of the reflexes proving that there was a true accommodative play in each eye. Transillumination, as far as could be usefully employed in such a young subject, did not reveal anything abnormal.

Repeated study, with every practical form of objective and subjective test that could be conveniently employed at the time, conclusively showed that the little patient could not see even the strongest light-stimulus with either eye. Her behavior and her manner were typically those of one who is blind.

The unusual pupil signs, such as the hemianopic iris inaction (or Wernicke's sign), and the Knies pupil-symptom were unsuccessfully searched for.

In spite of the extreme youth of the patient, her mental condition was so excellent that more or less perfect essays were unsuccessfully made to discover some of the higher psycho-physiological conditions,

such as mind-blindness, visual hallucinations, word-deafness, etc. There were not the slightest evidences of any hemianopsias. There was a most probable existence of a true cortical visual amnesia, the little patient being almost constantly unable to revive visual memory pictures.

Physical examination showed that the various organs were healthy. The thyroid glands were not enlarged. There were not any cardiac or pulmonic complications. Both the deep and the superficial reflexes, as far as could be practically obtained, seemed normal for age and condition. The teeth, which were the primary ones, were irregularly placed and badly cared for: they did not exhibit any coarse signs of general hereditary or acquired disease. Examination of the nasal cavities revealed, as was expected from the constant mouth-breathing, the presence of adenoids; these sprang from the lateral walls of the anterior ethmoid cells, and almost completely filled the vault of the nose. Examination of the urine was negative.

FIG. 2.



Although at this single visit almost every ocular symptom that was deemed of the least importance for the determination of the cause of the blindness was searched for, yet the child was admitted into the wards of Wills Eye Hospital in order that the writer might at his leisure restudy this now, to him, most interesting case, thus obtaining in its entirety the above report of the conditions.

Some careful cephalic measurements made at this time were mislaid, though, fortunately, two photographs (got by much diplomacy and with much trouble by Mr. Harrison), shown in the accompanying reproductions, illustrate the general facial appearances very well,¹ and thus, with the deterioration, offer themselves as no mean substitutes.

¹ It will be noticed that the hair has been intentionally dressed in such a way as to conceal the posterior portion of the head.

During the child's two or three days' residence in the hospital Dr. W. W. Bulette, of Pueblo, Col., who happened to be visiting the writer at the time, kindly removed all of the adenoid tissue.

Tapping of the spinal fluid and cranial and spinal trephining were thought of, but no opportunity was given to put these procedures into practice.

Several months later the child was said to have died from an inter-current and unrelated disease. Autopsy, though strenuously sought for, could not be obtained. The other child was not studied.

REMARKS. Unlike most of the cases of blindness found in association with deformed crania from improper synostosis, with its deficiencies and overdevelopments, this extremely rare occipital type of osseous disease failed to exhibit many coarse motor changes in and around the eye.

The family history of a dissipated father, whose ancestral tree was bad, and a probably infected mother gave answer in great measure for the obtainment of such a product. In view of Friedenwald's observation of the preponderance of the male subject in similar cases, the sex of the case—a female—is of interest.

The presence of the adenoids cannot in any way account for such a grouping of symptoms.

In this character of subject the early want of proper binocular stimulus from imperfect visual perception, with a constantly increasing interference of sensory receipt, soon disturbs much of the interrelation of the two motor portions of the apparatus that are intended for inter-association of vision, causing the eyeballs to diverge and nystagmus to appear.¹

Far different is this from that which is seen in the oxycephalic or even the gross hypsicephalic types of cranial deformation, with their characteristic steeple-shaped or dome-like heads. In this class of degenerates the signs of disturbance in the visual apparatus are marked by gross inflammatory and degenerative changes taking place in the orbital contents. In this type, in which there is an improper union of the parietal with the occipital and temporal bones, with compensatory osseous overdevelopment along the sagittal suture and in the position of the anterior fontanelle (giving the head the appearance of a sugar-loaf), the primary changes upon delicate structures, such as nerve, vessel, lymph-channel, and even the eyeball itself, soon set into activity a whole chain of low-grade inflammatory and pressure degenerations which result in the functional destruction of the ocular globes and optic nerves. Externally, grossly proptosed, widely divergent, coarsely degenerate in many of its parts, and but indifferently mobile, the worst

¹ It is possible that this last symptom is also due to an imperfect development of the paths between the visual cortex and the so-called primary optic ganglia.

cases of this type of ocular involvement to the veriest examples, with their signs of slight prominence, fixed irides to light-stimulation, and optic nerve degeneration, may be seen.

The scaphocephalic or boat-shaped malformation of the skull, with its extremely broad forehead, caused by an improper union of the sagittal suture between the medial margins of the parietal bones, is more disposed to give rise to all of the well-known symptoms of optic neuritis, followed by atrophy. Prominent, sightless, and divergent eyes, with more or less fixed irides to light-stimulation, in subjects of fair intelligence at best—victims of convulsive seizures, as a rule—characterize the most pronounced cases in this class of abnormality.

In the leptocephalic types, in which the heads are extremely small, caused by a premature union of the fronto-sphenoidal suture between the alæ of the frontal and sphenoid bones, post-neuritic atrophy is extremely apt to appear very early in life.

The most curious trigonocephalic or three-cornered type of cranial deformity, with its small end situated anteriorly, which is due to an improper union of the frontal and parietal bones or the frontal bones at the coronal or frontal suture, has post-neuritic atrophy for its most pronounced ocular sign.

In the present case—an extremely rare example of what might with propriety be termed the occipital or occipitoparietal type of cranial malformation, in which the configuration of the posterior portion of the head is that of a flattened, but slightly curved surface, extending irregularly in an upward, forward plane to meet the frontal protuberance—the most marked ocular signs are almost wholly sensory in character. Vision in each eye is nearly or entirely lost. The orbits are shallow, particularly at their postero-mesial parts. The eyeballs are but slightly proptosed, somewhat enlarged, and enjoy full freedom of movement. The entire motor apparatus of the exterior of the eyes, with the exception of a few minor discrepancies of probable improper nuclear action, is in proper working order. The pupils are but slightly if any oversized. The irides are prompt to light-stimulus, efforts for accommodation, and convergence. The ciliary muscles are active. The eye-grounds, in every detail of neuronie, vascular, and lymph structure, appear normal; in fact, the eyeballs, with their entire adnexa, are healthy, and perform their functioning duties properly.¹

This complexus of symptoms, with its absolute blindness² and con-

¹ Careful comparison of Stood's well-known though imperfectly reported case in a thirty-three-year-old man, shows that his patient was subject to convulsive attacks which might have developed in the writer's case had it lived longer.

² As may be inferred throughout this paper, the writer is disposed to concur with Friedenwald's suggestion that "in many cases the optic nerve affection is due to temporary increase in intracranial pressure." In this type the query arises, May not the probable cortical and

comitants of slight globular protrusion, divergence, and rotary nystagmus as the only ocular signs, constitute a most remarkable clinical picture. In it is seen a blindness the proving of which necessitates a most careful study of every possible direct and indirect ocular detail; a blindness that from the ocular signs and associated conditions may be safely assumed as intracranial in type, and, most probably, until autopsy proves to the contrary, cortical in character. In such cases it is fairly certain that there is a healthy receiving material which is properly functioning: all of the ethereal wave vibrations that ordinarily give rise to the perception of color being duly received and transmitted to an intracranial position that is intended for use in ultimate perception. This lower cerebral centre—known as the visual cortex—is, unfortunately in such cases, as in the one herein detailed, of such imperfect development and of so feeble a functioning and resisting power that it early loses much of its physiological activity, and, sooner or later, degenerates into a functionless and, at times, a useless organ.

DOUBLE HYDROCELE IN AN INFANT—PROLAPSUS OF THE
RECTUM—A CASE OF OSTEOMYELITIS OF THE TIBIA—
NECROSIS OF THE LOWER JAW FOLLOWING
MEASLES—A CASE OF KNOCK-KNEE—
CELLULITIS OF THE PENIS
AND SCROTUM.¹

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Double Hydrocele in an Infant.

The first case I show to-day is a male infant seven weeks old, whose parents bring him to the hospital on account of an enlargement of the scrotum. They say that the swelling has developed gradually and does not increase materially when the child cries.

The most common conditions which would cause enlargement of the scrotum in an infant would be either hernia, hydrocele, or a growth such as sarcoma or tuberculosis of the testicle. Upon examination of the swollen scrotum I find that it presents an elastic feel, that I can feel the testes, which are normal in size, and that the tumor cannot be

connected neuronic inflammation and degeneration have been dependent in measure upon localized stretching and inflammatory processes taking place in and around a series of imperfectly made and quite readily disturbed tissues in the occipital region?

¹ A clinical lecture delivered at the Children's Hospital, October 16, 1901.

reduced by taxis, and that it does not increase in size when the child cries. The latter conditions will separate it from hernia; its elasticity and the presence of testes of normal size will differentiate it from sarcoma or from tuberculosis of the testes. Finally, I examine it by transmitted light, and find that it is translucent, and that I can see the testes occupying their normal position at the lower and back portion of the scrotum. The case is therefore one of double hydrocele.

This condition, which consists of a collection of serum in the tunica vaginalis testis, is not an uncommon one in children, and is chiefly of interest as regards the question of diagnosis from hernia or tumor of the testes.

As regards the treatment, I will first order for this infant a lotion of ammonium muriate, ten grains to one ounce of water, to be applied constantly to the scrotum on lint, and under its use the fluid may disappear in a week or two. If, however, it does not disappear, I shall then tap each distended tunica vaginalis with the trocar and canula and remove the fluid, repeating the tapping if they become again filled up. This usually brings about a cure of the disease, but if this should fail after tapping, I will inject a few drops of tincture of iodine, carbolic acid, or alcohol. Radical operations, such as dissecting out a portion of the sac, are rarely required in the treatment of hydrocele in infants or children.

Prolapsus of the Rectum.

This boy, seven years of age, was admitted to the hospital with the history that the rectum for some time had been coming down at stool and also when he passed his urine.

The patient has suffered from frequent urination, and an examination of the urine shows a large number of leucocytes. When I examined the patient yesterday the rectum was prolapsed, but now, when he is under the influence of an anesthetic, it does not appear.

Prolapsus of the rectum is one of the most common rectal affections of childhood, and we recognize it in several varieties. The most common and simplest form is that in which there is relaxation of the submucous connective tissue of the rectum, which allows a ring of mucous membrane to protrude from the anus upon straining at stool. In another form we have both protrusion of the mucous membrane and the other coats of the rectum. In a third variety, which fortunately is rare, there is protrusion of the mucous membrane and the other coats of the bowel, with an invagination of the upper portion of the rectum into the protrusion.

The frequency of this affection in children is accounted for by Jacobi by the presence of angular flexures in the lower portion of the large intestine, which necessitate marked straining in the passage of feces. Improper diet, which produces frequent stools, is often the cause of this

affection. This we have seen demonstrated in this hospital, patients often being sent in for operation suffering from prolapsus, in whom, while taking the regulated diet of the hospital, the protrusion fails to appear, and they recover without operation. The habit of allowing children to spend a long time upon the chamber utensil, with marked straining, also conduces to this disease. Difficulty in urination, due to the presence of a stone in the bladder or a narrow preputial orifice, giving rise to straining efforts in micturition, may also cause prolapsus of the rectum.

Prolapsus of the rectum is only likely to be confused with hemorrhoids or polypus of the rectum. The former condition, hemorrhoids, is extremely rare in childhood. Polypus of the rectum is also a comparatively rare affection. The diagnosis can usually be made without difficulty by a careful examination of the parts. In hemorrhoids there are one or more isolated tumors; in polypus the protruded tumor has a well-marked pedicle; while in prolapsus there is protruded a circular mass covered with mucous membrane with a depression in the centre. The treatment of prolapsus of the rectum depends upon its variety; in mild cases regulation of the diet and the daily injection after stools of a few ounces of a decoction of oak bark will be followed by a cure. In other cases if the skin of the buttock is drawn to one side during the act of defecation the prolapsus will be prevented from appearing.

The operative treatment which I have usually found effective in mild and persistent cases is that recommended by Allingham, which consists in the cauterization of the protruded mass with nitric acid. I will employ this procedure in this case, but before doing so, as the patient has suffered from vesical irritation, I will introduce a sound into the bladder to ascertain whether there is present a stone. If the latter were discovered, its removal by operative procedure would cure the affection without resort to operation upon the prolapsus itself. In this case I find no evidence of stone in the bladder, although I feel that the walls of the bladder are roughened, as the result of the cystitis from which the patient has been suffering. You notice that the bowel is not prolapsed at the present time, but when I stretch the sphincter it appears. I first wipe off the surface with a weak solution of green soap to free it from mucus, and then thoroughly dry it with a pad of sterilized gauze. I next apply some cosmoline around the skin of the anus, to prevent it from being cauterized if any of the acid comes in contact with it, and then with a swab dipped in nitric acid I carefully paint over the whole exposed surface, and finally cover it with sweet oil, and reduce the mass, and apply a pad of gauze over the anus and secure it in position with broad strips of adhesive plaster and a T-bandage.

The after-treatment consists in keeping the bowels quiet for twenty-four hours, and if they do not move spontaneously at this time a mild laxative

may be given. There may be no further protrusion of the rectum, or it may protrude at the first or second passage, and then finally fail to come down when the bowels are moved.

As I stated before, I have seen excellent results following this method of treatment, but if in any case a cure does not result from this procedure the actual cautery may be employed in the same manner that it is employed for the cure of the affection in adults.

A Case of Acute Osteomyelitis of the Tibia.

I am glad to have the opportunity of showing you a case of acute infectious osteomyelitis which was operated upon about ten days ago, and I bring the patient before you to-day for dressing, that I may show you the progress of the case at this period.

The patient, a boy, eleven years of age, received a fall from a fence about a week before his admission to the hospital, striking upon the left leg. He complained for a few days of pain in the left leg a short distance above the ankle. The leg became swollen and painful, and his family physician asked me to see the patient with him. When I saw him a week after the accident I found that he had a high temperature and a rapid pulse, and complained of great tenderness and pain in the left leg. The leg was swollen and painful from the ankle to the middle of the tibia. There was a slight red flush over the anterior surface of the limb, and on pressure I could detect deep fluctuation. I advised his removal to the hospital and immediate operation, as I considered it a case of acute infectious osteomyelitis.

Upon his admission to the hospital the patient was etherized, the skin of the leg was sterilized, and an incision four inches in length was made over the tibia through the inflamed tissues. As soon as the periosteum, which was entirely separated from the surface of the tibia, was incised a considerable quantity of pus escaped. The exposed surface of the tibia was white and presented the appearance of necrosed bone. The anterior surface of the tibia for three inches or more was cut away with the gouge, exposing the medullary cavity, which was found filled with pus and granulation tissue. The opening in the bone was then extended downward almost to the lower epiphysis of the tibia. The exposed cavity was next thoroughly cleared of pus and granulation tissue with the curette and with gauze pads; the cavity was then loosely packed with strips of iodoform gauze, and a sterilized gauze dressing was applied to the limb, which was held in position by a bandage.

The after-treatment of the case consisted in removing the dressings and packing on the second day and washing out the cavity thoroughly with sterilized water or weak bichloride solution, and re-applying the dressings. This was done at intervals of two days, and you see at the present time, ten days after the operation, that the wound in the bone

is covered with healthy granulations and is healing rapidly. At the same time the patient's constitutional condition is excellent, his temperature and pulse are normal, and convalescence seems to be perfectly established.

Acute infectious osteomyelitis is not an uncommon affection in children, and may follow a traumatism or exposure to cold, or may develop during the acute febrile diseases of childhood. It most frequently attacks the long bones, and the portion of the bone near the epiphysis seems especially liable to be the seat of the affection. The infection is usually a mixed one, resulting from pyogenic and specific organisms. The organisms reach the medullary cavity by way of the circulation. A traumatism or a local congestion may determine the site of the infection.

The diagnosis of the affection is not difficult if one bears in mind the possibility of this condition. From the fact that it often involves the medulla of the long bones near their epiphyses, it is sometimes confused with acute rheumatism. Acute rheumatism is rarely monarticular, and if the case be examined carefully it will be found that in osteomyelitis the greatest swelling and tenderness is not in the joint, as in rheumatism, but close to it.

If prompt treatment is not applied in cases of acute infectious osteomyelitis, death may result from septicæmia or pyæmia, and if the patient survives the acute symptoms, extensive necrosis of the affected bone results. By prompt operative treatment, such as was employed in this case, the patient is saved the risks of septic infection, and recovery takes place without the occurrence of necrosis of the bone.

Necrosis of the Lower Jaw Following Measles.

The next patient I show represents a form of acute osteomyelitis following measles, which has resulted in necrosis of a considerable portion of the lower jaw. This condition was described by the older writers as exanthematous necrosis.

The child, aged eighteen months, had measles several months ago, after which it was noticed that there was an offensive discharge from the mouth. When admitted to the hospital, a few days ago, on examination it was found that there was necrosis of a considerable portion of the lower jaw, with extensive ulceration of the surrounding gum.

This condition is not infrequently seen in poorly nourished children as a sequel of measles. In some cases, in addition to the loosening of the teeth and the necrosis of the bone, there is extensive destruction of the gum and of the adjacent soft parts. Gangrene of the cheeks or of the chin often results, giving rise to the condition which is known as noma. The gangrenous process may at the same time involve the genitals. The lesions of the soft parts and of the bone in these cases result from pyogenic and specific organisms giving rise to a mixed infection. Many

different organisms have been isolated, but those of the pyogenic and leptothrix variety have been most constantly observed. The disease not only results from measles, but may occur after scarlet or typhoid fever. Many patients die within a few days after the disease attacks the gums and jaw, the fatal termination being due to septicæmia or pneumonia. In other cases recovery may take place with destruction of the gums, the jaws, or the cheeks, producing great deformity.

The treatment which we employ in these cases is carried out in the following manner: The patient is etherized, the mouth is exposed as widely as possible by the use of a gag, the inflamed tissues are wiped free from discharge with a pad of gauze, loose teeth are removed, and any exposed necrosed bone which is loose is removed with an elevator or by means of bone forceps. Gangrenous soft parts are removed with scissors or a curette, and the raw or ulcerated surface is then cauterized with nitric acid or with the actual cautery. The patient then has the mouth thoroughly washed out at frequent intervals with a 1 to 2000 permanganate of potash solution, alternating with a solution of chlorate of potash. Stimulants and tonics are given at the same time, and a nutritious and easily assimilated diet should also be prescribed. Under this treatment a certain number of cases recover, and perforation of the cheek or chin may be prevented. In spite of treatment, however, the mortality following this affection is very high.

A Case of Knock-knee.

This colored child, four years of age, presents a marked rachitic deformity known as knock-knee. You notice that when the internal condyles are in contact the legs are separated at the ankle for a distance of about 12 to 14 inches. You see at the same time that there is a decided flat foot. This condition may be treated by the application of braces or by an operative procedure known as osteotomy. The latter method in a patient in this class of life is the better procedure. I will in this case do an osteotomy just above the condyles of the femur—Macewen's operation.

The patient being etherized and the limbs having previously been sterilized, I make a puncture with a bistoury down to the bone about 1½ inches above the inner condyle of the femur, and pass an osteotome into this incision down to the bone, and turn it so that the blade is transverse to the long axis of bone. With the strokes of a mallet I next carefully divide the bone, keeping my incision well away from the posterior surface of the femur, to avoid possible injury of the important bloodvessels in that region. When I feel by the resistance offered to the osteotome that the bone is nearly divided, I remove the instrument, and grasping the thigh and the leg I complete the division by fracturing the remaining fibres of the bone. The limb is then brought into a

position of overcorrection, the small wound is closed by one or more sutures, and a compress of gauze saturated with the tincture of benzoin and collodion is placed over it. A layer of cotton is next applied to the ankle and the bony prominences about the knee, and a flannel bandage is applied to hold this in position. While the limb is held in a position of overcorrection a plaster-of-Paris bandage is carefully applied from the foot to the upper portion of the thigh. The same operative procedure is applied to the other limb, and a correction of the deformity is secured in the same manner.

There is usually no reaction following this operation and the plaster-of-Paris bandages are allowed to remain in position for four weeks. They are then removed and fresh bandages applied for three or four weeks longer. At the end of eight weeks union is usually quite firm, and the patient is then allowed to use the limbs in walking, without any support being applied to the limb.

Cellulitis of the Penis and Scrotum.

This infant, aged seven weeks, presents a swollen condition of the penis and scrotum, with, at the same time, numerous superficial points of ulceration over the inflamed surface. The condition is one which we recognize as cellulitis, and has existed for a week, and, according to the statement of the mother, appeared a few days after a forcible retraction of the prepuce to relieve a condition of phimosis. It is possible that this condition resulted from an infection of the tissues as the result of this procedure. I have seen retraction of the prepuce in an infant result in infection and gangrene of the prepuce, and therefore do not consider the procedure an entirely safe one unless great care is taken to thoroughly sterilize the parts and the fingers or instruments used before the procedure is employed.

In this case the condition seems at the present time to require no treatment other than great care in keeping the parts thoroughly cleansed by the use of castile soap and water and a lotion of boric acid, followed by a protective dressing of oxide of zinc ointment.

IMPACTED CALCULUS IN THE URETHRA IN CHILDREN.

A REPORT OF TWO CASES.¹

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VESICAL calculus in children, although infrequent in this country, is not of such rarity as to occasion comment when encountered. The

¹ Read before the Philadelphia Pediatric Society, November 12, 1901.

accident of impaction in the urethra is a sequel of more unusual occurrence, and having met with two such cases within a few months at the Children's Hospital has led me to make this report of them, with some remarks upon the diagnosis and treatment.

CASE I.—Edward N., white, aged three years; admitted February 13, 1901. The family and previous history were negative. For two weeks the child had been observed to strain while urinating and at stool. He had passed no urine for twenty-eight hours before admission, and had been very restless and cried a great deal during the preceding night. This morning it was noticed by the family that the abdomen, scrotum, and thighs were swollen and painful. On admission the temperature was 97.8° F., pulse 132, respirations 32. General condition fairly good. There was observed a very great enlargement of the scrotum, the swelling also involving the perineum and extending upward over the abdominal wall as high as the umbilicus, and laterally and below to about one-half inch below Poupart's ligament on either side. The prepuce and sheath of the penis were also involved in the œdema. The prepuce could not be retracted sufficiently to expose the meatus of the urethra. During the short time the child was under observation before operation, about one hour, the œdema could be seen to extend downward on the thighs in a line parallel to Poupart's ligament. A diagnosis of urinary extravasation was readily indicated by the history and the distribution of the swelling, and Dr. Wharton, who saw the case with me, expressed the opinion that the probable cause of urethral rupture was an impacted calculus. The child was speedily prepared for operation. Under ether the prepuce was split up on its dorsum and a sound passed into the urethra. A calculus was immediately met with in the spongy urethra, about two inches from the meatus. Owing to the smallness of the parts, no instrument could be introduced into the urethra that would grasp it, although a staff could be passed beside it into the bladder, evacuating a moderate amount of urine. With the child in the lithotomy position the urethra was opened upon the staff in the median line behind the scrotum, and after some little difficulty the stone seized where it lay in the bulb and extracted. Incisions were made on either side of the scrotum, and in both groins, to evacuate the extravasated urine, an English catheter was tied in the bladder through the perineum, and the wounds dressed. The stone was small, red, irregularly oval in shape, with a rough, mammillated surface—evidently a uric-acid calculus.

The patient did very well until the third day. The œdema rapidly subsided, and there was apparently no sloughing of the subcutaneous tissues as a result of the extravasation. On the morning of the 15th, after the temperature had dropped to normal, the child vomited, and in the evening the temperature rose to 106° F., falling again, after sponging, to 99° F. by the following morning, when a bright red rash developed on the body and limbs. The temperature rose again in the evening to 102.2° F., the pulse meanwhile ranging from 144 to 196, and the respirations from 32 to 50. During the two days following he vomited occasionally, and the temperature was irregular, varying from 98.2° F. to 101.4° F. The rash was strongly suggestive of scarlet fever, especially when considered in combination with the vomiting and rapid pulse. Dr. Griffith, who saw the case with Dr. Ashhurst and

myself, regarded it as probably one of scarlet fever, and the child was isolated. The local condition meanwhile progressed satisfactorily. The first attempts to dispense with the catheter on the third day were unsuccessful, and it had to be re-introduced. It was removed a couple of days later, and urine passed voluntarily through the perineal wound, and later through the penile urethra also. The temperature remained down from the 18th to the 20th, and the child seemed to be improving; but on the 20th it again rose to 105.4° F., and vomiting began again. On the 26th diffuse desquamation was noticed over the body and thighs. The child became much weaker, delirious, and very restless; the circulation failed rapidly, collapse developed, and he died the same evening.

The nature of the fatal complication was a matter of some doubt. In many ways it resembled scarlet fever, but it could not be positively stated that it was not a septic condition from urinary absorption, the rash being one of the so-called "surgical rashes," such as we occasionally see in children, and which in a children's hospital is a factor, sometimes causing much anxiety and alarm. The fact that powdered iodoform was sprinkled upon the wounds at the time of operation was also considered. The onset on the third day after admission precluded any question of intramural contagion, nor were there any other cases of scarlet fever at that time in the hospital.

CASE II.—Francis D., white, male, aged three and a half years, admitted May 27, 1901. Family and previous history negative. On the evening of the 26th the child complained of pain over the bladder. This was about 5 P.M. No urine was passed from this time until admission in the afternoon of the 27th, when the house surgeon, Dr. Walker, succeeded in introducing a catheter into the distended bladder and emptying it, and on withdrawing the instrument a stone was felt in the urethra. On the following morning I examined the boy with Dr. Ashhurst, and found a calculus in the bulbous portion of the urethra. As it could not be dislodged per urethram, for the reasons mentioned in the previous case, median urethrotomy was at once performed. The stone, owing to its small size and smooth, rounded surface, was not easily extracted, but was finally removed by means of a small, curved stone-forceps. It was a smooth, oval, uric-acid calculus, measuring three-eighths by one-quarter inch, and weighed, when dried, four and two-thirds grains. An English catheter was tied in the bladder through the perineum. The temperature never rose above 100.4° F., and the child's condition remained satisfactory. The first attempts to remove the catheter from the perineum three days after operation failed, as it had to be re-introduced the next day. It was finally removed on June 3d and a silver catheter passed per urethram. The perineal wound was healed by June 9th and the child discharged shortly thereafter.

In the first case there were symptoms of stone noted before impaction, or at least obstruction, developed, viz., straining during urination and defecation, but they were misunderstood by the parents and attributed to constipation. In the second case no symptoms had been observed

before the stone engaged. We are often led to suspect the presence of stone in children from the presence of bladder irritability, when none exists, and will sound many children for stone where none is present to one where a calculus will be detected. Reflex irritability of the bladder is such a common symptom in children, as a result of phimosis, hyperacidity of the urine, rugosity of the bladder wall, etc., that the presence of a calculus cuts a comparatively small figure; but it is one the possibility of which should never be forgotten if we would save ourselves on some occasion from a mortifying oversight.

Retention in children I believe to be usually reflex in character, and not very uncommon as a result of phimosis, etc. Rupture of the urethra from traumatism, and perineal abscess the result of inflammation or injury, can cause real obstruction, as may also stricture of the urethra, which has been shown to manifest itself even in very young children as a result of gonorrhœal urethritis. Abbe¹ has reported a case of impermeable stricture of the deep urethra in a male child, aged two and a half years, developing six months after gonorrhœal urethritis, associated with two other strictures of the anterior urethra, and demanding suprapubic aspiration of the bladder and external urethrotomy. But the most frequent cause of urethral obstruction in children is probably calculus. Holmes, in his *Surgical Treatment of the Diseases of Infancy and Childhood*, published many years ago, says that when summoned to a case of retention in a child we can almost always assume it to be due to impacted calculus. It may, when overlooked, easily lead to perineal abscess as well as to extravasation. Even the passage of the catheter may not detect its presence.

The removal of an impacted calculus in children is, from the small size of the urethra, more difficult than in adults. If situated near the meatus, or anywhere in the penile urethra, it can sometimes be seized with fine forceps after a preliminary meatotomy and removed. In the deep urethra attempts may be made to push it backward into the bladder, where it can be treated by litholapaxy. Failing these attempts, the urethra must be promptly opened. When the urethra is already ruptured no time should be lost, and urethrotomy and drainage of the infiltrated tissues at once proceeded with. Judging from our experience, it will usually be found necessary to leave an English catheter in the perineal opening for several days, as some swelling of the parts is to be expected, and the presence of a raw surface over which the urine must flow also probably excites a reflex spasm of the sphincter.

¹ New York Medical Journal, 1890, vol. llii. p. 666.

A CASE OF CHRONIC LYMPHATIC LEUKÆMIA IN AN INFANT.

WITH SOME OBSERVATIONS ON THE BLOOD CONDITION IN
OTHER CASES OF SPLENIC TUMOR.

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Our knowledge of the blood condition in many of the diseases of infants and children is at present hazy. Each individual case of anæmia in children carefully observed and studied may add something to our knowledge. It is with this hope that the present case is reported.

Peter M., an Italian boy, aged nine months, first came under the observation of Dr. Henry M. Fisher on May 20, 1900. The child's parents are both living and healthy; they present no indications of disease, though living in poor and squalid surroundings in the southern part of Philadelphia. There are three girls in the family, aged, respectively, twelve, ten, and three years. The eldest, who lives in Italy, is said to have had an enlarged spleen, with anæmia, from early infancy. She is still a delicate child. The other two children are healthy.

The parents said the boy presented no striking peculiarity until he was one month old, when it was noticed that he was unnaturally pale. Since that time he suffered frequently from recurring attacks of intestinal catarrh, with green stools containing much mucus. His mother was and still is nursing him at seventeen months, though her milk is scanty and is reinforced by other baby foods. Head was symmetrical; fontanelles almost closed; teeth were cut with normal rapidity. No evidence of rickets. At the first visit the spleen was enlarged extending fully four inches below the costal margin. There was no marked enlargement of either the liver or the external lymphatic glands. The heart and lungs were normal. The urine was non-albuminous and contained urates in excess. Blood smears were prepared and first examined on May 29th, though no blood count was made at that time. The slide showed a marked poikilocytosis, with many microcytes and macrocytes and a decided leucocytosis in which the lymphocytes were proportionately high. There was a considerable number of nucleated reds (normoblasts). There were no malaria parasites. The blood was subsequently examined, counted on June 15th, 25th, July 6th, November 23d, January 7, 1901, and May 23d. The results of these examinations will be found in the table on opposite page.

The child was placed upon quinine by suppository, and was also given one-half drop of Fowler's solution three times a day. During the latter part of May there was considerable diarrhœa, with green stools containing mucus. During July there was a slight improvement in the patient's condition, which seemed to follow the use of oil inunctions and eudoxine, 2 grains given every two hours. The child was advised to be given as much as possible the benefit of fresh air, and was at times taken to the sanitarium at Red Bank. The spleen had enlarged during this interval, and could now be felt to within one finger's breadth of the iliac crest, and posteriorly two fingers' breadth below

SCOTT: LYMPHATIC LEUKÆMIA IN CHILDREN. 21

the costal margin. He was not seen again until November 21st, when the child had frequent stools, with considerable bronchial catarrh. At that time he was put upon $\frac{1}{2}$ a grain of glycerin-phosphate of soda in solution with dilute phosphoric acid, $\frac{1}{2}$ minim of Fowler's solution and 2 grains of protonuclein, t. d., which treatment was continued through December.

BLOOD TABLE.

| | 1900. | | | | | 1901. | |
|--------------------|------------|------------|------------|-------------|------------|------------|------------|
| | May 24. | June 15. | June 27. | July 6. | Nov. 23. | Jan. 7. | May 23. |
| Erythrocytes, | | 3,120,000 | 3,080,000 | Not count'd | 3,360,000 | | 3,097,000 |
| Leucocytes, | | 105,000 | 54,000 | " | 18,600 | 36,700 | 80,733 |
| Hæmoglobin, | | 20 per ct. | 22 per ct. | | 20 per ct. | 30 per ct. | 15 per ct. |
| Polynuclear, | 14 per ct. | 27.5 " | 30 " | 28 per ct. | 25 " | 47 " | 15 " |
| Small lymphocytes | 65 " | 27 " | 17 " | 44 " | 44 " | 23.4 " | 66.9 " |
| Large lymphocytes | 18 " | 30 " | 44 " | 18.4 " | 9.6 " | 20.6 " | 13.8 " |
| Eosinophiles, | 3 " | 7.5 " | 2 " | 4.8 " | 8.6 " | 5.6 " | 2.2 " |
| Myelocytes, | 0 " | 8 " | 7 " | 4 " | 2.7 " | 3.4 " | 2.1 " |
| Normoblasts, | 33 | 92 | 39 | 46 | Many | 85 | 125 |
| Megaloblasts, | 8 | 2 | | 5 | | 18 | 8 |
| Microblasts, | 3 | 0 | | 1 | | 3 | 2 |
| Polychromatophilia | Yes | Yes | Yes | Yes | Yes | Yes | Yes |
| Number counted, | 200 | 200 | 225 | 250 | 185 | 500 | 1000 |

On November 30th Dr. Charles A. Oliver examined the eye-grounds and reported "a neuroretinitis, with engorgement of both the retinal arteries and veins, with extremely pallid blood; there were no hemorrhages." When last seen, May 23d, the child had suffered a relapse, for it had improved considerably during the early winter months. It presented a peculiar clayish pallor, the lips colorless, with but little emaciation, the abdomen enlarged and protuberant, the spleen hard and very much enlarged, with no enlargement of the superficial lymphatic glands, and the edge of the liver just palpable. Since the beginning of the illness he has never had any nose-bleed. Once, in January, he coughed up a small amount of blood. There have never been intestinal or gastric hemorrhages. The child died about ten days (June 2d) after the last blood examination, from asthenia; no autopsy could be secured, as Dr. Fisher was unfortunately not in Philadelphia at the time.

A brief résumé of the appearance of the blood slides is, I think, necessary at this time. The first film prepared by Dr. Fisher and sent to me for diagnosis showed many changes in the size and form of the red, with much loss of hæmaglobin and decided leucocytosis, the percentage of lymphocytes being comparatively high, some few myelocytes present, and nucleated reds in plenty, some undergoing mitotic change. During June there was a great increase in the number of leucocytes, reaching over 100,000 per cubic mm. There were again numbers of microcytes with very pale macrocytes, with large numbers of both young and old normoblasts, many of them mitotic. There were many polychromatophilic cells; degenerative changes in the reds were common, especially in the old normoblasts. At this time the proportion of large lymphocytes was rather high. In July there was little change to note in the blood except the reduction of the total number of the leucocytes, which continued to fall until, November 23d, they were but 18,600, with a proportionate increase in small lymphocytes. In January the

leucocytes again began to rise, the count at this time being 37,600, and when last seen in May, after a lapse of some four months, during which time treatment had ceased through neglect of the parents, the white count reached 80,000. During this period of almost a year the erythrocyte count has remained almost stationary (about 3,000,000), while the hæmaglobin has been low. The last differential counts are of considerable interest. They show a distinct increase in the lymphatic elements of the blood; in fact, the last films made, as can be seen by consulting the table, showed over 80 per cent. of lymphocytes. Over 66 per cent. of these are small, while but 13 per cent. are large lymphocytes; many of them, however, resemble nucleated reds, the nucleus remaining rather reticulated. Some of the red corpuscles show a basophilic granulation; some of the lymphocytes are undergoing segmentation, and three or four were observed with bipolar nuclei. Myelocytes are present but in rather scanty numbers; almost 50 per cent. of the eosinophiles are myelocytic.

DIAGNOSIS. In children enlargement of the spleen is common; the usual causes may be briefly stated as malaria, typhoid fever, congenital syphilis, rickets, *v. Jaksch's* disease, splenic anæmia, leukæmia, and acute and chronic infections. A differential diagnosis between two of these diseases at times becomes extremely difficult unless we carefully bear in mind the characteristic reaction of the infant and the child's blood in various diseases—*i. e.*, the ready response of the leucocytes, and especially the lymphocytes, to any call made upon them.

Thus *malaria* in the infant or child is usually associated with definite clinical symptoms, enlargement of the spleen, the absence of leucocytosis, the presence of the specific parasite. In chronic paludism this picture may be somewhat changed. The degree of anæmia may be severe, and corresponding changes may occur in the red cells, but there is no marked alteration in the leucocytes except a tendency to a lymphocytosis.

In *congenital syphilis* there is always anæmia. The hæmaglobin is reduced to the lowest figure. There is a great diminution in the reds, nucleated reds are seen in abundance, and the white cells, especially the lymphocytes, are increased. Loos reports a fatal case in which the leucocytes reached 58,000.¹ Cabot reports a case of syphilis with leucocytosis of 100,000, and Monti, Baginsky, and others have also reported excessive leucocytosis. If the skin lesions are severe there may be an excess of eosinophiles. The spleen is usually decidedly enlarged.

In *rickets* there is usually a simple chlorotic anæmia. The leucocytes may rise to 30,000. (Luzet, however, believes that all grades of leucocytosis, even to a true leukæmia, exist in rickets, and there is a decided belief among many observers that both syphilis and rickets not infrequently lead to leukæmia; a lymphocytosis is usually the result.) In complicated cases the blood picture becomes not unlike that of *v. Jaksch's* disease—that is, there are unusual numbers of nucleated reds with

¹ Ewing, p. 297.

many poikilocytes. In such cases Ewing states the hæmaglobin index is unusually high. The spleen in these cases is enlarged, and there are usually other evidences of rickets, such as the beading of the ribs and changes in the other bony structures.

In *v. Jaksch's disease*, which is supposed to be peculiar to infants and young children, the following changes in the blood are considered characteristic: There is a great and unusual diminution in the number of red corpuscles. There is always a leucocytosis, which, however, is not so great or progressive as in leukæmia. There is a remarkable variety of the forms and sizes of the leucocytes. The reds show great poikilocytosis; there are numerous nucleated reds to be seen. The clinical characteristics of the disease are the great enlargement of the spleen without marked enlargement of the liver, the edge of which is sharp, and without marked glandular enlargement. The prognosis is relatively good. Three cases which came to post-mortem presented no evidences of leukæmic infiltration. The disease has an anatomical basis, though Monti and Buggrum, in analyzing twenty cases, found evidences of rickets and syphilis in sixteen. It is also to be remembered that prior to 1892 (*v. Jaksch's disease* was described in 1889) there is no case on record, according to Fischl, where a differential count was made.

Splenic Anæmia. A disease not common in children, though recently numerous observers have reported cases in infants. In this condition the blood shows no pathognomonic changes. There is usually but little change in the form and size of the red corpuscles, while the hæmaglobin is much reduced—a chlorotic anæmia. There is usually but little or no increase in the leucocytes unless high temperature has been associated with the disease; indeed, in Osler's fifteen cases—all adults—a leukopænia was the rule.¹ Nucleated reds are rather scanty, and may not be found at all. The spleen is usually enlarged and tender; this enlargement precedes the anæmia,² the liver is often slightly enlarged, while there is sometimes slight jaundice present. The digestive symptoms are probably the most prominent. There is a tendency to hemorrhage either from the stomach or bowels, not infrequently from the nose. The duration of the disease is protracted, its cause is unknown, recovery has been seen.

Leukæmia. A disease which is rather uncommon in infancy, though the literature now contains quite numerous reports of cases occurring from one month up to extreme old age. For purposes of brevity we can assume an acute and chronic variety. The chronic variety, or myelogenous type, is the more frequent. The blood picture

¹ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, January, 1900, p. 54.

² Osler. *Ibid.*

is typical. The leucocytes are increased to astounding proportions, sometimes one to two, four or six. A large proportion of these cells are myelocytes; mast cells are present in considerable quantities, as are also nucleated reds; the mast cells Ewing considered almost pathognomonic. Eosinophilic myelocytes are numerous. The disease is chronic in its duration, we do not know its cause, and cure is almost unknown.

Lowit has described a protozoon, and many others have found similar bodies in leukæmic blood, but no one has been able to verify or follow up his findings. The lymphatic variety is more rare, and the acute cases are usually of this type, the majority of these cases occurring before the age of twenty-five. Thus, Muir states that of the seventeen cases reported by Ebstein, but seven were over thirty, and Muir's four were all under twenty-six. The blood picture is here almost characteristic. The leucocytosis is usually about 200,000. A large proportion, 85 per cent. to 95 per cent. of them, are lymphocytes. Myelocytes are found but in very scanty numbers. Nucleated reds are also extremely scarce, but are sometimes found. Segmenting lymphocytes and the nuclei of large lymphocytes, without cytoplasm, are frequently seen. Vacuolated and degenerated or disintegrating leucocytes are frequent. There is no marked reduction of the red corpuscles, nor is there much change in size or shape. The hæmoglobin keeps pace with the reds. The clinical symptoms depend to some slight extent upon the variety of the disease. In the spleno-myelogenous form the onset is gradual; epistaxis is a more common symptom; diarrhœa is usual; there is increasing weakness, a peculiar grayish pallor, with decided splenic enlargement. The latter is often noticed by the patient himself. In the lymphatic variety there is usually enlargement of the entire superficial lymphatic apparatus. The glands are rather elastic, but become soft. At times the superficial glands are not affected, though the blood picture remains the same as before stated. Under such cases there is usually a leukæmic infiltration of either the glands along the intestinal canal or in the viscera. The spleen is not usually affected, though sometimes it is slightly enlarged. Thus Muir¹ cites a recent case of his own in which the liver, kidneys, and adrenals were infiltrated, while the glands were almost unaffected. Lymphatic leukæmia, therefore, in infants is not common. Morse² gathers twenty cases of leukæmia; but one of these, however, was accompanied with a differential count, and as the diagnosis in some of the cases was made by clinical signs, without study of the blood, many of these cases must be thrown out. Morse's case was of mixed variety. A. E. Taylor in his studies on leukæmia³ reports six-

¹ Allbutt's System, vol. vi. p. 656.

² Boston Medical and Surgical Journal, August 9, 1894.

³ Pepper. Clinical Laboratory, vol. ii. p. 143.

teen cases of leukæmia, four of which are lymphatic, but none in patients under eleven years of age. Morse, again in 1898,¹ quotes seven acute cases in children from the literature, and describes his own case, in which but one blood examination was made. McCrae² reports an acute case of leukæmia occurring in a boy of three years, and includes thirteen previous cases from literature. Ewing (page 201) states that Audeod has observed leukæmia in fifty-six children; eleven times in the first year, twelve times from the second to the fourth year, twelve times from the fifth to the ninth year, and twenty-one times from the tenth to the fifteenth year.³

The blood picture in my own case corresponds accurately to the description given of pseudoleukæmia anæmia infantum, or v. Jaksch's disease, and the clinical picture also coincides with its description. In none of the cases, however, of v. Jaksch's disease have I been able to find a careful report with differential counting of the blood, and many of the cases reported as such have been based upon the presence of a leucocytosis, sometimes quite small, with evidences of anæmia and decided enlargement of the spleen. The differential counting of the leucocytes, it seems, must be practised in these cases before an accurate diagnosis can be made. This fact leads me to this query: "Does our knowledge of the blood at the present day allow us to acknowledge that the disease described by v. Jaksch is an entity?" If it is not, in what group of diseases can it be placed?

In order to discuss this question, let us briefly consider the effect upon the blood: First, of simple splenic enlargement. Second, removal of the spleen: (a) for injury, (b) in splenic anæmia, (c) in myelogenous leukæmia. Simple splenomegaly in acute diseases, such as typhoid fever, malaria, etc., is accompanied by a secondary anæmia, which is probably induced by the disease. The leucocytes are usually diminished in number (leukopænia). In the more chronic enlargements, such as malaria or splenic anæmia, there is again a leukopænia, or a mild grade of leucocytosis, as in rickets and syphilis; thus the most marked effect upon the blood seems to be a relative reduction in leucocytes. But this is relative, for with the onset of complications a decided leucocytosis takes place, showing that the leucocytes are present in the body, though not in the peripheral blood. This leukopænia I have also seen in cirrhosis of the liver, accompanied with marked ascites and splenic enlargement, and also in another case of simple splenomegaly whose blood showed an intense chloro-anæmia. This latter case was classed as a splenic anæmia for want of better diagnosis, though he presented none of the ordinary symptoms of the disease.

¹ Archives of Pediatrics, 1898, vol. xv. p. 180.

² Johns Hopkins Bulletin, May, 1900, p. 102.

³ Traite de Malade d'Enfance, t. II., p. 112.

Bovaird has reported two cases of chronically enlarged spleen with the pathological diagnosis of endothelioma, in which the blood changes were but slight. In the first case, a child, aged three years, the erythrocytes numbered 4,400,000; the leucocytes 9000. In the second case, a child, aged thirteen years, the erythrocytes numbered 2,880,000, and the leucocytes 4000.

Sailer, in a recent paper, has reported six cases of enlargement of the liver and spleen—with enlargement of the lymph glands—in which the presence of tubercle bacilli was demonstrated; in these cases the blood showed no special changes, there was no leucocytosis, and the differential count was normal.

The total removal of the spleen has a decided effect upon the blood. In comparatively healthy subjects Ewing states (page 241) that the spleen may be removed without affecting the blood more than any other laparotomy. Such an operation is usually followed by a leucocytosis ranging from 15 to 50,000, which may persist from two to six weeks, or even longer. In traumatic cases the blood may resemble an acute leukæmia, although this does not always follow. The spleen has also been removed for diseases such as malaria (ague cake), splenic anæmia (Osler only so advises when hemorrhages are recurrent), and myelogenous leukæmia. In the latter the result usually is fatal, and no blood counts of value, subsequent to the operation, are to be found. Warren, however, has included in his article on "Surgery of the Spleen," a case of myelogenous leukæmia, operated upon by Richardson, with recovery.

In splenic anæmia the results of operation are better. Osler, Warren, and Malcolm L. Harris, with Max Herzog, and others have reported cases of removal of the spleen for this disease. In Warren's case the leucocytes after operation remained up for three months, the highest count being 24,000. Harris and Herzog's two cases showed a leucocytosis of 28,000 before operation, with no subsequent increase after the removal of the spleen. They noted, however, a great increase in the eosinophiles, after four months to 14.4 per cent. This change in the differential count is said not to occur until some months subsequent to the operation. They also noted a decided increase in the mononuclear cells.

For malaria the spleen has frequently been removed. Hogan quotes sixty-four cases since 1890, with a mortality of 23.4 per cent. No statements are to be found as to its effect upon the blood. From the above it can be seen, therefore, that no one effect upon the blood is produced by simple enlargement of the spleen, or by its removal in health or in disease. Observers differ as to whether splenic enlargement precedes, as in splenic anæmia, the blood condition, or whether the latter produces the splenomegaly. In splenic anæmia Dr. Osler considers the

enlargement antedates the anæmia, and is not its cause. Harris and Herzog consider the splenomegaly the cause of the anæmia, and call attention to the good results of operation to support their contention.

In the case above considered there was present a typical clinical picture of chronic splenomegaly, with a leucocytosis of a slowly increasing lymphatic type; the presence of nucleated reds of all sizes and ages, degenerative changes in the reds, the same changes in the leucocytes, the presence of a few myelocytes, with death from asthenia, and, alas, no autopsy. That these cases exist without leukæmic infiltration of tissues is true, for in two cases coming to autopsy, Ewing has failed to find any such pathological change. The duration, the course, the blood picture, and the termination of this case are in common with the leukæmias of adult life; at the present time one cannot predict how long the leukæmias will be permitted to remain in the class of primary or essential anæmias: they present many of the characteristics of true secondary anæmias, and not a few observers consider that in the secondary anæmias, splenic anæmia and pseudoleukæmia anæmia infantum have their proper position. I am inclined to support this view.

[NOTE.—Since writing this article there has been published in the *Boston Medical and Surgical Journal* a comprehensive and elaborate article on the "Association of Anæmia with Chronic Enlargement of the Spleen," by Arthur H. Wentworth (concluded in the number of October 31, 1901). The conclusions reached in the article are valuable, while the entire literature from 1879 to 1900 is appended. Stengel, as early as 1896, in his article in the *Twentieth Century Practice*, contended that pseudoleukæmia anæmia infantum was not an independent disease.]

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 McCrae to 1900, with full bibliography attached.
 In Ewing's book will also be found a full bibliography.

UNCINARIOSIS (ANKYLOSTOMIASIS). A FURTHER REPORT OF A CASE, WITH NOTES UPON THE AUTOPSY.¹

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IN view of the fact that a number of cases of uncinariosis have been discovered in the United States (Missouri, New York, Louisiana, District of Columbia, Texas, Virginia, Maryland—not all yet reported) in the last few years, the majority in the past few months, and since, to many of us, unless engaged in teaching, it is little more than a name, it seems advisable to bring this serious disease to the attention of the profession in some detail.

It is my belief, from the nature of the disease, as well as from recent developments, that it is far more common among native Americans than has been the general impression heretofore. Besides, there is the ever-increasing danger of its importation in emigrants from localities where the disease is prevalent, and in those returning from our newly acquired possessions.

Uncinariosis (ankylostomiasis) is a chronic disease due to infection by a small thread-worm known as the *uncinaria duodenalis* (ankylostoma duodenalis), which is characterized clinically by intense anæmia, dyspnœa, weakness, gastro-intestinal disturbances, and, in advanced cases, by œdema of the face and extremities and serous effusions into the cavities of the body.

HISTORY. The disease has been described under various names: tropical chlorosis, Egyptian chlorosis, geophagia, mal d'estomach, cachexie aqueuse, cachexia africana, tunnel disease, miner's disease, etc.

In the *Papyrus Ebers* (German translation by Dr. H. Joachim), the oldest book upon the subject of medicine, written in the middle of the sixteenth century before Christ, is recorded a disease among the Egyptians known as the *ā a ā*, which so strongly resembles uncinariosis in its symptomatology that it seems at least possible that they are identical. The similarity is further strengthened by the mention of remedies given for worms in the intestines of those afflicted with this disorder.

From the fact that it is several times spoken of as the deadly *ā a ā*, it must have been very fatal in those ancient times.

According to Sandwith² the first mention of the disease in modern times was in Brazil in 1648, and in the West Indies and in

¹ Read before the Medical Society of the District of Columbia, November 6, 1901.

² Observations on Four Hundred Cases of Ankylostomiasis, *Lancet*, 1894, vol. 1.

Guiana in the early part of the eighteenth century, while in Europe it was first noted among the miners of Auzin in 1802.

To Angelo Dubini, of Milan, is due the credit of discovering the nematode in man which is the cause of the disease. He, in 1838 (published 1843), found the parasite¹ in the small intestine of a woman dead of pneumonia; but the importance of the discovery was not realized until 1854, when W. Griesinger declared, upon the evidence of a single autopsy, performed in Cairo, April 17, 1852, that Egyptian chlorosis was caused by the *ankylostoma duodenalis*.² This fact was apparently lost sight of until, in 1866, it was confirmed by Wucherer.³

The disease came into prominence in Europe in 1879 or 1880, when a number of workmen in the St. Gothard tunnel developed a severe form of anæmia, which was found to be uncinariosis.

The geographical distribution is wide, the disease having been reported from most of the countries of Europe, in Africa (especially Egypt), Australia, South America, the Indies, the United States, and other localities. As would be expected, it is more common in the tropical and subtropical countries. Sandwith states that the parasite has been found between the parallels of 52° N. and 30° S., but that above 47° N. it can develop only in sheltered localities, such as mines, tunnels, etc.

As our possessions, excepting Alaska, lie entirely below the 50th parallel, there seems to be no reason why we should not meet with uncinariosis more frequently than we have heretofore.

I am told by Dr. Stiles that parasites of the same genus as that found in man cause the death of many dogs in this locality, and are very common in Alaska in the fur-bearing seal, and through the South in sheep, goats, and cattle.

There is thus far no proof, however, that these parasites are transmissible to man.

According to Manson, in Egypt the parasite is found in nearly every cadaver, and in India it was recovered from 75.58 per cent. of 1249 natives to whom thymol had been administered.

It must not be supposed, however, that all those who are infected show the symptoms of uncinariosis; far from it, many are strong and healthy. The weak and poorly nourished are those who are least able to withstand the loss of blood occasioned by the parasite.

PARASITE. The worm is nearly cylindrical in shape, from 0.4 to 1 mm. ($\frac{1}{25}$ to $\frac{1}{5}$ inch) in diameter, and from 6 to 18 mm. ($\frac{1}{4}$ to $\frac{3}{4}$ inch) in length. It is yellowish or grayish-white in color, with translucent edges. The males are more slender and more transparent than the

¹ Dubini gave the name of *ankylostoma duodenalis* to the worm, which, according to zoologists, is wrong, since the genus is identical with that found in the wolf, in 1789, by Föllch, and called by him *uncinaria*.

² Archiv für Physiologische Heilkunde, 1853-1854.

³ Gazeta Medica da Bahia, 1866, and Archiv f. klin. Med., 1872.

females by reason of the less extensively developed sexual organs. The head end is always bent backward. The tail, which can be distinguished by the swelling at the end (the bursa copulatrix) is much more curved than the head, sometimes appearing even to be rolled up. The length of the mature male is from 6 to 11.5 mm. The diameter is nearly the same for the posterior two-thirds (about 0.5 mm.), but the anterior third tapers down and ends in a truncated cone at the head. The female is usually larger (from 6 to 15 mm.), much thicker (about 1 mm.), and is generally found slightly curved dorsally. The number of the females is, as a rule, much larger than that of the males.¹ The armamentarium of the parasite consists in four claw-like hooks set in the margin of the mouth capsule, two on either side of the ventral line, and two conical teeth, one on each side of the dorsal line. By means of these the worm clings to the mucous membrane of the small intestine and sucks the blood from its host.

From the fact that the blood cells are found unchanged in the parasite, it is believed that only the serum is used for its nourishment; thus it becomes a most wasteful feeder.

The ova, which are a perfect oval in shape, measure 0.055 to 0.065 mm. by 0.032 to 0.043 mm., and are covered with a transparent shell through which may be seen the segmented yolk.



Ova of the *uncinaria duodenalis* (multiplied about 300 times).

Looss² has proved by experiment that the development of the egg depends upon the surrounding temperature, its accessibility to the air, the amount of moisture, and upon the character of the culture media. Thus he found that in Alexandria, Egypt, where the temperature in summer varies around 80° F., the eggs on the surface of the media develop and the embryos escape in twenty-four hours. If a cool night intervenes, with a temperature of from 70° F. to 72° F., these changes do not occur for thirty-six to forty hours, and that a temperature of 33.8° F. kills the eggs in twenty-four to forty-eight hours. He also found that too much moisture not only delayed the development of the eggs, but caused the death of a large number of them.

About the second or third day the rhabditiform embryo, when under favorable circumstances, undergoes the first ecdysis—*i. e.*, casts its cuticle, which has by this time become loosened. From the fifth day

¹ J. Ch. Huber. Twentieth Century Practice of Medicine.

² Centralbl. f. Bakter., u. Parasitenkunde, 1896.

on the second ecdysis takes place. The cuticle is seen to divide into two layers, which separate more and more until the outer cuticle is separated from the body, but remaining around it, thus giving rise to the theory of encystment.

The young worm has now reached the infection stage and is ready to enter its host and begin its parasitic life. Looss has succeeded in keeping worms alive in this condition for three months. The ripe embryo prefers water as its medium, and may thus easily be taken into the system. He has shown that both eggs and embryos are killed by being dried out, which tends to disprove the theory of infection through dust which may be blown about.

Looss¹ estimates that about four or five weeks are necessary after infection for the worm to become fully mature, and that in Egypt five or six weeks is the probable period necessary for the freshly laid eggs to develop into individuals containing eggs.

As there is no development of the eggs in the host it follows that any new infection must come from outside. This seems a very fortunate circumstance, as unless the primary infection is a large one, or the exposure is repeated, there will be no ill effects in the majority of instances.

The parasites, which sometimes number thousands, are said to be found in greatest abundance in the jejunum, secondly in the duodenum, and to a lesser extent in the ileum. This, however, was not my experience (see autopsy). The young worms change their positions frequently, thus allowing a large amount of blood to escape from the wounds deserted by them for new fields.

MODE OF INFECTION. The embryo finds its most favorable habitat in damp soil, hence the vast majority of those infected are either earth eaters or are engaged in work which brings their hands in contact with the earth. Thus miners, gardeners, workers in brickyards, etc., are especially subject to the disease. Sandwith found in 200 cases carefully studied that 190 worked in more or less damp soil with their hands. These people probably convey the infection to their mouths by eating without having previously cleansed their hands. Some, no doubt, are infected through drinking-water, but Giles' experience does not bear out this theory. He examined fifty-six water-supplies of infected villages, and found one doubtful embryo.

F. M. Sandwith² rather supports the idea suggested by Looss, that infection may take place by the penetration of the embryo through the skin, and quotes an experience of that author as follows: Looss accidentally let a drop of water which contained numerous embryos fall upon his hand. Noticing that the spot became red and burned, he let

¹ *Centralbl. f. Bakter. u. Parasitenkunde*, 1897, p. 913.

² *British Medical Journal*, September 14, 1901.

fall another drop, which produced the same result. On careful examination of the area he found countless sheaths which had been discarded by the embryos, indicating that they had thrown off their outer covering and entered the skin. Shortly thereafter he developed symptoms of uncinariosis and found ova in his evacuations. This, however, was not positive proof that he was infected at this time, as he had previously suffered from the disease. Loos then tried several experiments to prove that the embryos could penetrate the skin, one of which was successful. He allowed a drop of infected water to fall on the leg of a boy who was to have the limb immediately amputated. That portion of skin was then hardened, and sections made which showed that the embryos had entered the skin by way of the hair follicles.

Whether the embryos are able, after penetrating the skin, to reach the intestinal tract remains to be proved.

MORBID ANATOMY. "The pathological anatomy is that of extreme anæmia plus the local lesions in the small intestine due to the presence of the uncinaria." There is general bloodlessness of all the tissues; œdema and effusions into the cavities; the fat is usually fairly well preserved; the heart is enlarged, dilated, and may have undergone fatty change; the spleen and kidneys are very often lardaceous and waxy, or the spleen, and still more frequently the liver, presents a certain degree of atrophy. If the autopsy is performed a short time after death the parasites may be seen clinging by their mouths to the mucous membrane of the small intestines. If the body has grown cold they are found in the mucus.

The intestinal wall may be thickened from a catarrhal process. Scattered about the intestine in and under the mucous layer, and visible through the serous and muscular coats, may be seen small ecchymotic spots which, on close inspection, show minute punctures indicating the points where the parasites are attached.

In a few instances parasites have been found in small cavities filled with blood under the mucous membrane. The cavities communicate with the interior of the intestine by small punctures through which the worms have gained an entrance. The intestinal contents may be streaked with blood, and very occasionally there may be extensive hemorrhage.

The intense anæmia is probably due, first, to the actual abstraction and leakage of blood from the mucous membrane of the intestine; secondly, to interference with the proper digestion and absorption of food because of a catarrhal process set up in the intestine by the bites of the parasite; and, lastly, it has been suggested that there is an absorption of poisons produced by the parasite itself or from the intestinal contents through the wounds made in the mucous membrane which aids in causing the anæmia.

There is apparently no actual hæmolysis, as the amount of iron in the liver is not increased, as is the case in pernicious anæmia.

SYMPTOMS. According to Manson¹ "the essential symptoms are those of a progressive anæmia, an anæmia which is usually associated with dyspeptic trouble, but which, in uncomplicated cases, is not associated with wasting. If the progress of the case is unchecked serous effusions in different organs and fatty degeneration of the heart ensue, and death may occur from syncope or intercurrent complications." There may be either diarrhœa or constipation, or the bowels may be regular. Occasionally the evacuations have a reddish-brown shade, due to the admixture of blood, and very rarely there may be distinct hemorrhage from the intestine. The appetite is variable; it may be excessive, capricious, or lost entirely. In advanced cases there is dyspnoea on slight exertion, with palpitation, œdematous swelling of the face, feet, and ankles, and extreme weakness. The urine is usually alkaline or neutral, with a specific gravity of 1010 to 1015; a trace of albumin may be noted in advanced cases. There is often stupidity so dense as to amount, in some cases, almost to weak-mindedness. If a severe infection occurs before puberty that period is apt to be delayed. The temperature is said, as a rule, to be subnormal, though there may be transient flashes of fever. The heart is usually enlarged, and hæmic murmurs are to be heard at the base and over the vessels of the neck. As a rule, there is no enlargement of either liver or spleen. The blood shows the following changes: A marked reduction in the red cells, sometimes below 1,000,000; a proportionate reduction in the hæmoglobin, poikilocytes, and nucleated red cells. Some observers have noted megaloblasts as well as normoblasts.

Of nineteen cases studied in Porto Rico by B. K. Ashford,² fourteen showed normoblasts and seven megaloblasts. The megaloblasts were, however, never in excess of the normoblasts (an excess of megaloblasts is considered quite characteristic of pernicious anæmia).

There is no leucocytosis in uncomplicated cases. Ashford noted an excess of eosinophiles in several cases.

DIAGNOSIS. It has very aptly been said that the way to make a diagnosis of uncinariosis is "first to suspect its presence." Once having had the suspicion aroused by the peculiar anxious expression of the face and the intensely anæmic appearance of the patient, it is a very simple matter to examine the stools for the ova, which are present usually in large numbers. The parasites themselves are almost never seen until after the administration of an anthelmintic.

Both ova and parasites are readily recognized from the cuts in works on clinical diagnosis.

¹ Tropical Diseases.

² New York Medical Journal, 1900.

If a microscopist is not at hand, the administration of thymol to suspects would cause the parasites to appear in the stools, where they may be seen with the naked eye.

The disease bearing the closest resemblance to uncinariosis is pernicious anæmia. The similarity is most striking in the insidious onset, pallor, gastro-intestinal disturbances, circulatory symptoms, weakness, dyspnœa, œdema, tendency to periods of improvement followed by relapses, marked reduction in the number of erythrocytes, presence of poikilocytosis and nucleated red cells, and absence of leucocytosis.

It is said that in uncinariosis the color index is usually low, while in pernicious anæmia it is high—i. e., there is a greater proportional reduction in hæmoglobin in uncinariosis; but this is not always the case. In fact, it is many times almost impossible to differentiate between the two diseases without an examination of the evacuations to determine the presence or absence of the ova.

In leukæmia the blood is very different, as a rule, though similarity has been reported.

True chlorosis is readily differentiated by examinations of the blood and feces; malarial cachexia by the enlarged spleen, presence of plasmodia and absence of the ova of the uncinaria.

In every case of anæmia not attributable to some definite cause the evacuations should be examined microscopically for the ova of the uncinaria.

PROGNOSIS. If the diagnosis is made and proper treatment instituted before the disease has existed long enough to produce serious changes in the tissues and organs of the victim, recovery is prompt; but if the condition goes unrecognized for months or years the result is not infrequently fatal. A small number of cases are very acute and severe, death resulting in a few weeks after the first appearance of symptoms. As a rule, however, the course extends over some years.

Of Sandwith's 400 cases, 89.5 per cent. were cured, or greatly improved; 2.5 per cent. were unimproved, and 8 per cent. died.

In prolonged cases there are usually periods of temporary improvement.

Even if death does not result directly from uncinariosis, those afflicted are especially prone to succumb to some intercurrent disease.

TREATMENT. The recognized treatment is thymol administered as follows: 15 to 30 grains repeated four times at intervals of one and one-half to two hours. If the bowels do not act spontaneously in twelve hours after the last dose, give a purgative, which is usually not necessary. It is well to clear the bowels the day before with a purgative, and place the patient on liquid diet. The fact that thymol is poisonous must not be lost sight of, and the patient should be carefully watched. Since the drug is freely soluble in alcohol, ether, turpentine, chloroform,

oil, glycerin, and certain alkaline solutions, these should be withheld during its administration.

In seven or eight days the stools should again be examined, and if the ova are still present the same programme should be carried out and repeated until all the parasites have been destroyed.

The treatment of the anæmia from uncinariosis is the same as in that secondary to any other cause.

HISTORY IN THE UNITED STATES. It seems very probable that the form of anæmia spoken of by Jean Louis Chabert (1821), James B. Duncan (1850), and others, as existing to a considerable extent in our Southern States was uncinariosis.

The number of authentic cases, however (in which the diagnosis was established by the finding of the ova), reported by observers in the United States is as yet small, and, as far as I have been able to learn, appeared in the following order:

The first by W. L. Blickhahn,¹ of St. Louis (1893); five cases by F. G. Möhlan² (1896); one case by C. H. Tebault, Jr.³ (1899); one by J. H. Dyer⁴ (March, 1901); my own case⁵ (June, 1901); Allyn's case⁶ (July, 1901); one case by Wm. B. Gray⁷ (September, 1901); and one by M. Charlotte Schaefer,⁸ from Texas (October 26, 1901), making twelve in all. Of these twelve cases, five undoubtedly had their origin in the United States; three probably brought the infection from abroad, one from Mexico having been in the United States only a short time when symptoms appeared; in three the data upon which to base an opinion upon this point was not given.

Dr. Gray also reports a second case, a sister of the one noted above, but as neither ova nor parasites were found it does not seem justifiable to include it among the authentic cases.

My friend Dr. Allen J. Smith, of the University of Texas, writes me that he is inclined to believe that the disease is quite common in Texas, since he found the ova in the evacuations of eight out of eighty-eight students. The majority of these cases, Dr. Smith says, have never been outside of the State. His forthcoming paper upon his observations will be very interesting.

G. R., American, aged nineteen years, of Westmoreland County, Va., occupation, light farm work, was admitted to the Garfield Hospital, of Washington, D. C., May 31, 1901. The patient was a small baby at birth, but was said to have developed normally until about three years previous to his admission. This, however, was prob-

¹ Medical News, December 9, 1893.

² Buffalo Medical Journal, 1896-1897.

³ New Orleans Medical and Surgical Journal, 1899-1900.

⁴ Interstate Medical Journal, St. Louis, March, 1901.

⁵ Philadelphia Medical Journal, June 29, 1901.

⁶ American Medicine, July 13, 1901.

⁷ Virginia Medical Semi-Monthly, September 27, 1901.

⁸ Medical News, October 26, 1901.

ably a mistake on the part of his friends, as the boy's development was that of a lad under fourteen. He was small, childish in voice and manner, and the genital organs were infantile. His mentality was much below the average; in fact, he seemed very stupid and dull. He had never had any of the diseases of childhood, nor had he had malaria. About three years prior to his admission it was noticed that the boy was pale; one year later the anæmia became very marked, and there was swelling of the face, abdomen, and feet. There was, however, an improvement until about three months before he came to the hospital.

The chief symptoms were great weakness, dyspnoea on the slightest exertion, and pallor. Contrary to the rule in these cases, there was no complaint of any gastro-intestinal disturbance.

The expression was anxious and dejected. The skin was of a faint yellow tint; the face was bloated, especially about the eyes.

The mucous membranes were extremely pale, the body well nourished, but the muscles flabby. The tongue was clean. The heart's impulse was widely diffused, the apex-beat was in the fifth interspace and almost as far to the left as the mid-axillary line. Over the base there was a loud systolic murmur, the point of maximum intensity being in the pulmonary area. This murmur was also heard in the vessels of the neck. The second pulmonary sound was accentuated. At the apex was heard a systolic murmur which was transmitted to the axilla and to the angle of the left scapula. A faint diastolic murmur was also present over a small area near the apex. The feet and ankles were oedematous. The urine was clear, pale, acid, and showed a specific gravity of 1010. The lowest blood count, June 4th, showed the red cells to be 1,450,000 per cubic millimetre (29 per cent. of normal), the whites 4400 (slightly below the average), hæmoglobin 30 per cent. Dr. J. B. Nichols' report upon the stained cover-slips, June 9th, is as follows: Macrocytes and microcytes a few; polychromatophilia, not observed; poikilocytes, a few; microblasts, normoblasts, and megaloblasts, none.

Leucocytes: Small mononuclear, 15 per cent.; large transitional, 9 per cent.; polynuclear, 71 per cent.; eosinophiles, 5 per cent.; microscopic examination of the evacuations showed innumerable ova of the uncinaria, upon which the diagnosis of uncinariosis was made, and later confirmed by Dr. C. W. Stiles, of the Department of Agriculture, who very kindly saw the case with me and gave valuable advice as to the recovery of the parasites, etc. No Charcot-Leyden crystals were observed in the evacuations, which is of interest in connection with the absence of eosinophilia.

During the early treatment of the case an attempt was made to count all the worms passed; this, however, was abandoned after two weeks, as the number had then run up to 407, and the supply seemed inexhaustible in spite of weekly administrations of thymol in two doses of 30 grains, separated by an interval of two hours and followed by a purgative.

The ova were looked for each week, and gradually decreased, but up to the time of the patient's death, from cerebral hemorrhage, two months after admission, never wholly disappeared.

The general condition improved, but there was always fever, the temperature ranging between 99° and 100°. This continued fever is contrary to the rule.

The last blood count was on August 3, the day before his death, and showed a marked improvement. Reds, 3,033,000 (60 per cent. of normal); whites, 3200; hæmoglobin, 52 per cent.

The appetite was fairly good, the weight had increased, and there seemed to be every reason to hope for a favorable outcome, when, upon August 4, the patient fell unconscious with the symptoms of cerebral hemorrhage, which shortly proved fatal. A similar fate to that of one of Sandwith's cases.

One point which seems worthy of note is the remarkable resistance to treatment shown by the worms. In spite of weekly administrations of thymol in large doses for a period of two months, there were still a few parasites left.

The autopsy was performed by Dr. Nichols.

Necropsy. Body fat and well nourished, but pale. Genitals infantile. The small intestine was of large calibre and had thick walls, especially the duodenum and jejunum. There were no perceptible induration, cicatricial markings, or contractions of the intestinal walls. The valvulæ conniventes were very prominent, and along their bases were rows of very minute points of congestion or hemorrhage; aside from these doubtful points, no distinct hemorrhagic areas, obvious macroscopically, were present. The mucous coat of the bowel was lined with viscid chyme or mucus in which were embedded some uncinaria which were in relatively small numbers. Twenty-nine of the worms were found distributed indifferently along the entire small intestine. A few others may have escaped notice. One of the worms was found with its head attached to the mucosa of the ileum.

The descending colon was studded with small round gray points, apparently enlarged solitary follicles. The mesenteric glands were enlarged. The liver was much enlarged, weighing 2390 grammes; firm, pale in places. The pancreas was firm and hard. The spleen much enlarged, 17x11x5 cm. in size, weighing 410 grammes; congested, fairly firm, and mottled.

The kidney of horseshoe variety, the two organs being united at the lower end; much enlarged, the whole weighing 455 grammes. There was one ureter on each side, both passing down in front of the conjoined kidney. The bladder was distended with urine. The pericardial sac contained a noticeable amount of serum. The heart was much enlarged, weighing 467 grammes. The wall of the left ventricle thickened, the cavity nearly empty, the right containing soft, post-mortem clot. Valves normal. Foramen ovale closed. The left lung much contracted and retracted back into the thoracic cavity, with extensive pleural adhesions easily broken up; weight 273 grammes. Right lung normal, weight 273 grammes.

The brain showed a general subdural hemorrhage over the left cerebrum and a subdural hemorrhage on the right side over the upper portion of the fissure of Rolando. On the right side there was softening of the cerebral substance about the upper end of the fissure of Rolando, giving rise to hemorrhage, the extravasation of blood extending outward under the dura. In the left cerebrum there was similar softening in the occipital lobes with hemorrhage extravasating under the dura. The ventricles contained no blood.

Cultures from liver and spleen were negative.

The only points wherein the pathological findings differed macroscopi-

cally from those usually observed in these cases were in the enlargement of the liver and spleen and in the scarcity of ecchymotic spots in the wall of the small intestine. The latter is readily accounted for by the fact that the parasites had nearly all been destroyed before the patient's death.

The microscopical observations, also made by Dr. J. B. Nichols, were as follows:

Specimens removed from various points in duodenum, jejunum, and ileum showed no material alterations. The subepithelial connective tissue of the mucosa was thickened, studded with lymphoid cells. The glandular epithelium was shrunken and apparently degenerated, but this was probably a post-mortem change. The epithelium cells of the glands of Brunner appeared enlarged and pale.

The colon showed enlargement of the solitary follicles with erosion of the overlying mucosa.

The mesenteric glands presented a normal appearance, the enlargement noted macroscopically being due to general hypertrophy of the gland structures. The spleen presented a normal appearance, without congestion or extravasation of blood; its enlargement consisting in a general hypertrophy. The heart-muscle was normal. The kidney showed congestion, bloodvessels and capillaries being filled with blood-cells. The parenchyma was practically normal, no fatty or amyloid change being observed. *The section from the liver was lost.* The only abnormalities noted were the tendencies to hyperplasia of the lymphatic structures.

LAPAROTOMY FOR PERFORATION IN TYPHOID FEVER.

REPORT OF SIX CASES.

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THE report is based upon the six cases of laparotomy for intestinal perforation during the course of typhoid fever, occurring in Lakeside Hospital since its opening in January, 1898. These were performed in 1899 and 1900 during the writer's service as resident surgeon, and, with the exception of the last case, came under his immediate observation and care. The inclusion of this last case in the series is perhaps of doubtful propriety, since the patient entered the hospital as a case of general peritonitis and was treated as such, the case containing very little of interest with reference to the surgical treatment of typhoid perforation. For the purpose of completeness, however, it has been added.

It is due to the courtesy of the visiting staffs of Lakeside Hospital that the present report has been made possible, and to these gentlemen the writer wishes to acknowledge his obligation.

CASE I.—Male, aged forty-seven years. Moderately severe case. At entrance, leucocytes 5700 per cubic millimetre. Widal reaction negative, diazo positive. Bowels rather loose, no hemorrhage. Symptoms of perforation on the twenty-first day of the disease; abdominal pain, sensitiveness, rigidity, and moderate distention. Rise in pulse and temperature; slight vomiting; leucocytes 4460 per cubic millimetre. Condition unfavorable.

Operation. Five hours after appearance of symptoms. Chloroform. Time, thirty minutes. Right lateral incision. Dark-brown abdominal fluid. Perforation of ileum 8 centimetres from cæcum; closed with continuous Lembert catgut sutures. No threatening perforation found. Irrigation with sterile water; intestines not removed. Gauze drainage. Cultures from abdominal fluid, bacillus coli communis. Death seventy-five hours after operation, apparently from exhaustion.

Autopsy, restricted. Local peritonitis about lesion and gauze packing, catgut sutures absorbed, and perforation patent. Four other neighboring perforations opening into the same area of local peritonitis. Confluent ulcers of cæcum, not perforating. No general peritonitis. Cultures from local peritonitis, bacillus coli communis; from general peritoneal cavity, negative.

C. K., medical No. 769; surgical, No. 909. Service of Dr. H. H. Powell. Male, aged forty-seven years, white, German, married, carpenter. Admitted August 5, 1899; discharged August 15th. Dead.

Family History. Negative.

Previous History. Had always been well, strong, and active. Twelve years ago he had malarial fever; six years ago he had what was taken to be typhoid fever, being ill for nine weeks; no other illnesses recalled; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, loss of appetite, diarrhoea, and fever. Headache was the first symptom, beginning two weeks previous to entrance, and persisting. There was also anorexia and diarrhoea, the latter being quite troublesome. There was some abdominal pain in the region of the umbilicus.

Physical Examination. Patient well-nourished and developed; mind clear; face flushed; tongue coated. Pulse 80, good quality. Temperature 101.8°. Lungs negative. Heart not enlarged; soft systolic murmur at apex. Spleen enlarged on percussion, not palpable. Abdomen soft, slightly sensitive about umbilicus; numerous rose-spots. Leucocytes 5700 per cubic millimetre. Urine, no albumin; faint diazo reaction. Widal reaction negative.

During the next six days the symptoms were those of a moderately severe case of typhoid, the temperature running 101° to 103°, during which time he had six tub baths, four of which were given in the last two days. The bowels remained rather loose. The urine continued to show a diazo reaction, and there was a slight trace of albumin. No subsequent Widal tests were made.

On August 12th, the twenty-first day of the disease, the patient began to have abdominal pain, coming on rather gradually and being first noticed at 6.30 A.M. At first it was confined to the lower half of the abdomen, gradually spreading upward and becoming general. Pain was also noticed at end of penis. He vomited a slight amount of curdled milk at 8 A.M. The temperature at 6 A.M. was 101°, and at 9 A.M. had risen to 102.2°, and the pulse had risen from 72 to 104 during

the same period. The leucocytes at 9 A.M. were 4460 per cubic millimetre, and this unfortunately was the only count made. At 9.30 A.M. the patient was seen with Dr. W. H. Nevison. The abdomen was moderately distended and tympanitic. There was marked sensitiveness over the entire lower half of the abdomen, and the abdominal wall was generally quite rigid. Respirations 28, largely costal. Pulse was 124, dicrotic, readily compressible. The lungs were clear, the heart as previously described. The general condition of the patient was poor, although not altogether unfavorable for operation.

Operation, August 12th, 10.30 A.M., by Dr. W. H. Nevison, five hours after first appearance of symptoms. Anæsthetic, chloroform, preceded by morph. sulph., gr. one-quarter, hypodermically. Time, thirty minutes.

Light anæsthesia. Incision eight centimetres long in right linea semilunaris opposite anterior superior spine. Considerable dark-brown fluid escaped, fecal odor, containing small particles of extraneous matter thought to be feces. Cæcum readily found and ileum carefully inspected for about one hundred and twenty centimetres. A perforation was found about eight centimetres from the cæcum. It was round, about three millimetres in diameter, on the free side of the bowel, and from it fecal matter was escaping. The area of induration surrounding this opening was about two centimetres in diameter. No other perforations were found in the explored ileum or cæcum, nor were any perceptibly thinned areas appreciable, although careful search was made. The perforation was turned in without excision by a double row of continuous Lembert catgut sutures. The ileum was dark and congested, the cæcum somewhat so. Other coils of intestine seen were slightly congested. Appendix normal. Parietal peritoneum seemed but slightly congested. Presence of enlarged glands not observed. No fibrinous flakes seen. There were no adhesions. Abdominal cavity thoroughly irrigated with a large quantity of sterile water, special attention being paid to the pelvis and the lumbar fossæ. The intestines were not handled, but the irrigation was thoroughly accomplished by means of a hard-rubber flushing tube. Site of suture left near the surface, and to it iodoform gauze drainage was placed, brought out through the lower end of the wound. Above this the wound was closed with interrupted silkworm-gut sutures through all layers. During the operation the patient received strychn. sulph., gr. one-fifteenth, hypodermically, and an infusion of seven hundred cubic centimetres of normal salt solution. The radial pulse was very rapid and weak, imperceptible at times. The operation was associated with considerable shock, the patient being in a poor condition on leaving the table.

The subsequent treatment of the case was by strychnine, nutrient stimulating enemata, liquids without milk by mouth, subcutaneous infusions of salt solution, sponge baths, and oxygen. Patient took nourishment well during entire time. He was slightly delirious and semi-conscious at times, but for the most part his mind was clear and active. There was slight abdominal distention, but practically no pain except on the day following the operation, when it was quite marked in the lower half of the abdomen for a short time. The discharge from the abdomen was very profuse during the last twenty-four hours, and was dark colored, which, together with the pain mentioned above, suggested strongly a second perforation in the locality of the gauze drain-

age. The temperature remained about 102.5° to 104° rectal, and the pulse 120 to 130. On August 15th, three days after the operation, the patient began to show evident signs of dissolution, the pulse-rate increased steadily, and the quality became progressively worse. There was some general abdominal distention, most marked in the epigastrium. There was an accumulation of coarse, moist râles throughout the chest, and the respirations were labored. Oxygen was given during the last seven hours. He was unconscious for six hours before death. He died August 15th, at 3.05 P.M., seventy-five hours after operation.

The bacteriological examination of the abdominal fluid at the time of the operation showed on cover-slip preparations a medium-sized bacillus and a small coccus, singly and in pairs. Plate cultures showed a pure growth of *bacillus coli communis*. No growth of the coccus mentioned could be found.

Autopsy. This unfortunately was restricted. About the gauze packing and the site of the lesion in the ileum there was a localized peritonitis, as was to be expected. Opening into this localized area was the original ulcer. The catgut suture had been almost entirely absorbed. There were four small neighboring perforations of the ileum, which also opened into this same localized area. There were several confluent ulcers of the cæcum which had not perforated. There was no general peritonitis. Cultures taken from the area of local peritonitis showed *bacillus coli communis*. Cultures and cover-slip preparations from general peritoneal cavity were negative.

CASE II.—Male, aged seventeen years; severe case. At entrance, leucocytes 5980 per cubic millimetre; Widal reaction negative; diazo reaction negative. Marked constipation; slight distention. Hemorrhage on seventeenth and eighteenth days of disease, preceded by considerable abdominal pain. Symptoms of perforation on eighteenth day of disease; abdominal pain, sensitiveness, rigidity, moderate distention, rise in pulse, temperature unaffected, slight vomiting. Condition very unfavorable.

Operation. Eight hours after appearance of symptoms. Chloroform. Time, twenty-five minutes. Right lateral incision. Dark-brown abdominal fluid with curds. Perforation of ileum twenty-five centimetres from cæcum; one threatening perforation close by; both secured with continuous Lembert silk sutures. Irrigation with salt solution and sterile water; intestines not removed. Gauze drainage. Cultures from abdominal fluid, *bacillus coli communis*. Death sixteen hours after operation; general peritonitis.

Autopsy, restricted. Intestinal suture intact. No other perforations found. General peritonitis. Cultures from general peritoneal cavity, *bacillus coli communis*.

J. B., medical, No. 779; surgical, No. 925. Service of Dr. J. H. Lowman. Male, aged seventeen years, white, American, single, student. Admitted August 13, 1899; discharged August 22d. Dead.

Family History. One brother died of typhoid fever; one sister ill with typhoid fever at present time.

Previous History. Children's diseases. Always strong and active; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, constipation, and general malaise. Headache was the first symptom, and was first noted ten days previously. Bowels constipated, no move-

ment for forty-eight hours before entrance. No hemorrhages. Chilly sensation during last two nights; no distinct rigor. Epistaxis on day of entrance.

Physical Examination. Patient well nourished and developed; athletic. Face flushed; tongue slightly coated, trembles when protruded. Pulse 116, low tension, dicrotic. Temperature 102.8°. Lungs negative. Heart slightly enlarged to the left, loud systolic murmur at apex. Spleen slightly enlarged on percussion, not palpable. Abdomen rather tense, not tympanitic, no especial sensitiveness, no rose-spots recorded. Leucocytes 5980 per cubic millimetre. Urine, no albumin, no diazo reaction. Widal reaction negative.

During the next four days the symptoms were those of a severe typhoid. The nervous symptoms were marked. There was a little abdominal pain on two occasions following tub baths. Baths were given every three hours with but few intermissions. Reactions following baths were not very good. The pulse ranged from 90 to 120, and was of rather low tension. On one day patient complained of considerable pain on inside of right leg, extending down to the knee.

On August 18th, at 8 P.M., following tub bath, there was sudden sharp pain in lower half of abdomen, which continued for some little time, but with rather less severity. The temperature was unaffected, but the pulse-rate was increased somewhat. Sponge baths were substituted. No blood examination was made. During the next day there was slight abdominal pain with an occasional increase in severity. The abdomen was somewhat distended, but this passed off after several free bowel movements. Following this the pain practically disappeared, and the patient was comfortable. There was no vomiting. Pain was present along inner side of right thigh. Leucocytes were 8000 per cubic millimetre in the morning, and 10,000 per cubic millimetre in the afternoon. The next day, August 20th, the abdominal pain had practically disappeared, but there was slight sensitiveness above the pubes and in the left inguinal region. Tub baths were resumed, and were borne rather better than previously. A small amount of old clotted blood was passed in one of the stools.

August 21st, the eighteenth day of the disease, the patient had been rather more comfortable than usual. At 2 A.M., while in tub bath, there was sudden severe pain in the lower abdomen. The abdomen was tense, not distended, and quite generally sensitive. The temperature was unaffected. The pulse rose from 80 to 110, and was not well sustained. At 2.45 A.M. the patient passed about two drachms of blood in a bowel movement. The patient vomited curdled milk at 3 A.M. Abdominal pain continued; sensitiveness was quite marked, more so in the right lower quadrant. Sponge baths were substituted. No blood examination was recorded. At 9 A.M. the patient was seen with Dr. W. H. Nevison. His general appearance was that of a very sick and much reduced man. The finger nails and lips were slightly cyanotic. Pulse was 120, regular, small volume, low tension. Respirations were 24, thoracic. The abdomen showed moderate general distention and tympanites. There was general sensitiveness, slightly more marked in the lower half, and marked muscular resistance. The condition of the patient seemed quite unfavorable for operation.

Operation, August 21st, 9.45 A.M., by Dr. W. H. Nevison, eight hours after perforation was thought to have occurred. Anæsthetic,

chloroform, preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty-five minutes.

Light anaesthesia. Incision in right linea semilunaris nine centimetres long, at level of anterior superior spine. A moderate amount of dark fluid containing particles of curdled milk escaped. Cæcum readily found, and ileum searched for about one metre from cæcum. About twenty-five centimetres from cæcum a perforation was found on the free margin of the gut, three millimetres in diameter; base generally thickened. About six centimetres from this thickened area a very thin centre was made out. Nothing more approaching perforation was found. Perforation was turned in without excision by two rows of running fine silk Lembert sutures for a distance of about two and five-tenths centimetres. The thin ulcer was also turned in with a single running silk suture. About the perforation was a little gray fibrinous deposit. The ileum had lost its gloss in places, but in general looked well. No fibrinous exudate outside the region of perforation. The cæcum and appendix normal. Remaining coils of intestines and parietal peritoneum appeared to be in good condition. No adhesions were found. Abdomen flushed with hot salt solution and sterile water in large quantities, special attention being paid to the dependent portions. This was done by means of a hard-rubber flushing tube. The intestines were not removed from the abdominal cavity. The site of suture left near wound, and to it was placed iodoform gauze drainage, brought out through lower end of wound, above which the incision was closed with interrupted silk-worm-gut sutures through all layers. During operation patient's pulse became very feeble, respiration shallow, color poor. He was given strychn. sulph., gr. one-fifteenth, hypodermically, divided, and a saline infusion of eight hundred cubic centimetres, under which the condition improved somewhat.

The subsequent treatment was by strychnine, nutrient stimulating enemata, subcutaneous infusions, and sponge baths. The reaction to the sponging was very marked, and the baths were given with considerable care. The patient's condition improved slightly for a few hours following the operation, after which time he failed steadily, and died August 22d, at 2 A.M., sixteen hours after the operation.

Bacteriological examination of the free abdominal fluid at operation showed a pure growth of bacillus coli communis on plate cultures. No cover-slip preparations were made.

Autopsy. This was very restricted. The intestinal suture was intact. No further perforation was found. There was a general peritonitis with thin turbid fluid. Cultures from the general peritoneal cavity showed bacillus coli communis; no cover-slip preparations were made.

CASE III.—Male, aged thirty-one years; mild case with a moderately severe relapse. At entrance leucocytes, 5700 per cubic millimetre; Widal reaction positive; diazo reaction negative. Bowels rather constipated; no hemorrhages. Symptoms of perforation on forty-third day of disease, twelfth day of relapse; severe abdominal pain, general sensitiveness, moderate distention, marked muscular rigidity on right side; rise in pulse, temperature unaffected; slight vomiting; leucocytes, 31,600 per cubic millimetre. Condition of shock, but fairly favorable for operation.

Operation. One hour after symptoms. Cocaine solution, 1 per cent. Time, twenty-five minutes. Right lateral incision. Little brownish

abdominal fluid. Perforation of ileum forty-five centimetres from cæcum, closed with double continuous Lambert silk suture; no threatening perforations found. Irrigation with sterile water, intestines not removed. Gauze and glass-tube drainage. Cultures from abdominal fluid showed staphylococcus pyogenes aureus. Death fifty-eight hours after operation; typhoidal toxæmia.

Autopsy, restricted. Sutures intact; no other perforation; no general peritonitis; no peritoneal adhesions except around drainage. Cultures from general peritoneal cavity, negative.

T. D., medical No. 992; surgical No. 13044. Service of Dr. E. F. Cushing. Male, aged thirty-one years, white, Welsh, married, coachman. Admitted January 25, 1900; discharged February 28th. Dead.

Family History. Negative.

Previous History. Always strong and active, no definite diseases recalled; habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, weakness, and lack of energy. Symptoms began ten days before entrance, with malaise. Following this there was severe headache, anorexia, slight cough, and marked weakness. He also had several attacks of abdominal pain described as "colic." The bowels were loose, but there had been no hemorrhages. Patient had vomited several times. There was no epistaxis.

Physical Examination. Well nourished and developed. Mind clear; tongue coated. Pulse 100, full, not dicrotic. Temperature 102°. Lungs and heart negative. Spleen enlarged on percussion, not palpable. Abdomen slightly distended and slightly sensitive on pressure; covered with scattering rose-spots. Leucocytes 5700 per cubic millimetre; hæmoglobin, 90 per cent. Urine, no albumin, no diazo reaction. Widal reaction positive.

For the next three weeks the patient ran a course of mild typhoid fever without complications; the temperature rarely rose above 102.5°, and for a large portion of the time was below 102°. The patient suffered no abdominal pain, and there were no intestinal hemorrhages.

On February 14th, the thirty-first day of the disease, the temperature began to rise, and the patient suffered a well-marked relapse. During the next twelve days the symptoms were somewhat more aggravated than during the primary attack; the temperature ran somewhat higher, though at no time was extreme; on several occasions there was nausea and a limited degree of vomiting. There was no abdominal pain, the abdomen was soft, not distended. Urine contained a faint trace of albumin; diazo reaction positive. Patient was somewhat constipated; there were no intestinal hemorrhages.

On February 26th, the forty-third day of the disease and the twelfth day of the relapse, the patient was given an enema at 9 A.M., which was but slightly effectual. Following this he complained of feeling a little distended, and there was slight abdominal pain. This pain, however, soon passed off, and at 10 A.M. the patient was found sleeping quietly; pulse 100. When seen fifteen minutes later he was in a condition of evident shock; he was cyanotic and trembling; the pulse had reached 120, and was very compressible; temperature unaffected. There was quite severe general abdominal pain, the abdomen was somewhat distended, but not very sensitive. The liver appeared to be pushed upward. The patient vomited once, about an ounce of greenish

fluid. Leucocytes 31,600 per cubic millimetre. The patient was seen with Dr. D. P. Allen at 10.30 A.M. Cyanosis was still present. Pulse was 130, low tension. Respirations were 30, thoracic. The abdomen was moderately distended; there was general, not localized, sensitiveness, and there was marked muscular rigidity on the left side; peristalsis was present. The heart sounds were weak, the lungs clear. Patient's condition was fairly favorable for operation.

Operation, February 26th, 11 A.M., by Dr. D. P. Allen, one hour after appearance of symptoms. Local anæsthesia, cocaine solution, 1 per cent., preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty-five minutes.

Incision six centimetres long in right linea semilunaris opposite anterior superior spine, carried into peritoneal cavity without causing the patient pain. Abdominal cavity contained a little brownish fluid. Cæcum readily found, normal. Appendix normal. Perforation of ileum about forty-five centimetres from cæcum, on free border, about two millimetres in diameter. Perforation closed without excision by two rows of continuous silk Lembert sutures. No other perforations or threatening perforations found, but the ileum was not examined beyond the lesion. Abdomen irrigated with large quantities of hot sterile water with flush tube, intestines not being removed; special attention was given to the lumbar fossæ and pelvic cavity. Ileum considerably congested. Peritoneal surfaces otherwise apparently unaffected. No fibrinous flakes. No adhesions seen. Glass-tube and gauze drainage placed to seat of suture, on either side of which the wound was closed with interrupted silkworm-gut sutures. The patient's condition did not appear to be affected by the operation; the pulse was as good as before operation. Very little pain was experienced except during irrigation, when it was moderate.

Subsequent treatment of the case was by strychnine, nutrient and stimulating enemata, subcutaneous saline infusions, sponge baths, and liquids by mouth. The patient appeared to be unaffected by the operation. During the next two days the course was that of an increasingly severe typhoidal toxic condition with the accompanying evidence of exhaustion during the second day. There was failing pulse, marked trembling, moderate delirium at times. The abdomen was only slightly distended, was generally tympanitic, and was but moderately sensitive. At times there was sharp abdominal pain. There was but little discharge from the wound. Urine showed a large trace of albumin, and contained an abundance of hyaline and granular casts. The patient died at 9.10 P.M., February 28th, fifty-eight hours after operation.

Bacteriological examination of the free abdominal fluid at the time of operation showed on cover-slips a few cocci and numerous bacilli. Tube cultures showed a pure growth of *staphylococcus pyogenes aureus*.

Autopsy, restricted. The intestinal sutures were intact, the perforation being firmly closed. No additional perforations found. There was no general peritonitis. There were no peritoneal adhesions except those about the gauze packing. Cultures and cover-slip preparations from general peritoneal cavity were negative.

CASE IV.—Female, aged sixteen years; very severe case. At entrance leucocytes 9932 per cubic millimetre; Widal reaction positive; diazo reaction positive. Bowels moved readily with enema, no hemorrhages. Marked delirium. Symptoms of perforation on the

fifth day of the disease; abdominal pain, rigidity, distention; rise in pulse and temperature; no vomiting; leucocytes 15,080 per cubic millimetre; general constitutional change marked. Exploration advised.

Operation. About four hours after first appearance of symptoms. Cocaine solution $\frac{1}{2}$ per cent. Time, twenty minutes. Short right lateral incision. No perforation; peritoneal cavity apparently normal. Wound sealed with collodion dressing. Cover-slips and cultures from abdomen negative. Medical treatment uninterrupted by operation. Subsequent course of fever severe; convalescence slow.

H. B., medical No. 1035. Service of Dr. H. S. Upson. Female, aged sixteen years, colored, American, single, cleaner. Admitted January 4, 1900; discharged March 24th. Cured.

Family History. Negative.

Previous History. Children's disease; no other illness recalled; habits good.

Present Illness. Patient was transferred from gynecological service complaining of general weakness. Two days before patient complained of general malaise and ached all over. There had been a little cough, occasional epistaxis, anorexia, and vomiting with moderate frequency. Bowels regular.

Physical Examination. Patient well nourished and developed; mind clear; tongue coated. Pulse 100, fair strength and volume, slightly dicrotic. Temperature, 104.2°. Lungs and heart negative. No enlargement of spleen perceptible. Abdomen soft, no rose-spots. Leucocytes, 9932 per cubic millimetre; hæmoglobin, 85 per cent. Urine, albumin faint trace; diazo reaction present. Widal reaction positive.

From the start the symptoms were those of a very severe typhoidal infection. The temperature was high, averaging 104° to 105°. The reaction from the tub baths was not satisfactory, and the drops in temperature very moderate. The pulse for the first three days averaged 110 to 120, but was of fair quality. There were marked nervous symptoms, the patient being delirious most of the time and rarely giving an intelligent answer to questions. The amount of stimulation required was considerable.

On January 6th the patient complained somewhat of abdominal pain, and there was slight distention but no rigidity. On January 7th, the fifth day of the disease, the patient's condition began to get markedly worse in the early part of the evening. The patient was delirious, so that her subjective sensations were of little avail in diagnosis. In spite of the delirium, however, she complained of abdominal pain, which it seemed must be at least moderately severe to arrest her attention at all. The abdomen was moderately distended, and was everywhere tympanitic. There was a considerable degree of muscular rigidity. No definite sensitiveness could be localized. The pulse had risen from 120 to 140, was weak and dicrotic. The temperature had risen from 102.8° at 5 P.M. to 105.5° at 8 P.M. The leucocytes at 8 P.M. were 15,080. The patient was seen about 8 P.M. with Dr. G. W. Moorehouse, and essentially the same condition found as noted above. A definite diagnosis of perforation could not be made. The constitutional effect, however, from some cause or other had been very marked, and owing to the delirious condition of the patient and apparent insensibility to ordinary degrees of pain it was felt that a perforation under

the circumstances would be very easily overlooked, and that the physical condition was such as one might readily expect in a patient so severely ill if a perforation had actually occurred. On account, therefore, of the possibility of overlooking a perforation, together with the degree of abdominal symptoms afforded by the patient in even such a delirious condition, an exploration under local anæsthesia was advised. The risk of such an operation was very slight, whereas the danger from an unrecognized perforation was of the gravest possible character.

Operation, January 7th, 9.45 P.M., by Dr. C. E. Briggs, about four hours after the appearance of symptoms. Local anæsthesia, cocaine solution, $\frac{1}{2}$ per cent., preceded by morph. sulph., gr. one-quarter, hypodermically. Time, twenty minutes.

Incision three centimetres long in right linea semilunaris opposite anterior superior spine. On opening the peritoneum no free fluid was found. There was no injection of the cæcum or adjoining portions of the ileum. No adhesions were found, and the peritoneal cavity appeared to be perfectly normal. Cover-slip preparations from right iliac fossa and pelvis showed no organisms. Wound closed with buried silk sutures in layers. A collodion dressing was applied, consisting of several alternate thin layers of sheet wadding and collodion, making a firm and absolutely impervious dressing. Condition of patient apparently unaffected by operation. Permission given to continue baths within four hours if desirable. Patient remained on the medical service.

The subsequent history of the case is of little interest from the present stand-point, except for one attack of abdominal pain. She ran the course of a very severe typhoidal infection, the toxic symptoms being so severe on several occasions that her recovery seemed hopeless. The temperature remained persistently high for nearly ten days, and was but slightly affected by tub baths. On only one occasion, January 20th, the eighteenth day of the disease, was there any subsequent suggestion of an abdominal complication. On this day she was awakened at 2 A.M. by severe abdominal pain. The pulse was but slightly affected. The temperature was unchanged. The abdomen was soft and not sensitive, and there was very slight sensitiveness on deep pressure in right lower quadrant. The pain could not be definitely localized, although the patient was perfectly rational, and it passed off within a short time. The leucocytes, however, at 2.30 A.M. had risen to 50,660 per cubic millimetre; two days before the leucocytes were 8060, and five days later 9600 per cubic millimetre. The convalescence was slow and uneventful. Following the operation, baths were given as required without reference to the incision. The incision healed by first intention. The patient was discharged March 24th, cured.

Bacteriological Examination. Cultures taken from the right iliac fossa and pelvis showed no organisms in cover-slip preparations, and there was no growth on cultures.

CASE V.—Male, aged seventeen years; mild case. At entrance leucocytes 4900 per cubic millimetre; Widal reaction positive; diazo reaction positive. Bowels rather constipated, no hemorrhages. Symptoms of perforation on twentieth day of disease; severe abdominal pain, general sensitiveness, slight distention, marked muscular rigidity in lower half of right side; rise in pulse; fall in temperature of 1° ; no vomiting; leucocytes 9200, 13,000, 13,200 per cubic millimetre. Condition of patient favorable for operation.

Operation. Three hours after first appearance of symptoms. Cocaine solution, 1 per cent.; chloroform. Time, fifty-five minutes—forty minutes under chloroform. Right lateral incision. Yellowish-gray abdominal fluid. Perforation eleven centimetres from cæcum, closed with continuous Lambert silk sutures; no threatening perforations found. Irrigation with sterile water. Intestines not removed. Glass-tube drainage from pelvis. Cultures showed bacillus mucosus capsulatus. Recovery.

C. W., medical No. 1208; surgical No. 1530. Service of Dr. E. F. Cushing. Male, aged seventeen years, white, American, single, laborer. Admitted June 16, 1900; discharged August 19th. Cured.

Family History. Negative.

Previous History. Has always been well and strong. A few years ago had erysipelas, and later a fever the nature of which he did not know. Habits good; no venereal history.

Present Illness. Patient entered hospital complaining of headache, backache, and pain in stomach. Two weeks previously symptoms began with general aching sensation and tired feeling. Shortly after this he began to have nausea, and there was anorexia. Epistaxis was frequent. Bowels rather costive. Had been in bed one week.

Physical Examination. Fairly well nourished and developed; mind clear; face flushed; lips dry; tongue coated. Pulse 80, full, but readily compressible, dicrotic. Temperature 101.8°. Lungs and heart negative. Spleen apparently not enlarged. Abdomen not distended, soft, and showing numerous rose-spots. Leucocytes 4900 per cubic millimetre; hæmoglobin, 85 per cent. Urine, albumin faint trace; diazo reaction present. Widal reaction positive.

During the next five days the symptoms were those of a rather mild case of typhoid fever, the temperature coming down gradually, with good drops following tub baths. There was no diarrhoea, no intestinal hemorrhages. The abdomen was soft and flat. The progress of the case was very satisfactory.

On June 22d, the twentieth day of the disease, the patient had been as usual all through the day. No tub baths had been given. At 6.30 P.M. he was seized with a sharp abdominal pain which was not definitely localized. When seen a few minutes after this there was no abdominal distention, but marked muscular rigidity and general sensitiveness. The pulse was 76 and of good quality; the temperature 102.8°, which was 1° higher than at 4 P.M. The leucocytes at 6.45 P.M. were 9200 per cubic millimetre. The pain was sufficient to make the patient groan audibly. A small enema with turpentine, two drachms, was given, which was followed by a light-yellow stool and a small amount of gas, but afforded no relief from the pain. Morph. sulph., gr. one-quarter, was given hypodermically. At 7 P.M. the pulse was 88, and the temperature had fallen 1°. At 7.30 P.M. the patient was seen with Dr. J. L. Martin. The pulse was 110, low tension, readily compressible. The patient had a dusky appearance, and there was marked cyanosis of the lips and finger-nails. Respirations were shallow, mostly thoracic. The abdomen was not distended, but there was marked rigidity over the lower half, and was a little more evident on the right side; there was considerable general sensitiveness. The patient still complained of moderately severe pain in lower half of abdomen. At 8 P.M. the pulse had risen to 120. The cyanosis was slightly less marked,

but otherwise the condition was about the same. The leucocytes were 13,000 per cubic millimetre. At 8.30 P.M. the leucocytes were 13,200 per cubic millimetre.

Operation, June 22d, 9.30 P.M., by Dr. C. E. Briggs, three hours after first appearance of symptoms. Local anæsthesia, cocaine solution, 1 per cent., preceded by morph. sulph., gr. one-quarter, hypodermically; chloroform. Time, fifty-five minutes, forty minutes of which were under chloroform.

On reaching the operating-table the abdominal pain was still present, and there was slight distention. The cyanosis was considerably less. Pulse 120. Under local anæsthesia an incision was made six centimetres long in right linea semilunaris. Although the patient suffered no pain, he became very nervous and quite beyond control, and light chloroform anæsthesia was substituted. Owing to the boy's nervous condition the opening of the abdomen under cocaine required great patience, consuming fifteen minutes. On opening the peritoneum a moderate amount of thin, yellowish-gray fluid escaped. Cæcum readily found, ileum reached, and perforation found on its free border about eleven centimetres from the ileocæcal valve. It was about two millimetres in diameter and exuded bowel contents when pressed. About it the bowel was red and thickened for about two centimetres in diameter. The perforation was wiped off with bichloride and turned in with two rows of running fine silk Lembert sutures for a length of three centimetres. Over these were placed three interrupted Lembert sutures to relieve the strain, as the bowel was very friable. The cæcum and ileum for about one metre were searched with eye and finger for perforations or thin ulcers, but nothing suggesting a threatening perforation was found. The ileum for about half this distance was injected and seemed somewhat thickened. Appendix normal. No fibrinous flakes were observed. No adhesions were found. Incision enlarged to nine centimetres. Abdomen was thoroughly irrigated with a large amount of sterile water, with special reference to the pelvis and flanks; the intestines were not removed. The pelvis contained a large amount of thin, purulent-looking material, rather different in appearance from that seen on opening the abdomen. Examination of cover-slip preparations from this fluid showed numerous large and small bacilli and a few small cocci. Intestinal coils near pelvis were injected, but remainder of abdomen appeared nearly normal. Abdomen flushed till perfectly clear. Glass drainage-tube placed in pelvis, brought out through lower end of wound, above which the wound was closed with interrupted silk-worm-gut sutures. The site of perforation was left immediately below the incision. During the operation the patient's pulse remained at 120, good quality, rising a little during recovery from anæsthesia. Patient was but very slightly under chloroform, and was moving most of the time except during closure of the abdominal wall. The forty minutes under chloroform were largely spent in careful examination of the bowel, and in very thorough irrigation of the peritoneal cavity.

The subsequent treatment of the case was by nutrient and stimulating enemata, strychnine, sponge baths, and liquids by mouth. During the first night there was very profuse sweating, which was relieved by atropine. The day after the operation, June 23d, the patient began to vomit a dark-brown fluid, and this continued for several hours, the patient's general condition becoming considerably affected. The

stomach was washed out, about three pints of dark-greenish fluid being evacuated, after which the vomiting ceased, and there was immediate and steady improvement.

From this time on the patient made a progressive and uninterrupted recovery, the pulse coming down to between 80 and 90 on the second day after the operation, and the temperature falling gradually to 99° by the end of the first week. The leucocytes on the second, sixth, and eighth days after operation were 15,400, 5400, 5600 per cubic millimetre, respectively. The discharge from the wound was moderate. The glass tube was gradually withdrawn, being replaced by gauze packing on the eighth day, which was entirely omitted four days later. The wound was very indolent, and there was considerable sloughing, but no active suppuration. The wound was entirely healed four weeks after operation, but owing to the persistence of a slight degree of fever—99° to 99.5°—the patient was kept in bed nearly two weeks longer. The cause of this temperature was never discovered. The patient was discharged August 19th, fifty-eight days after operation, in excellent condition.

Bacteriological Examination. Cover-slips and cultures taken from the abdominal fluid when the peritoneum was first opened were negative. Cover-slips taken from the seat of the ulcer and from the turbid pelvic fluid mentioned showed medium-sized bacilli and a few small cocci. Plate cultures from these two localities showed bacillus mucosus capsulatus. Cultures taken from the bottom of the drainage-tube four days after operation gave a pure growth of the same organism.

CASE VI.—Male, aged twenty-eight years. Patient entered hospital with general peritonitis; no history obtainable. At entrance leucocytes 10,600 per cubic millimetre; Widal reaction positive; diazo reaction positive. Diarrhoea followed by constipation; no intestinal hemorrhages. Marked abdominal distention, rigidity, pain, and sensitiveness. Date of perforative symptoms not determined.

Operation. Period after perforation not known. Ether. Time, thirty minutes. Right lateral incision. Turbid abdominal fluid. Perforation of ileum thirty centimetres from cæcum; closed by continuous Lembert catgut sutures. Irrigation with sterile water; intestines not removed. Gauze and glass-tube drainage. Cultures from abdominal fluid, unidentified bacillus, probably bacillus coli communis, or bacillus mucosus capsulatus. Death four hours after operation, general peritonitis.

Autopsy. Intestinal sutures intact, no other perforations. General peritonitis. Cultures from heart, lungs, kidneys, and brain, bacillus mucosus capsulatus; from general peritoneal cavity, negative.

J. K., medical No. 1302; surgical No. 1660. Service of Dr. E. F. Cushing. Male, aged twenty-eight years, white, German, single, laborer. Admitted August 20, 1900; discharged August 21st. Dead.

History was obtained subsequently for purposes of record, but no information was at hand at time of entrance for aid in diagnosis.

Family History. Negative.

Previous History. No former illnesses known. Habits good.

Present Illness. Patient entered hospital complaining of abdominal pain. The symptoms began with headache eight days previously. For the last six days he had abdominal pain with some distention. There was diarrhoea at first, but during the last few days there was but one

movement. There was no vomiting or epistaxis. No fever was noted. Was in bed only part of this time. Abdominal pain had been constant.

Physical Examination. Patient entered in the evening, and no history was available to aid in diagnosis. Moderately well developed, poorly nourished. He had the appearance of a very sick man. Tongue coated and dry. Pulse 120, irregular, but moderately strong. Temperature 100.8°. Lungs: on right side below clavicle there was slightly increased vocal fremitus; otherwise negative. Heart appeared not enlarged, sounds weak. Liver dulness from fourth to sixth rib in mammary line. Spleen: dulness anterior to about mid-axillary line; palpation impossible on account of distention. Abdomen distended, tympanitic, moderately rigid, sensitive to pressure, more marked in lower half and possibly slightly increased on right side; no rose-spots; doubtful dulness in both flanks. Leucocytes 10,600 per cubic millimetre at 9 P.M. Urine: albumin $\frac{1}{2}$ per cent.; diazo reaction faint. Widal reaction positive; both of these examinations made the following morning.

The case was thought to be one of general peritonitis from perforation of a typhoid ulcer, although the existence of typhoid fever was not absolutely determined until the next morning. Circumstances, however, made an immediate operation impossible. During the night he was very actively stimulated. The next morning the Widal reaction determined the diagnosis. The case was seen by Dr. D. P. Allen about 9 A.M. The abdominal distention was extreme. There was general tympany, marked muscular rigidity, and general sensitiveness. The patient was in a condition of profound shock. Pulse 140 and very poor quality; temperature 105.6°; respirations 40. The case was considered practically hopeless.

Operation, August 21st, 10.30 A.M., by Dr. D. P. Allen. Time, after onset of symptoms not known. Anæsthetic, ether. Time, thirty minutes.

Incision seven centimetres long in right linea semilunaris opposite anterior superior spine. On opening the peritoneum a moderate quantity of straw-colored fluid and thin pus escaped, containing fecal matter. Perforation of the ileum was found on the free border about two millimetres in diameter, about thirty centimetres from the cæcum. This was closed with two rows of running catgut sutures, strengthened by four interrupted silk sutures. There was considerable agglutination of the intestinal coils in the neighborhood of the perforation, together with congestion of the remaining coils of intestines and the parietal peritoneum. No other perforations found. A considerable amount of thin pus was found high up under the liver and a considerable amount in the pelvis. The abdomen was thoroughly irrigated with sterile water, the intestines not being removed. Gauze and glass-tube drainage from pelvic cavity and subhepatic region. Pulse during the operation 180, very weak, often imperceptible. The patient was highly stimulated with strychnine and subcutaneous saline infusions. There was no reaction, however, and he died at 3 P.M., four hours after the operation.

Bacteriological Examination. Cover-slips from the abdominal fluid showed cocci and small bacilli. Tube cultures showed a pure culture of bacillus coli communis or bacillus mucosus capsulatus, the exact identity apparently not having been established.

Autopsy, complete, No. 194, August 26th. Anatomical diagnosis, typhoid fever, ulceration, and perforation with general peritonitis; apical tuberculosis and hypostatic pneumonia; acute splenic tumor. Special lesion; thirty centimetres above cæcum there was a perforating ulcer of the ileum, closed with catgut sutures, which were still intact; there were several other ulcers with very thin bases in the neighborhood, but no perforations. Cultures from heart, lungs, kidneys, and brain showed bacillus mucosus capsulatus; from general peritoneal cavity, negative, owing probably to the recent irrigation.

GENERAL CONSIDERATIONS. That all six cases were typhoid fever is readily established. The Widal reaction was positive in four cases—III., IV., V., and VI. In the first two cases of the series the Widal reaction was negative at entrance, and for some reason was not recorded later. The course of the disease was perfectly typical in all instances. A perforation was found in all cases except Case IV., and this case had a positive Widal reaction. Autopsy in the fatal cases—I., II., III., and VI.—showed typical bowel lesions.

The cases with one exception—Case V.—were all severe infections, and this severity, not the complication, was held responsible for the death in two cases—I. and III. One case—III.—occurred during a moderately severe relapse, following a rather mild primary course.

The bowels were constipated in three cases—II., III., and V.—in the first of which the condition was very marked. In one case—Case I.—there was moderate diarrhœa. In Case IV. the bowels moved easily with enema, neither marked constipation nor diarrhœa being observed. In Case VI. the bowels were loose at first, followed by rather aggravated constipation.

Intestinal hemorrhages occurred in only one instance—Case II. In this case it was present on the seventeenth and eighteenth days of the disease, was slight in amount, but was accompanied with considerable abdominal pain. The last hemorrhage was apparently coincident with the perforation.

The ages were sixteen, seventeen, seventeen, twenty-eight, thirty-one, and forty-seven. There was but one female in the series—Case IV. She was colored, and no perforation was found. The remaining cases were males, and white. In Finney's series but one case occurred in a negro, and that was fatal.

SIGNS AND SYMPTOMS. As compared with the large proportion of the more extended collections, the cases presented show a remarkably distinct set of symptoms. In Case VI. the patient entered with unmistakable signs of general peritonitis, and the primary symptoms of perforation in this case were not observed. The discussion of the signs and symptoms is restricted, therefore, to the first five cases.

The date of appearance of symptoms was the fifth day in Case IV.; the eighteenth day in Case II.; the twentieth day in Case V.; the

twenty-first day in Case I.; the forty-third day, the twelfth day of a relapse, in Case III. In Case VI., judging from the probably inaccurate history, the perforation occurred early, probably on the fifth or sixth day.

The abdominal symptoms were quite well marked in all cases. Pain appeared suddenly in three cases—Cases II., III., and V., and came on gradually in two cases—Cases I. and IV. It was confined to the lower half of the abdomen in three cases—Cases I., II., and V., but in Case I. it eventually radiated to the upper half and became general. In Case III. it was general, and in Case IV. it appeared to be general, but the delirious condition of the patient made accurate observation impossible. The pain was severe in three cases—Cases II., III., and V.—and moderate in two cases—Cases I. and IV. In Case I. pain also radiated to the penis, which was the only instance. Sensitiveness to pressure was slight in two cases—Cases II. and IV.; moderate in Case III.; marked in Cases I. and V. It was general in three cases—Cases III., IV., and V., and was confined to the lower half of the abdomen in two cases—Cases I. and II. Increased sensitiveness in the right lower quadrant so frequently spoken of was not observed.

Muscular resistance was a very marked sign in all but one of the cases—Case IV. In this instance the resistance was but moderate, and no perforation was found. The resistance was general in three cases—Cases I., II., and IV., and was confined to the right half of the abdomen in one case—Case III. In Case V. it was confined to the lower half of the abdomen, and was considerably more marked on the right side. Distention was observed as moderate in Cases I., II., III., and IV., but was present to a slight degree only in Case V. Tympany to a greater or less extent was observed in all of the cases. Slight vomiting was observed in Cases I., II., and III., and was absent in Cases IV. and V.

The pulse showed a marked rise in all instances—20 to 50 beats. The quality of the pulse also became progressively worse with the increase in the number of beats, and was a very valuable sign. In Case IV., where no perforation was found, the pulse rose very gradually from 120 to 140. The marked severity of the case, however, made all of the signs and symptoms in this instance particularly difficult to estimate with much accuracy. The temperature was unaffected in two cases—Cases II. and III.—and in two cases—Cases I. and IV.—there was a distinct rise, 101° to 102.2° , and 102.8° to 105.5° , respectively. In but one case—Case V.—was there a fall in temperature, and in this instance of but 1° only, 102.8° to 101.8° . The respiratory rate was markedly increased. The nature of the respirations, however, was noted as largely thoracic in all the cases. This was especially marked, together with a very shallow quality, in Case V.

The condition of general systemic shock, as observed shortly before operation, was very marked in Case II., and was present to a considerable degree in Case IV. It was noted as moderate in Cases I., III., and V. Cyanosis, especially of the lips and finger-nails, was marked in Case V., moderate in Case III., slight in Cases I. and II., and absent in Case IV.

The white blood count as recorded showed a distinct rise in all but one instance—Case I. The records are as follows: Case I., entrance, 5700 per cubic millimetre; perforation, six days later, 4460 per cubic millimetre. Case II., entrance, 5980; perforation, eight days later, no count recorded. Case III., entrance, 5700; perforation, thirty-two days later, 31,600. Case IV., entrance, 9932; symptoms of perforation, two days later, 15,080. Case V., entrance, 4900; perforation, five days later, 9200, 13,000, 13,200. Case VI., entrance, 10,600. It is to be noted in Case IV. that subsequent to the operation the leucocytes on one occasion rose from 8060 to 50,660, associated with considerable abdominal pain, but no other constitutional effect.

THE CONDITION OF PATIENT AT OPERATION. The cases with but one exception—Case V.—were rather unfavorable to operation, two being quite so, and one practically hopeless. As this is regarded as of considerable importance, the conditions will be given in more detail: Case I. The patient's condition was poor, although not absolutely unfavorable to operation. The perforation occurred on the twenty-first day of the disease, in a moderately severe case, before the patient had begun to recover at all from the effects of his illness. He was weak and emaciated, and had comparatively little recuperative power. Case II. This case was quite unfavorable for operation. The patient was a strong, athletic boy, but the case had run a severe course, and the perforation occurred on the eighteenth day during the very height of the disease. The patient was weak and exhausted, and the rapidity with which he succumbed to the peritoneal infection was not surprising. Case III. This case was somewhat more favorable, although not very promising. The perforation occurred during the height of a rather severe relapse, being the forty-third day of the disease, the twelfth day of the relapse. He was quite weak, but appeared at the time to have a fair degree of strength. Case IV. The case was desperately sick at the time perforation was feared, but the symptoms appeared so early, the fifth day, that the patient's constitution had not been badly reduced. On this account it is believed she would have stood fairly well a more extensive operation. Case V. The condition of this patient was very favorable for operation, in marked contrast to the other cases. The case was mild, and when the perforation occurred, the twentieth day, the temperature had begun to come down; he was not badly reduced. Case VI. The case, as stated, was in *extremis* at entrance, and was regarded as practically hopeless.

DURATION OF SYMPTOMS. The lapse of time between the first appearance of symptoms and the operation was short in all but one instance—Case II. In Case III. the operation was performed one hour after the appearance of symptoms. Cases V., IV., and I. were operated upon within three, four, and five hours, respectively, although the exact time of suspected perforation in Case IV. was somewhat indefinite, owing to the delirium of the patient. Case II. was opened eight hours after definite symptoms first appeared; three days previously the patient had had abdominal pain following a tub bath, which grew less the next day, disappeared entirely the day before active symptoms, and was entirely unassociated with constitutional signs suggesting perforation. The duration of symptoms in Case VI. could be only roughly estimated, as perforation occurred before entrance, but it was probably twenty-four to forty-eight hours.

OPERATION. The technique of the operations presented very little that was distinctive. The anæsthetic was general in four cases, local in two cases. Chloroform, very lightly given and preceded by morph. sulph., gr. one-quarter, was administered in Cases I., II., and V. Ether was similarly given in Case VI. Local anæsthesia, cocaine solution, $\frac{1}{2}$ and 1 per cent., preceded by morph. sulph., gr. one-quarter, was employed in Cases III. and IV. In Case V. the abdomen was entered under cocaine, but the nervous condition of the patient required the administration of chloroform.

The incision in each instance was in the right linea semilunaris opposite the anterior superior spine, three to nine centimetres long. In each instance, except Case IV., the cæcum was readily found, the ileum, cæcum, and appendix examined. In Case IV. the peritoneal cavity was so visibly normal that the ileum was not withdrawn. This, however, was considered a fault in the operation. An examination of the ileum was made for about one metre in Cases I., II., and V., but in Cases III. and VI. the inspection was not carried beyond the perforation. In all cases careful search was made for thin areas, threatening perforations, as well as for complete perforations. The perforation was excised in no instance. The closure of the perforation was by two rows of continuous Lembert sutures in each case, in two of which—Cases V. and VI.—these were strengthened by another line of interrupted silk sutures. The suture material was catgut in Cases I. and VI., and silk in the remainder. The irrigation was with very large quantities of sterile water, by means of a large flushing tube. With this tube all parts of the peritoneal cavity were readily reached, special attention being given to the lumbar fossæ and pelvic cavity. Only the portion of the ileum examined was removed from the abdomen, and an effort was made to handle the intestines as little as possible. Drainage was used in all cases except the exploration—Case IV. Iodoform gauze

alone was used in Cases I. and II.; glass-tube and iodoform gauze in Cases III. and VI., and a single glass drainage tube from the pelvis in Case V. The site of the suture was left close to the abdominal wall in all instances. The wounds were all partially closed with interrupted silkworm-gut sutures through all layers, except Case IV., which was completely closed with interrupted buried silk sutures. The time of operation was twenty minutes in one case, twenty-five minutes in two cases, and thirty minutes in two cases. In Case V. fifty-five minutes were consumed, fifteen minutes in going through the abdominal wall under cocaine, and forty minutes, or the remainder of the operation, under chloroform. The condition of the patient was apparently unaffected by the operation in Cases III. and IV.; it was excellent in Case V.; was poor in Cases I. and II., and very bad in Case VI.

PATHOLOGY. Pathological observations of the cases are of special interest, and it is to be greatly regretted that more complete autopsies could not be obtained.

There was a moderate degree of peritonitis at the time of operation in all the cases of perforation, judging from the amount of free fluid in the abdominal cavity. In Case I. there was considerable dark-brown fluid with fecal odor; in Case II. a moderate amount of dark fluid; in Case III. a small amount of brownish fluid; in Case V. a moderate amount of thin, yellowish-gray fluid, and in the pelvis a large amount of thin, purulent-looking fluid; in Case VI. a moderate amount of straw-colored fluid with a large accumulation of thin, purulent fluid under the liver and in the pelvis.

The perforation was single in each instance, and occurred on the free border of the ileum. In only one case—Case II.—was a threatening perforation found. The perforations were all small, two to three millimetres in diameter, and were surrounded by an indurated friable base about two centimetres in diameter in several instances. In one case—Case II.—there was a small amount of fibrinous exudate in the neighborhood of the perforation. The perforations were eight, eleven, twenty-five, thirty, and forty-five centimetres from the cæcum in Cases I., V., II., VI., and III., respectively.

The ileum, aside from the perforation, was altered in each instance, ranging from slight congestion to a thickened dark appearance. The appendix was normal in all cases. The cæcum was appreciably congested in Case I. only. The remaining coils of intestines were slightly congested in Case I. In Case V. those lying in the pelvis were considerably congested; in Case VI. there was a marked congestion, and agglutination of the coils in some places. The parietal peritoneum was appreciably congested in Cases I. and VI. only. In none of the cases were there fibrinous flakes in the peritoneal cavity, except for a small amount of deposit about the perforation in Case II. No adhesions

were observed in any of the cases except in Case VI. In Case IV., in which the symptoms appeared on the fifth day and no perforation was found, the abdominal cavity was perfectly normal so far as could be observed.

Bacteriology. Bacteriological examination of the free abdominal fluid was made in all cases, but in only one instance was a culture taken from the perforation. Cover-slip preparations from Case I. showed a medium-sized bacillus and a small coccus; plate cultures grew bacillus coli communis only. Case II. showed a pure growth of bacillus coli communis in plate cultures. Case III. showed in cover-slips a few cocci and numerous bacilli; in plate cultures, a pure growth of staphylococcus pyogenes aureus. Case IV., in which no perforation was found, cover-slips and cultures were negative. Case V., cover-slips and cultures from fluid on first opening the abdomen were negative; cover-slip preparations from the perforation and the turbid pelvic fluid showed numerous medium-sized bacilli and a few cocci. A plate culture from these two localities gave a pure growth of bacillus mucosus capsulatus; a tube culture taken from the bottom of the drainage-tube in the pelvis four days after operation showed a pure culture of the same organism. In Case VI. cover-slip preparations showed cocci and small bacilli, the nature of which was not definitely determined, but was apparently bacillus coli communis or bacillus mucosus capsulatus.

Autopsies, complete, Case VI., or restricted, Cases I., II., and III., were made in the four cases of death. General peritonitis was present in two cases only—Cases II. and VI.—in the other two cases the general peritoneal cavity being free from fluid and adhesions, and the cultures from the same being negative. Case I., death seventy-five hours after operation, showed a local peritonitis about the gauze packing, into which the original perforation and four small neighboring perforations opened; the catgut sutures were almost entirely absorbed; no general peritonitis; the ileum showed several large confluent ulcers, not perforating. Cultures from the local peritonitis showed bacillus coli communis; cover-slips and cultures from general peritoneal cavity were negative. Case II., death sixteen hours after operation, sutures intact, no other perforations; general peritonitis, cultures growing bacillus coli communis. Case III., death fifty-eight hours after operation; intestinal sutures intact, no additional perforation; no general peritonitis, cover-slips and cultures from general peritoneal cavity negative. Case VI., death four hours after operation; suture intact, no other perforations: general peritonitis; cultures from heart, lungs, kidneys, and brain showed bacillus mucosus capsulatus; cultures from general peritoneal cavity were negative, probably owing to recent irrigation.

RESULTS. In considering the results of these cases from the standpoint of operations for the relief of typhoid perforation, it is manifestly

improper to include Case VI., which was suffering from general peritonitis before the case was seen at all, and was operated upon solely for the relief of the abdominal infection. Case IV., in which no perforation was found, adds one other to the increasing list of explorations without untoward results in cases of suspected perforation, but cannot, of course, be included in the list of cases where perforation has actually occurred. In the four remaining cases with perforation, one alone recovered, Case V. In all four cases there was a sufficient degree of peritonitis to cause an accumulation to a greater or less degree of free abdominal fluid, in which organisms were found in both cover-slip preparations and cultures. Only one case—Case II.—as shown at autopsy, died of general peritonitis. In this instance the operation was deferred eight hours, the longest delay in the series, and an evident mistake. The other two fatal cases—Cases I. and III.—showed local peritonitis about the packing, as occurs in any abdominal wound that is drained, but the general peritoneal cavity was free from fluid and adhesions, and no organisms were obtained from it in cover-slip preparations or cultures. It can be safely said, then, that whatever else one may wish to consider the cause of death in these two cases, it was not general peritonitis. A very large percentage of the fatal cases following operation so far reported, at least 80 per cent., died from general peritonitis. The percentage cannot be given with any great degree of accuracy, however, owing to the small number of post-mortem examinations. The fact that it was possible to prevent the occurrence of general peritonitis in three out of four cases of perforation is worthy of note despite the fact that only one case recovered. The two fatal cases without general peritonitis—Cases I. and III.—lived seventy-six and fifty-eight hours, respectively, after operation. In Case III. the perforation occurred at the height of a moderately severe relapse, in which the clinical evidence of increasing typhoidal toxæmia was apparent before operation. The operation was performed under local anæsthesia without shock to the patient that was apparent to careful observation. It is confidently believed that this death was due to typhoidal toxæmia, and was independent of the operation. As a complete autopsy was not obtainable, however, this statement cannot be maintained with absolute certainty, although it is very strongly supported by clinical symptoms and the absence of general peritonitis. In Case I. perforation occurred on the twenty-first day during the height of the fever in a moderately severe case, before the patient had begun to recover from the effects of the disease, and it was evident that he had very little recuperative power. The clinical appearance of the patient on the last day of life was one of exhaustion and gradual heart failure, and was not unexpected, owing to the condition of low vitality which was apparent before operation. This of course was

added to very materially by the shock of operation, which was, unfortunately it is believed in this instance, performed under general rather than local anæsthesia. It was also undoubtedly added to in a considerable degree by four subsequent perforations near the original lesion. The closure of the perforation was not successful, owing to the subsequent absorption of the catgut sutures. By rare good fortune the four subsequent perforations had opened along the line of drainage, and the extravasation was being cared for completely, as far as could be seen at autopsy, by the gauze drainage going down to the original perforation. The general peritoneal cavity was entirely free from evidences of peritonitis, there being no fluid or adhesions, and the cover-slip preparations and cultures from the same being negative. It is believed that the patient died of exhaustion. It is certain he did not die of general peritonitis. Here, again, however, the absence of complete autopsy leaves the absolute cause of death somewhat in doubt.

TWO CASES OF TYPHOID FEVER COMPLICATED BY NOMA.

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(From the Service of Professor John H. Musser.)

THE following cases are reported partly because of their extreme rarity, partly because they illustrate a serious but, I believe, under certain circumstances, avoidable complication, and partly because they furnish additional proof of the etiological nature of a hitherto obscure condition.

CASE I.—T. T., white, aged fourteen years, a rag-picker by occupation, was admitted to the service of Professor Musser at the University Hospital, July 2, 1901. He had always been a rather delicate boy, and his appetite had never been good. His habits were apparently unexceptionable. His work was very severe, and he was kept at it constantly. His parents were healthy. Four brothers and two sisters were said to be well. Two brothers and one sister have chronic cough with expectoration, but it is not known whether or not this is tuberculous. One brother and one sister died of pneumonia, and the patient stated that it was supposed that his grandmother was suffering from consumption. There was no history of malignant disease in the family. The mother had had one miscarriage. In childhood the patient had measles and spasms. There was no history of influenza or of typhoid fever. For over a year he had had a severe cough with expectoration, and last summer he had an attack of diphtheria. From time to time he had difficulty in speaking, presumably caused by hoarseness. His present illness commenced on June 27, 1901, with headache and pain in the abdomen. He subse-

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quently developed fever and diarrhoea, and on July 1 had an attack of epistaxis. When admitted (July 2d) the temperature was 104°, the pulse 112, respiration 48. There was a profuse reddish papulary

FIG. 1.



Fourth day of the noma. July 16th.

FIG. 2.



Sixth day. July 18th.

eruption with white centres over the body; the pharynx was congested, but there was no membrane. The tongue was heavily coated and black on the dorsum. The abdomen was moderately distended, but not painful. The leucocytes were 4320. A diagnosis of typhoid

fever was made and the blood sent to the Pepper Laboratory for the purpose of determining the presence of the Widal reaction. The patient was then sponged and tubbed. The first tub was followed by a fall of 5° in temperature, and the pulse became very weak. From July 4th the Widal reaction was positive. On this day the physical

FIG. 3.



Seventh day. July 19th.

FIG. 4.



Eighth day, showing the improvement after the second injection of antitoxin. July 20th.

examination gave the following results: Heart not enlarged; sounds clear and normal in intensity. Respirations slightly harsh, almost puerile. Liver commenced at sixth rib and extended to the costal border. Respiratory movements were distinct. The spleen was not palpable. The abdomen was slightly distended, and there was some

gurgling in the right iliac fossa. There was an eruption over the body consisting of elevated papules with whitish centres—evidently an acne. Distinct typhoid spots could not be made out. The face was flushed; the tongue broad and black on the dorsum; the lips dry and the seat of a slight herpetic eruption. The throat appeared to be normal. The pulse was medium, full, slow and compressible. The vessels were soft. The knee-jerks were markedly exaggerated. On July 5th the diazo reaction was positive. The urine contained a faint trace of albumin and a few leucocytes. On the following day a distinct and abundant roseolar eruption appeared on the abdomen. The patient's condition continued serious, but not alarming, for several days. From time to time he had an involuntary evacuation of the bowels, and there was some distention of the abdomen. There was cough, with copious dirty brown expectoration, which, when examined, showed numerous forms of micro-organisms, including a form resembling the leptothrix. Tubercle bacilli were not found. On July 12th, the sixteenth day of the disease, it was observed that the right jaw was

FIG. 5.



Ninth day, just before death. July 21st.

swollen and that the right lower molars had become loosened. There was also some ulceration of the mucous membrane of the cheek and of the gum over the right lower alveolar process, and the eruption on the lips was more pronounced. On July 13th three of the lower molars became so loose that they were removed. The patient was seen by Drs. Cryer and Frazier, who confirmed the diagnosis of gangrenous stomatitis. The condition rapidly progressed. The right submaxillary gland became swollen, and a reddish spot appeared on the cheek just below the right corner of the lip. By July 15th a small purplish spot appeared about one-half inch below this, and the ulceration of the interior of the mouth had become very severe and of a dark greenish-purple color. The purple spot on the outside of the face rapidly increased in size in the following manner: First, the area around became reddish, then a blister formed, which was in turn invaded by the gangrene. Dr. Frazier suggested hot poultices of 1 to 8000 bichloride constantly applied to the lesion, and a mouth wash of bichloride. The patient's condition not being satisfactory he was given free stimulation. The

cultures made from the mouth were sent to the Pepper Laboratory and to the pathological laboratory of the medical school for diagnosis. On July 17th the latter reported that the cultures taken from the mouth, which were made only upon agar, showed streptococcus pyogenes and staphylococcus albus. Dr. Kneass, of the Pepper Laboratory, reported that cultures on blood serum gave a typical growth of the Klebs-Loeffler bacillus and other cocci. The patient was immediately isolated and given 4000 units of anti-diphtheritic serum. During the following night the patient rested more easily, the gangrene did not spread, but toward morning he was slightly delirious. The second culture taken from the mouth was examined on July 19th by Dr. Kneass, who again found and demonstrated to me a typical growth of diphtheria bacilli. On July 19th the patient again received 4000 units of antitoxin. By this time signs of perforation had appeared in the gangrenous area. The following day the general condition was much improved, and the local condition had ceased to spread. There was also a very marked decrease in the oedema surrounding the gangrenous area, and the patient was able to open the right eye without difficulty. On July 21st the lesion again commenced to spread and the pulse was weaker. At 2 P.M. 4000 units of antitoxin were given and that night the patient again was slightly delirious. He grew weaker, and finally died at 10 A.M. on July 23d, 1901.

CASE II.—M. T., female, aged eight years, a sister of T. T., was admitted to the service of Professor Griffith on July 8, 1901. The patient had always been delicate, but there was no history of diphtheria or other acute disease. One week before admission she had complained of headache, and had had a slight diarrhoea, poor appetite, and a disposition to sleep most of the time. When admitted she still complained of headache; was slightly delirious; hearing was slightly impaired; the abdomen was not distended or tender; there was slight diarrhoea. Physical examination showed a poorly nourished, pale girl; the tongue was coated and red at the edges; the breath was fetid; the respirations were rapid; the lungs were negative, with the exception of a slight harshness in the right axillary line. The spleen was distinctly enlarged; the urine was normal; the leucocytes were 9000; the Widal reaction was positive. From this time the case gave the typical appearance of a moderately severe attack of typhoid fever in a child. July 12th there was a moderate eruption of rose-spots on the abdomen; the temperature ranged between 104° and 105°, and she responded poorly to antipyretic measures. July 17th she complained of so much soreness in the mouth that it was necessary to feed her through a tube. The pulse was rapid and weak, and the temperature still remained elevated. Her condition remained exceedingly serious until July 28th, when such distinct improvement occurred that she was able to take milk by the mouth. Investigation of the mouth showed at this time some necrosis of the upper jaw on the right side, and the canine tooth in the necrotic area was loosened. July 30th a culture was taken from this necrosed area, which in the meantime had extended slightly. The leucocytes were 9000. August 3d it was noted that the necrosed area had not increased. The temperature was lower and the patient was obviously better. August 7th the report returned from the Pepper Laboratory stated that the diphtheria bacillus was present in the necrotic area, and an injection of anti-diphtheritic serum was immediately given. August 8th a

discharge was noted from the right ear. On this day a second culture from the mouth was positive, and a culture from the discharge from the ear also developed diphtheria bacilli. August 9th, 500 units of antitoxin were administered. A loose tooth fell out. August 11th another tooth fell out and a culture was made from the cavity, diphtheria bacilli being found. On this day the leucocytes were 10,900. August 15th the necrosed area was healing rapidly; the temperature was normal, and the patient had apparently recovered. August 20th a culture from the mouth was negative, and the patient was discharged cured two days later.

At the time that this patient was in the hospital another brother was also in the children's ward suffering from typhoid fever. He did not develop necrosis of the jaw, and unfortunately cultures from the throat were not made.

Briefly summarized, the interesting features of these cases are that two brothers and a sister from the same family were admitted to the hospital in a short period, all suffering from typhoid fever. The previous summer one of the brothers had had an attack of diphtheria from which he had apparently recovered completely. Toward the end of the typhoid infection the sister and the brother who had had diphtheria, developed necrosis of the jaw, followed in the brother's case by typical cancrum oris. Diphtheria bacilli were obtained from the necrotic and gangrenous areas in both cases.

Noma is an exceedingly rare complication of typhoid fever. In the majority of text-books that I have consulted it is not even mentioned. Dr. Keen states that in 1700 cases with surgical complications that he analyzed, noma was recorded 9 times. Five of the patients died; 3 recovered; and the termination in one was not given. As these were selected cases, it is evident how very unusual noma really is. Walsh, however, in his analysis of the statistics collected by Hildebrandt and Perthes, notes that noma followed typhoid fever in 26 of 133 cases, a proportion that is not, he says, in accordance with his own experience. Worochinin¹ reports a great number of cases of noma, 46 of which were treated in the hospital; and in 31 of these the associated or pre-disposing disease could be determined. In these 31, 2 followed typhoid fever, and it is interesting to note that in both the patients had complained of sore-throat. In one of these the condition was described as faucial diphtheria, and in the other as a cachectic angina. Of course, the majority of these cases were observed before the discovery of the Klebs-Loeffler bacillus. Of the remaining 29 cases, one followed scarlet fever and diphtheria; 2 were associated with ulcerative stomatitis; and one, a case of noma of the vulva, occurred in a girl of three years, who was said to have suffered from diphtheritic vulvitis. Only 3 of 22 cases in which the results were given recovered.

¹ Jahrbuch für Kinderheilkunde, 1887, vol. xxvi. p. 167.

The first¹ article in which attention was called to the possibility that noma, in some cases at least, is due to the diphtheria bacillus, was that of Freymuth and Petruschky.¹ Curiously enough, this paper attracted very little attention at the time. They observed 2 cases: one in a girl, aged three years, who had an attack of measles followed by noma of the vulva and of the adjacent parts. The Klebs-Loeffler bacillus was found in the gangrenous areas, and the patient subsequently developed diphtheria of the fauces. Antidiphtheritic serum was given in large and repeated doses, and the patient recovered. Petruschky had also discovered the diphtheria bacillus in a case of gangrene of the foot, and has even found one similar to it, if not identical with it, in measles. In discussing the bacteriology of the case he notes that the culture obtained was only slightly pathogenic for guinea-pigs, that is to say, it caused death in about ten days, with the characteristic changes, such as swelling of the adrenals. In the same year² they reported the second case occurring in a boy, aged eight years, who, in the sixth week of typhoid fever, developed stomatitis, followed by typical noma. The Klebs-Loeffler bacillus and the pseudodiphtheria bacillus were found, and the former was only slightly pathogenic for guinea-pigs. The authors express the opinion that probably the Klebs-Loeffler bacillus would be found in many cases of noma if it were sought for, particularly as diphtheria and measles and noma and measles are very frequently associated.

The next publication on the subject was that of Passini and Lenier,³ who reported a case occurring in a boy, aged eight years, living under unfavorable hygienic conditions, who developed a blister upon the cheek, then a black spot, and finally typical noma. When brought to the hospital he was moribund, but nevertheless the Klebs-Loeffler bacillus was found in the gangrenous area. At the autopsy tuberculosis of nearly all the organs was found.

Perhaps the most important contribution to this subject is that of Walsh.⁴ Being familiar with the paper of Freymuth and Petruschky, and having an opportunity to study 8 cases of noma, he examined them for the diphtheria bacillus, and found it in all cases. In addition, he made cultures from 15 cases of simple ulcerative stomatitis, all of which were negative. Five of the cases of noma had recovered from measles; 1 from diphtheria, and 2 apparently occurred spontaneously. In addition to these articles it may be mentioned that Nicolaysen⁵ found in 2 cases of noma a non-pathogenic bacillus resembling the diph-

¹ Deutsche medicinische Wochenschrift, 1896, p. 232.

² Ibid., 1900.

³ Wien. klin. Woch., 1899, p. 734.

⁴ Proceedings of the Pathological Society of Philadelphia, June, 1901, p. 179.

⁵ Norsk. Mag. for Lægevid, 1896, quoted by Klautsch, Archiv für Kinderheilkunde, 1899, vol. xxvi, p. 245.

theria bacillus. In view of the observations of Petruschky¹ regarding the diminished virulence of the diphtheria bacillus associated with noma, it is possible that Nicolaysen was actually dealing with the true germ. Aside from the diphtheria bacillus, the number of micro-organisms that have been discovered is considerable. Among the most important are the long, thread-like bacilli found by Grawitz,² and observed in smear preparations from the mouth of Case I., and a very straight, non-motile bacillus with round ends, found in 3 cases by Babes and Zambilovici,³ and by Guizetti.⁴ The latter micro-organism produced gangrene in rabbits.

A hypothetical history of the cases I report might be given as follows: The first patient contracted diphtheria, from which he recovered, but the diphtheria bacilli remained in the buccal cavity. At the same time, his sister either had the same disease in a mild form, or else received diphtheria bacilli from her brother in a state of such diminished virulence that they were not capable of causing inflammation. In both cases, however, the micro-organisms retained sufficient pathogenicity to produce necrosis and gangrene in tissues whose vitality was lowered by another grave infectious process. That the germs were not very pathogenic was proved by the fact that two guinea-pigs into which pure cultures were injected became very sick for a few days, but did not die.⁴

These cases, I believe, are important for the following reasons: In the first place, they tend to confirm the opinion that has been frequently urged, but has not acquired sufficient adherents, that in all cases of sore-throat an examination for diphtheria bacilli should be made, and that if they are found a prophylactic injection of antidiphtheritic toxin should be given to the patient. In the second place, they seem to show that one of the causes of noma is the diphtheria bacillus; that it acts as such a cause usually in the presence of some other infectious disease, particularly in patients whose physical condition is, aside from the infectious disease, bad; and that as a general rule the diphtheria bacillus in these conditions is of diminished virulence, and not capable of producing the characteristic signs and appearance of true diphtheria.

In conclusion, I desire to express my thanks to Professor J. P. Crozier Griffith for his generous permission to report the second case, and to Dr. Levi, my resident physician, for his assistance in the study and observation of the first case.

[Since the above article was written the admirable report of an epidemic of noma by Drs. Blumer and McFarlane has appeared in THE

¹ Deutsche medicinische Wochenschrift, 1890.

² Annales de l'Institut de Pathologie du Bucarest, 1895.

³ Policlinico, 1896.

⁴ Unfortunately, Dr. Kneass was absent from the city, and further experiments could not be made.

AMERICAN JOURNAL OF THE MEDICAL SCIENCES for November, 1901. The authors believe that noma is most frequently due to a thread-like organism of the leptothrix type that does not grow upon ordinary media. The diphtheria bacillus was apparently excluded as a causative factor in their cases.]

THYROIDITIS COMPLICATING TYPHOID FEVER.*

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INFLAMMATION of the thyroid gland is not uncommon in those localities where goitre is endemic. In such cases it occurs usually as an acute condition, in a gland the seat of previous hypertrophy. This is known as strumitis acuta. Inflammation of a previously healthy gland, however, is rare. Bardeleben, in Eulenberg's *Encyclopædia*, said he had never seen a case, and both Paget and Semon similarly expressed themselves when their attention was drawn to Barlow's case, though Semon has since reported one coming under his own observation.

Lücke,¹ in Pitha and Billroth's *Surgery*, in his article on "Acute Inflammation of the Thyroid," gives a concise description of the inflammations of the previously healthy gland, and divides them into: 1, idiopathic; 2, traumatic; 3, metastatic. The idiopathic form, he says, is extremely rare, occurs especially in young people about the age of puberty, and is etiologically very obscure. According to Godlee,² the following varieties are met with: 1. Idiopathic, with spontaneous subsidence. 2. Epidemic, as described by French military surgeons. 3. Septic form. This classification takes no cognizance of the traumatic form.

Mygind³ divides thyroiditis into the simple and suppurative, the former terminating in resolution, and the latter in abscess. Though this grouping fails to take any note of the etiological factors, it seems to me the best, if for no other reason than that it obviates the use of the term idiopathic. Admittedly rare, as already stated, Mygind was only able to collect from the literature seventeen cases of acute inflammation occurring in a previously healthy gland, and ending in resolution.

The condition occurs most commonly in females, according to Mygind,⁴ particularly between the second and third decades; rarely in previously healthy persons and without any apparent cause, sometimes as a result of traumatism, and finally, in the so-called metastatic cases, during or

* Read by title at the meeting of the State Medical Society held in Philadelphia, September, 1901.

immediately following an attack of typhoid fever, rheumatic fever, diphtheria, influenza, malaria, erysipelas, puerperal fever, sepsis, orchitis, or parotitis. The traumatic and metastatic forms almost invariably terminate in suppuration, except those possibly occurring in the course of rheumatic fever or malaria. Sajous⁴ says that the uterine, menstrual, and climacteric disorders tend to cause congestion, which, often repeated, may give rise to active inflammation of the thyroid gland. French writers⁵ on military surgery have reported many epidemics of acute enlargement of the thyroid occurring among soldiers in garrisons, and Viry and Richard, who reported the Belfort epidemic of 1877, claimed that the epidemic disease is an acute specific one, having no relation whatever to ordinary goitre. The French writers further speak of the resemblance of this form of thyroiditis to mumps.

Semon has reported a case of thyroiditis alternating with mumps. Most commonly, rheumatic or typhoid fever is the immediate antecedent of thyroiditis. Moliere,⁷ of Lyons, in 1873, was the first to record a case of acute thyroiditis supervening on acute rheumatism. It developed when all the joints had ceased to be painful.

Vulpian,⁸ in 1877, reported a case following arthritis of the fingers, associated with generalized pain. Raymond⁹ another following multiple arthritis. Barlow's¹⁰ case was a child of three years, the youngest I have seen recorded. The boy had had a mild attack of erythema nodosum and was almost well when the thyroid became acutely inflamed; fever, dysphagia, and stiffness of the neck supervened, reached its acme in four days, and subsided without suppurating after a course of about ten days. Angel Money,¹¹ in the discussion of Barlow's paper, spoke of a case of a young woman who had had repeated attacks of rheumatism with heart disease and erythema papulatum and marginatum, who, when convalescent from one of the attacks, acutely developed inflammation of the thyroid. It soon resolved. Given¹² reports the case of a nurse, aged twenty-three years, who had just recovered from an attack of rheumatism when she suddenly fell ill with general malaise, pain and swelling of the neck in the region of the thyroid, temperature 105.3°, dysphagia, dyspnoea, inspiratory stridor, tenderness over thyroid, and subsequent resolution without suppuration.

It is, however, as a complication, or more frequently as a sequel of typhoid fever that we usually meet with acute thyroiditis, and in the majority of cases it goes on to suppuration, though it must not be lost sight of that even in typhoid the condition is rare, as the following statistics show:

Among 349 autopsies in Hamburg, Curschmann¹³ did not see a single case, and only twice clinically during the large epidemic in that city. In Leipzig, too, he says it is quite rare. Walther,¹⁴ who published the only case of thyroiditis typhosa occurring in Curschmann's clinic, had

previously shown that of 73 collected cases of acute strumitis and thyroiditis, 40 were associated with typhoid fever. The relative frequency of thyroiditis in Switzerland explains the findings of Griesinger¹⁵ and Liebermeister.¹⁶ The former, four times in 118 autopsies, the latter, 15 times among 1700 patients, in 6 of whom suppuration developed. Among the complications of typhoid fever, Topfer¹⁷ mentions 3 cases of abscess of the thyroid in 927 autopsies.

The immediate exciting cause of thyroiditis occurring during the course, or as a sequel of typhoid fever, is worthy of some attention. Is the typhoid bacillus ever pyogenic, or are the various grades of inflammations due to the ordinary pus-producing organisms, especially the staphylococcus and streptococcus? There is no longer any doubt that the bacillus of Eberth assumes pyogenic properties in certain cases. This was definitely proved, first by Fränkel,¹⁸ who recovered it in pure culture from a case of encysted peritonitis; but two years before, Freund¹⁹ claimed that inflammation of bone occurring during convalescence from typhoid was due to the specific action of the typhoid bacillus. Ebermaier,²⁰ among 8 cases of typhoid periostitis, observed 2 which went on to suppuration, and recovered from the blood and tissues of the affected part a pure culture of typhoid bacilli; Valentini²¹ the same from a case of empyema complicating typhoid fever; Achalme²² in osteoperiostitis of the tibia occurring in the beginning of convalescence from the same disease. Chantemesse and Roux²³ have experimentally produced pus by injecting rabbits with a virulent typhoid culture, as has Honl,²⁴ who tabulated his results as follows:

1. When injected into certain parts of the animals, typhoid bacilli may cause pus.
2. Toxins and old cultures have pyogenic properties.
3. The concentration of the culture has no influence on increasing the pyogenic function.
4. Bacilli from abscesses disappear very rapidly.

In the course of typhoid, especially toward convalescence, various inflammatory affections may arise, due to invasion of germs with pyogenic properties, only rarely to the typhoid organism alone. Honl²⁵ has summed them up in the following order of frequency:

- a. Secondary invasion of pyogenic cocci.
- b. Secondary microbiotic mixed infection, *i. e.*, through cocci or other microbes plus typhoid bacilli.
- c. Exclusively through the bacillus of Eberth.

Honl states that in the Pathologico-Anatomical Institute of Prague, in ten years, 1883 to 1893, 165 autopsies were performed on typhoid cases, in 16.39 per cent. of which some purulent process had been the cause of death. Of more direct interest is the finding of Spirig,²⁶ who was the first to cultivate typhoid bacilli in a case of strumitis; he also

found staphylococci. Chantemesse,²⁷ from the pus of a case of suppurative thyroiditis complicating the defervescent stage of typhoid fever, recovered the typhoid bacillus with the ordinary organisms of suppuration. The bacillus of Eberth was also found in typhoid thyroiditis by Lichtheim-Tavel²⁸ and Jeanselme.²⁹ The mixed infections are, as a rule, the most serious; indeed, they are often but part of a general sepsis.

The case which I wish to present did not go on to suppuration, but illustrates the simple type of Mygind. The patient, a man, aged forty-two years, so far advanced in convalescence from typhoid as to have reached a normal temperature, began to complain of difficulty in swallowing. The thyroid gland became enlarged quite acutely, the right lobe being more involved than the left, it was quite tender on palpation, giving a sense of semi-fluctuation. Soon after the onset he had a slight chill, his temperature became moderately elevated, he complained of a feeling of constriction about his throat, dysphagia became more pronounced, indeed, it was the most troublesome feature of the attack; slight dyspnoea supervened, and owing to the pain in the neck, his head was held rather rigidly. For three days he was scarcely able to swallow liquids, but with the subsidence of the tumefaction all manifestations gradually vanished, the process terminating in resolution after about ten days. Such attacks are usually sudden in onset.

When the left lobe of the thyroid is especially implicated, dysphagia is apt to be more pronounced, owing to greater opportunity for pressure because of the left lateral deviation of the œsophagus. Dyspnoea is seldom severe and rarely associated with stridor. Hoarseness is exceptional. It is due to pressure on the inferior laryngeal nerve, which is situated between the lobes of the gland and the œsophagus. Mygind's³⁰ is the only case of the kind reported, though Galtier speaks of the possibility of such a condition. In Mygind's case complicating facial erysipelas in a man of thirty-eight years, the great hoarseness led to an examination of the larynx. The mucous membrane was found inflamed and swollen, the left vocal cord being fixed in the cadaveric position during deep respiration, and only moved a little toward the median line on phonation. It is very rarely that pressure on neighboring vessels gives rise to any symptoms. Of course, all symptoms are apt to be more pronounced in suppurative cases, but very seldom do threatening symptoms supervene.

Griesinger³¹ has published a death from suffocation, and Forgue³² another, in the latter case due to perforation of a thyroid abscess into the trachea. Luigi Porta,³³ in an extensive article on diseases and operations of the thyroid gland, speaks of the possibility of death by erosion of a large vessel, and he further gives a most grave prognosis in those cases of deep-seated abscesses in which the pus finds its way into the mediastinum or pleura. The abscess may be single or multiple,

but in any case is apt to involve one lobe more than the other. Of the ten case of thyroiditis complicating typhoid fever collected by Keen,³⁴ in all but one suppuration occurred. In four of them the gland was previously hypertrophied. In seven the attack began during convalescence, *i. e.*, after the third week, in two as late as the seventh or eighth week. This is characteristic of glandular involvement in typhoid, be it of the testicle, ovaries, parotid, or thyroid, that it is a consecutive or secondary manifestation, and belongs to the period of decline and convalescence. Tavel and Laveran³⁵ have shown that it may very rarely develop with the onset. One very curious case published by Kummer and Tavel and quoted by Keen³⁶ was as follows: Suppuration occurred in a goitrous gland, from the pus of which Eberth's bacillus was recovered. No typhoid bacilli were found in the stools, nor any other evidence of typhoid infection.

The prognosis is good, threatening symptoms are unusual, and fatalities are among the greatest rarities, as has been stated. The consensus of opinion is that goitre never results, but Luigi Porta³⁷ says hypertrophy may occur in consequence of unresolved inflammation. I do not know of any recorded case.

The course of the attack is generally short, ten to fourteen days.

The treatment of the simple variety of thyroiditis consists merely in the use of ice locally. If suppuration is impending it may be hastened by hot fomentations, and an incision should be made as soon as evidence of pointing or fluctuation occurs. If the gland is the seat of pre-existing hypertrophy, it would be advisable to extirpate the suppurating half, as Kummer did. This would be done, of course, at some subsequent period when the patient had well advanced in convalescence.

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CAUSE OF DEATH IN ANEURISMS OF THE THORACIC AORTA WHICH DO NOT RUPTURE; REPORT OF FIVE CASES.

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OWING to the frequency with which incomplete post-mortem examinations have been made in cases of thoracic aneurism, statistics are not entirely satisfactory. Hare and Holder¹ have collected from medical literature 953 cases of aneurism of the arch of the aorta. Among their conclusions these writers make the observation² "that in a large proportion of cases death did not ensue from rupture but from pressure by the growth." In another place they say:³ "Death occurs as a result of aneurism of the aorta either from rupture of the aneurismal sac, from pressure of the sac upon important nerves and bloodvessels, or from the secondary changes which take place in these tissues and in other vital organs as a direct or indirect result of such pressure."

From various data in their article I have made the following tabulation of the number of cases in which it was stated that death occurred without rupture.

| | | |
|-----------------|-----------|-------------------------------|
| Ascending arch, | 570 . . . | Unruptured, 93 = 16 per cent. |
| Transverse " | 104 . . . | " 20 = 19 " |
| Descending " | 110 . . . | " 17 = 15 " |
| Unclassified, | 169 . . . | " 24 = 14 " |
| <hr/> | | |
| Total, | 953 . . . | " 154 = 16 " |

I have also made a tabulation of the cases in which rupture is definitely stated to have occurred.

| | | |
|-----------------|-----------|------------------------------|
| Ascending arch, | 570 . . . | Ruptured, 339 = 60 per cent. |
| Transverse " | 104 . . . | " 39 = 38 " |
| Descending " | 110 . . . | " 72 = 66 " |
| Unclassified, | 169 . . . | " 74 = 44 " |
| <hr/> | | |
| Total, | 953 . . . | " 524 = 55 " |

Browne,⁴ in a series of 173 cases, found that only 84, or 49 per cent., were due to rupture, a smaller percentage than that among the cases reported by Hare and Holder. Sibson⁵ made an analysis of 584 reported cases of aortic aneurism and of 296 museum specimens. Of the total number of 880 cases 703 were instances of aneurism of the thoracic aorta. Of these latter 358 ruptured, or 51 per cent.

| | | |
|-----------------|--------------|------------------------------|
| Hare and Holder | . 953 cases. | Ruptured, 524 = 55 per cent. |
| Sibson | . . . 703 " | " 358 = 51 " |
| Browne | . . . 173 " | " 84 = 49 " |
| Total | . . 1829 " | " 966 = 53 " |

The inference seems almost as reliable that nearly one-half of the cases of aneurism of the thoracic aorta die without rupture of the sac.

Turning, now, to the analysis of the 154 cases collected by Hare and Holder, of which it was stated that death was not due to rupture, I have made the following tabulation of the causes assigned for death by the various observers: *

| | |
|--|----|
| Obstruction to air-passages | 66 |
| Dyspnoea | 26 |
| Suffocation | 10 |
| Pressure on trachea | 27 |
| Pressure on bronchus | 2 |
| Edema of larynx | 1 |
| Exhaustion | 50 |
| Affections of lungs and pleura | 28 |
| Apoplexy of lung | 3 |
| Congestion of lungs | 2 |
| Pneumonia from pressure | 2 |
| Pneumonia | 1 |
| Pleuropneumonia | 1 |
| Abscess of lung | 1 |
| Bronchitis | 2 |
| Serous pleural effusion | 14 |
| Pleurisy | 2 |
| Pericardial affections | 8 |
| Serous pericardial effusion | 7 |
| Pericarditis | 1 |
| Pressure on the vena cava superior | 1 |
| Collapse | 1 |

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It is clear at a glance that this information is vague and unsatisfactory. The largest single item is death from exhaustion, comprising practically one-third of the cases. In too many of these cases, I fear, the reporter thoughtlessly said "exhaustion" without any special effort to see if there were a better explanation. Suffocation, credited with ten deaths, leaves the mind guessing as to the exact way in which it was caused. Pressure on the trachea, credited with twenty-seven deaths, is an adequate cause of death if the pressure is extreme, but moderate

* It may be noted again that the faults in this table are due to the imperfect report of cases, and not to the excellent compilation made by Hare and Holder.

pressure does not necessarily cause death, though doubtless it may be one of the factors in producing that result. Dyspnoea is fatal not in itself, but from the condition of which it is a symptom. It may signify trouble in the respiratory organs, in the circulation, or irritation of certain parts of the nervous system. The twenty-six deaths caused by dyspnoea might certainly be more carefully analyzed. It is a noteworthy fact that any complication involving the heart is not once mentioned specifically as a cause of death in these cases, though recognized by all the leading authorities as holding an important place in this connection. Only one case is mentioned of pressure on the great vessels about the heart—another well-recognized factor in the fatal results. Undoubtedly more careful observation would have shown that many of the cases classed under the heads of exhaustion, dyspnoea, suffocation, etc., were really deaths due to disturbance of the circulatory apparatus. It is not an exaggeration to say that many of these supposed explanations do not explain, and I think it is evident that there is need of a closer scrutiny of the fatal cases which do not rupture and a more thoughtful consideration of the real cause of death.

Let us consider briefly some of the problems to be thought out in this connection. We may divide the cases into three classes :

I. Cases in which an aneurism of the arch of the aorta exists, but in which it is not the important factor in the fatal result.

II. Cases in which death ensued from disease or disturbance of the circulatory system not caused by direct pressure of the aneurismal tumor.

III. Cases in which death was due, directly or indirectly, to pressure of the aneurismal tumor upon organs of vital importance.

Class I. comprises those cases which die from some intercurrent disease whose origin cannot properly be ascribed to the aneurism. Diseases of other parts than the thoracic organs or circulatory apparatus need no special discussion, and even some diseases of the thoracic organs may be so obviously due to other causes than the aneurism that its existence is plainly an unimportant incident. On the other hand, it will not be easy in other cases to decide how far the aneurism may have been a contributing factor in the causation of such disease. This is illustrated by the following case :

CASE I.—Male, aged fifty-one years, stationary engineer. His family history and previous history are unimportant, except that when twenty years old he was thrown from a locomotive and injured his back so that he lost the use of his legs for one month, and that he had two attacks of pneumonia nine and seven years ago. No history of syphilis could be obtained. Alcohol was not used to excess. On December 31, 1897, he was assaulted by two men, strangled, and beaten until insensible. It is probable that the strangulation, together with the violence of his struggles, may have been the immediate cause of the giving way of the

arterial wall. Certain it is that as a result he suffered from broken cardiac compensation for several months.

The more serious symptoms of his present trouble date from April 1, 1901. His first serious attack of dyspnoea occurred on April 19th. This attack resembled angina pectoris in the nature of its distress and anxious suffering. It also suggested to the attending physician emphysema with an asthmatic attack. There was marked respiratory dyspnoea, "rattling breathing," and stridor. There was very little cough. On the night of April 25th, after drinking a certain amount of liquor, while walking on the street, he "felt queer," rapidly choked up, lost consciousness, and did not awaken until some hours later at the hospital.

At this time the physical examination showed the pupils equal, with normal reaction. Pulse was irregular, slow, of good volume and tension. The walls of the radial arteries were sclerotic. The right border of cardiac dulness was $3\frac{1}{2}$ cm. to right, and the left border $10\frac{1}{2}$ cm. to left of the median line; upper border at the third rib. The apex was in the fifth interspace, 10 cm. to left of median line. The heart sounds were faint. No murmurs were detected. The lungs were negative. No signs of aneurism were detected. He improved and left the hospital April 27th at his own request.

On April 28th he had two attacks of dyspnoea, similar to his first attack. On April 29th he had two attacks; on April 30th one attack. His next attack was on May 12th. Four attacks followed in the next two days, and the final attack began on the night of May 15th.

He was brought to the hospital in a semi-conscious condition. The peculiar crowing respiration and brassy cough at once attracted attention, and suggested the possibility of aneurism. A tracheal tug was found. Dulness in the second interspace extended 5 cm. to the right of the median line, but there was no impulse, thrill, or bruit detected. The right radial pulse was entirely obliterated, the left was regular and of only fair strength and volume. The heart was the same as at the previous examination, but weaker. Percussion showed fair pulmonary resonance throughout the chest. Auscultation showed exaggerated, crowing respiration, with many coarse bubbling and fine moist râles. He did not improve, the pulmonary oedema increased, and he died early the next morning.

The autopsy showed a sacculated aneurism of the transverse arch of the aorta, and a dilatation of the ascending arch; chronic endocarditis of the mitral valve; hypertrophy and dilatation of the left ventricle; general chronic passive congestion; arterio-sclerosis; lobar pneumonia; acute congestion, and oedema of the lungs.

The heart muscle appeared of good quality and showed no fatty changes microscopically. There was nothing noteworthy beyond a moderate thickening of the mitral valve and chordæ tendinæ and moderate hypertrophy and dilatation of the left ventricle.

The aortic orifice measured 8 cm. in circumference; one centimetre higher the aorta measured 9 cm., and six centimetres beyond the valve it measured 11 cm. At nine centimetres from the valve a sacculated dilatation began, extending backward from the transverse arch. The dilated aorta merged into the aneurismal sac without a definitely marked border on the side toward the heart. The distal edge of the sac had a definite rounded border. At this latter point the aorta measured 6 cm. in circumference, and continued of this size to the diaphragm.

The opening from the aorta into the sac measured 4 to 5 cm. in diameter, and 14.5 cm. in circumference. The circumference about the sac and aorta was 15 cm. The intima of the aorta and the lining of the sac showed irregular thickened patches, but no calcification.

The posterior wall of the sac was in contact with the trachea just above its bifurcation. The wall of the sac was eroded at this point over an area 4 by 2 cm., and here the rough tracheal wall and the cartilaginous rings could easily be felt. The tracheal wall was somewhat thinned at this place. The trachea was somewhat flattened antero-posteriorly, but at the post-mortem examination there was no definite bulging of the aneurism into the lumen of the trachea.

The cavity of the aneurism contained fluid blood except for a small amount of fresh red clot. The left pneumogastric nerve ran down the side of the sac anteriorly, but seemed perfectly free. The right pneumogastric nerve was not in direct relation to the sac.

The vessels of the neck arose from the anterior wall of the sac. The left carotid and subclavian arteries appeared normal. There was no opening to the innominate artery from the sac. Where the opening should be was a smooth, rounded thickening with a very small depression. The smallest probe could not find a passage. On opening the innominate artery from its distal end it appeared normal until within one centimetre of the sac, when it suddenly narrowed and was completely occluded by a firm, reddish-yellow thickening beneath the intima on one side. The arteries and veins of the right arm appeared normal and were filled with blood.

An area of acute lobar pneumonia occupied about the upper fourth of the lower lobe of the right lung. It also extended to a small area at the root of the upper lobe, but did not involve the middle lobe. The remaining portions of both lungs showed intense acute congestion and œdema. Death was evidently due to this congestion and œdema of the lungs, which in turn was caused probably by the toxins of the pneumonic inflammation. The stage of development of the pneumonia indicated that it was about the fourth or fifth day, which suggests that the series of attacks beginning May 12th were induced by the pneumonic process.

What part did the aneurism play in causing the death of this patient? It certainly was not the immediate cause, yet it is hard to believe that it did not have an important contributing influence. In the first place the recurrent attacks of extreme and prolonged dyspnoea must have materially affected the general condition and strength of the patient. This reduced the resisting power of the system of an individual who was easily susceptible to pneumonic infection, as shown by the history of two previous attacks of pneumonia. It would seem probable, also, that the obstruction to the passage of air to the lungs might have induced changes in the lung tissue, rendering those tissues more susceptible to invasion by the pneumococcus. Finally, after the pneumonic process started, the frequent attacks of dyspnoea from the pressure of the aneurism on the trachea must have had a bad effect upon the patient's vitality in fighting the pneumonia. It is not clear how far the

last attack of dyspnœa was due to the aneurism, and how far to the general acute congestion and œdema of the lungs. When he was examined at the hospital pulmonary œdema was already well established, but he also had the crowing respiration and brassy cough of aneurism.

In Class II., which includes cases of death from circulatory disturbance not caused by direct pressure on the bloodvessels, the chief doubt will arise in determining how far in cases associated with arterio-sclerosis death may have been caused by that disease rather than by the aneurism itself. Arterio-sclerosis may cause death independent of the aneurism, as a result of changes in the nutrition of vital organs, by rupture of the diseased arteries, by the plugging of the arteries from thrombosis or embolism, or by its combined effect on the work and the nutrition of the heart. If death occurs from embolism, its source may be the aneurism itself, since coagulation of blood is favored in the aneurismal sac, and the danger of embolism is thus increased. Or, again, the aneurism may be an important factor in causing death by its effect upon the heart.

What effect may an aneurism of the aorta have upon the heart? It has been claimed by some that it causes hypertrophy of the heart. Calvert⁶ has shown that this is not necessarily the case. His conclusion is based on statistics gathered by Dr. Oswald A. Browne from the cases occurring in St. Bartholomew's Hospital in thirty years. Of 124 cases, 68 showed no hypertrophy; in 47 the hypertrophy could be satisfactorily explained by other causes; and in the remaining 9 hypertrophy could be explained as probably due to other causes. In arterio-sclerosis we have a very common cause of heart hypertrophy, so that in many cases the aneurism and the heart hypertrophy are to be regarded as common results of the same cause and not as one dependent upon the other. One form of aneurism, however, certainly has a direct effect upon the heart. I refer to aneurisms situated close to the aortic valve and involving a general dilatation of the aorta. If such an aneurism enlarges enough it will by its stretching cause dilatation of the aortic orifice, from which follows aortic regurgitation with its results. Case II. belongs to this group.

CASE II.—Male, aged forty-four years, choreman, came to the outpatient department of the Boston City Hospital, February 18, 1899. Family history is unimportant. He had syphilis twenty-five years before. He had used alcoholic drinks moderately.

His chief complaint was of pain and a feeling of pressure in the præcordial region, increased by exertion. He had suffered from this about one week. His trouble, however, began at least a year before, at which time he began to have a severe boring pain in the back of the chest, together with dyspnœa on exertion. At the time of the examination he was not hoarse, but he had had several attacks of hoarseness in the last few years.

Inspection showed the pulsation of the carotid arteries more marked and jerky than normal. On the right side of the neck, just above the

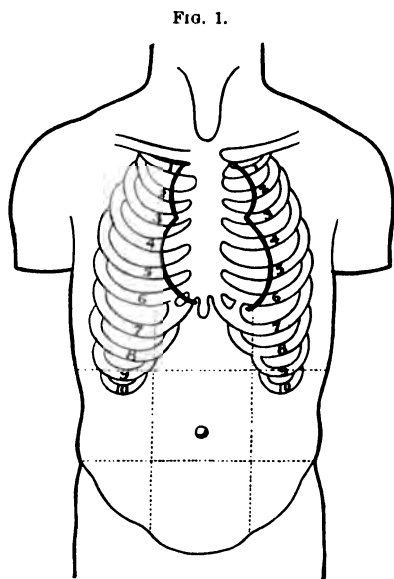
inner end of the clavicle and also in the episternal notch, a marked pulsation was seen. Palpation showed that the pulsation in the episternal notch had a distinct lateral thrust from the right, indicating its origin from the innominate artery and not from the arch of the aorta. A thrill was plainly felt just outside the sternomastoid muscle above the clavicle. The right radial pulse was weaker than the left, but there was no perceptible difference in time. A slight but distinct tracheal tug was made out. The pupils were equal. The apex-beat of the heart was felt in the sixth interspace in the left mammary line.

Percussion over the lungs was normal. Relative dullness of the heart extended to the left mammary line, four and seven-eighth inches to the left of the median line, and on the right its border was one inch to the right of the median line. The area of dullness over the great vessels was increased in breadth. On the left, at the base of the heart, dullness extended two inches from the median line, diminishing to one and five-eighth inches higher up. On the right the distance was one inch from the median line at the base of the heart, increasing to one and three-eighth inches just below the clavicle. The line of dullness bounding the great vessels on the right, together with the abnormal pulsation beneath the inner end of the right clavicle, made the diagnosis of aneurismal

dilatation of the innominate artery practically certain. The breadth of the area of dullness over the vessels just above the base of the heart, with the tracheal tug and the associated aneurism of the innominate artery, made it highly probable that an aneurism existed in the ascending and transverse parts of the arch of the aorta. Physical examination gave no signs of the involvement of the descending arch.

Confirmation of the diagnosis was sought in an X-ray examination, which was very kindly made for me by Dr. F. H. Williams. The accompanying diagram shows the outline of the shadow cast by the heart and great vessels as shown by the fluoroscope.

The point of special interest in the X-ray examination lies in the shadow with a curved outline above the left base of the heart. This shadow extends upward three inches from the base of the heart,



Fluoroscopic examination.
Fusiform aneurism of ascending, transverse,
and descending arch.

and outward to the left one and one-quarter inches beyond the origin of the great vessels. Distinct expansile pulsation was seen in this shadow just following the systolic contraction of the ventricle. It evidently shows an aneurism of the aorta extending from the transverse arch to the descending arch. A distinct outline of the innominate aneurism could not be obtained, perhaps owing to its relation to the shadow of the spine.

By the fluoroscopic examination we made certain the existence of an aneurism of the arch of the aorta, whereas before we had great probability rather than certainty; we also learned that this aneurism extended to the descending arch, of which fact the ordinary physical examination did not give even a clue, and we were able to outline the size of the heart more accurately than by percussion.

Auscultation of the lungs was normal. At the aortic area a systolic and a diastolic murmur were heard. The systolic murmur had its maximum intensity at the aortic area. It was transmitted upward along the aorta and could be heard faintly in the left carotid and subclavian arteries. Just at the inner end of the right clavicle the systolic murmur was markedly reinforced, and had an intensity even greater than at the aortic area (a condition which was interestingly explained by the post-mortem findings), and the murmur was quite loud in the right carotid and subclavian arteries. The diastolic murmur could not be heard in the left carotid or subclavian arteries, but was faintly audible in the corresponding vessels of the right side. Its maximum intensity was at and just above the aortic area. It was not carried downward beyond the anatomical position of the aortic valve (the junction of the third left costal cartilage with the sternum), and at this point the second sound became distinct and somewhat accentuated. A slight systolic murmur was also heard at the apex, but was not transmitted far in any direction. It was not clear whether this was the aortic systolic murmur transmitted or not.

The diagnosis was aneurism of the aorta, involving the ascending, transverse, and descending portions of the arch; aneurism of the innominate artery, and regurgitation at the aortic valve. The patient was ordered to take iodide of potash, beginning with ten grains three times daily, and he was especially cautioned against sudden or marked exertion.

On February 23d, five days later, he returned. He felt much better. The pain in the shoulders and upper chest was much less, but he still complained of the præcordial pain. Physical examination was the same, except that the radial pulse was fuller and apparently stronger. His condition was apparently improved. The dose of iodide of potash was increased to fifteen grains.

This observation was made about 1 P.M. The patient was found not long afterward on a door-step not far from the hospital. He looked sick and distressed, and was very weak. He told the person who found him that he had been at the hospital for treatment. This person assisted him to arise, but finding he was too weak to walk, he left him and hurried to the hospital. An ambulance was sent at once. The patient was carried directly to the ward, and was seen immediately by the house physician. He was in collapse, pale, and with no radial pulse. His mind was clear. He did not respond to stimulants, but grew steadily worse and died in about ten minutes. This was about one hour after he was seen at the out-patient department. It was naturally supposed that the aneurism had ruptured.

At the autopsy the following pathological conditions were found: Fusiform aneurism of the aorta and of the innominate artery; general arterio-sclerosis, with calcification and atheroma; general chronic passive congestion; oedema of the lungs.

The aneurism had not ruptured. The heart was enlarged, somewhat

distended with blood, and the right auricle was especially full and prominent, extending further to the right than normally. The aneurismal sac was not tensely distended, but rather relaxed. The aneurism was of the fusiform type, involving the whole of the arch. There was also a fusiform aneurism of the innominate artery, extending from its origin at the aorta to its bifurcation.

The circumference of the aortic valve was 8.5 cm.; six centimetres above this the circumference of the aorta was 13½ cm.; at the innominate artery it was 13 cm.; at the left subclavian artery, 10 cm., and at the first intercostal branches, 7 cm. The opening into the innominate artery had slightly thickened walls and measured 1.1 cm. in diameter. The artery gradually increased in size as it ascended, until it reached a diameter of 2½ cm. From this point the enlargement gradually diminished to the division into the carotid and subclavian arteries, which were both normal in size. There was a marked general arteriosclerosis, with calcification and some atheroma, involving the whole extent of the aorta.

The autopsy, then, thoroughly confirmed the diagnosis, and showed that the extent of the aneurism had been accurately recognized. Moreover, the shape of the innominate aneurism gave an adequate explanation of the peculiar intensity of the systolic murmur near the clavicle, namely, that a new murmur was here created as the blood rushed through the narrow opening into the larger space of the dilated innominate artery beyond.

Death came from the cardiac complication. The existence of aortic regurgitation had been recognized clinically by the aortic diastolic murmur, the enlargement of the left ventricle downward and outward, and the characteristic jerky pulsation of the carotids. At the autopsy the aortic cusps were practically normal, but there was dilatation and slight hypertrophy of the ventricle, and the aortic orifice was somewhat stretched, sharing in the dilatation of the aorta just above. This is shown by comparing the measurements of this case with the normal measurements (Mallory and Wright').

| | Normal | Case II. |
|-------------------------------------|---------------|-------------|
| Circumference of aortic orifice, | 7.7- 8 cm. | 8.5 cm. |
| " mitral " | 10.4-10.9 " | 10 " |
| " pulmonary orifice, | 8.9- 9.2 " | 8 " |
| " tricuspid " | 12 -12.7 " | 12 " |
| " ascending aorta, | 7.4 " | 8.5-13.25 " |
| " pulmonary artery, | 8 " | ? " |
| Thickness of left ventricular wall, | 0.7- 1 " | 1.2 " |
| " right " " | 0.2- 0.3 " | 0.4 " |

Here we find the measurements of all other orifices corresponding pretty closely to the minimum figures of the normal table, while we find the aortic orifice nearly one centimetre larger than the minimum figure of the normal table.

The autopsy also showed a general chronic passive congestion of the organs. This suggests that the systolic murmur heard at the apex was due

to mitral regurgitation. This regurgitation was secondary to the dilatation of the left ventricle, as the mitral curtains and chordæ tendinæ were normal. The œdema of the lungs, the distention of the cavities of the heart, and the clinical story of the rapid failure and death all point to failure of the heart muscle as the immediate cause of death. The muscle seemed to be in good condition, and microscopical examination failed to find fatty degeneration. The coronary arteries were patent. We have to fall back on the well-known clinical fact that death is apt to ensue suddenly in aortic regurgitation—a fact for which we are not yet always ready to furnish a satisfactory explanation. We are apt to think of death from aortic regurgitation as more rapid than this, which lasted somewhere between one-half and one hour. But Balfour² has called attention to the fact that ingravescent asystole occurs in aortic regurgitation, and may even be much more prolonged than in this case.

Our story in this case would seem to be clear, and the sequence of events as follows: Syphilis, arterio-sclerosis of the aorta, fusiform aneurism of the arch, resulting dilatation of the aortic orifice, aortic regurgitation, dilatation of the left ventricle, mitral regurgitation, general passive congestion, and death from asystole accompanied by œdema of the lungs. The responsibility of the aneurism for the death is indirect.

This case illustrates so well the view of fusiform aneurisms held by Sir Douglas Powell that I will quote a few of his remarks on the subject. He says: “Clinically the phenomena characteristic of aneurism are scarcely ever observed except in association with the sacculated form, the so-called ‘fusiform aneurism’ being merged, as regards prognosis and treatment, in the class of heart disease with which they are most closely connected. These general dilatations involve, for the most part, the first part of the aorta, and are associated with valvular lesions and secondary cardiac dilatations and hypertrophies; they give rise, with rare exceptions, to no pressure signs; they do not tend to cause death by rupture or by compression of vital parts, but rather through cardiac failure, angina, or syncope. The treatment is that appropriate to heart disease.”

Class III. includes by far the greatest number of cases. It is true in the vast majority of cases of aneurism of the aorta that if death is not due to rupture it is due, directly or indirectly, to pressure.

In its course from the left ventricle to the diaphragm the thoracic aorta lies in close relation to many important structures. It is worth while, in the study of the effects of pressure by the aneurismal sac, to briefly recapitulate those organs which may be affected.

The origin of the aorta is embedded in the structures at the base of the heart. In front lie the conus arteriosus of the right ventricle and the auricular appendage of the right auricle. The right auricle and

the vena cava superior are in close contact on the right. Around the left the pulmonary artery winds from the front, while behind lie the left auricle and the right pulmonary artery.

Just above the heart the edges of both lungs separate the ascending arch of the aorta from the sternum in front. The pulmonary artery is on the left. Behind and below are the right branch of the pulmonary artery and the root of the right lung. On the right lies the vena cava superior and the right lung. It should be remembered that the pericardium extends up on the aorta from one to one and one-half inches above the base of the heart.

The relations of the transverse arch are perhaps the most important of all. The ascending arch has a certain freedom of movement, and after it emerges from the structures at the base of the heart it may become considerably enlarged without serious results to surrounding organs—it displaces rather than compresses them. The transverse arch has much less freedom of movement, its relation to surrounding parts is closer, and these parts cannot readily be moved aside. Enlargement here causes compression rather than displacement. Symptoms of pressure, therefore, develop early in the process of enlargement.

The transverse arch passes backward and to the left in front of the trachea and above the left bronchus. The bifurcation of the pulmonary artery is beneath the anterior part of this section of the aorta. Above lies the left innominate vein. Further back than the trachea lie the œsophagus and thoracic duct. The relations of the nervous structures to this part of the aorta are important. The deep cardiac plexus lies behind the aorta, between it and the trachea, and above the bifurcation of the pulmonary artery. This is the main centre for the nerves distributed to the heart. The superficial cardiac plexus lies between the aorta and the right pulmonary artery. The left recurrent laryngeal nerve passes down in front of the aorta, passes backward beneath it, and ascends behind it and in front of the trachea. In front also pass the left vagus and phrenic nerves. From the transverse arch are given off the innominate, the left carotid, and the left subclavian arteries.

The descending arch passes backward and downward to the left of the trachea, behind the root of the left lung, to the left anterior aspect of the dorsal vertebræ. As it passes downward the œsophagus and thoracic duct lie on its right. The left pleura and lung lie to the left and anteriorly. The descending arch of the aorta and the thoracic aorta below are held firmly in place by the intercostal branches which are given off from the aorta.

We may now turn our attention to the ways in which pressure from an aneurism upon the organs surrounding the aorta may contribute to a fatal result. It is clear that the results will depend partly upon the

size of the aneurism and partly upon the vital importance of the organ or organs pressed upon. It is also manifest that the size of the aneurism is of less importance than the place where the pressure is exerted. A striking case to illustrate this point is reported by Irvine.¹⁰ It was a case of double aneurism of the arch of the aorta. A relatively large aneurism of the ascending arch caused very little trouble, whereas death came because a small aneurism of the descending arch compressed the left bronchus and induced extensive changes in the left lung.

Before considering the effect on individual structures, however, let us note that death may come from the gradual wearing out of the patient's strength without any extreme pressure on vital organs. When an aneurism enlarges enough to press upon peripheral nerves pain results. Pain may also be of the nature of "referred pain," as in cardiac disease. Pressure on these nerves may also cause dyspnoea or cough, especially of a paroxysmal type. The suffering from the pain, the dyspnoea and cough, and sleeplessness due to any of these factors are the main causes of death in some cases, and such death may be properly called "death from exhaustion."

Pressure on the nerves may contribute to the death in other ways. Spasm of the glottis may result reflexly from irritation of the vagus nerve. And Gibson¹¹ says the same result "may possibly be an early symptom of interference with the motor nerve of the larynx," the recurrent laryngeal. To what extent pressure on the cardiac nerves or ganglia may exert an inhibitory or paralytic effect on the cardiac muscle is not perfectly clear. It is mentioned by some writers, and it may be a possible explanation of some of the cases where the most careful post-mortem examination fails to reveal any adequate explanation. This subject needs further investigation, as does also the question of what effect pressure on the vagus may have on the condition of the lung.

In considering the effect of pressure on the heart and the great vessels, we may practically disregard the left ventricle and the aorta, for it is from the left ventricle and through the blood in the aorta that the pressure is carried to the periphery of the aneurismal sac. We cannot expect this pressure to be greater than its source, hence it cannot compress the left ventricle or aorta. An aneurism arising at the root of the aorta may, however, by pressure distort either the aortic or pulmonary valve. In either case we get regurgitation into the corresponding ventricle as the chief factor in causing cardiac failure and death. Compression or obliteration of the innominate or left carotid or left subclavian arteries may occur. This leads to impaired nutrition of the parts supplied by these arteries, but the collateral circulation is so good that a fatal result is hardly to be expected from this cause alone, unless all three were occluded together, of which occurrence I have found no reported case. The supply of blood to the brain is, of course, the most

important item in considering obstruction of these arteries. Of the other branches of the general arterial system we need consider only the bronchial arteries—the nutrient arteries of the lung. Obliteration of one of these will cause gangrene of the part of the lung supplied by it, with fatal termination. This is a less frequent occurrence than was at one time supposed, as the bloodvessels of the roots of the lung are so situated that they are generally protected by the resistance of the bronchi.

Returning to the heart, we have to consider pressure upon the auricles, the conus arteriosus of the right ventricle, the pulmonary artery, and the pulmonary veins. In these cases we are dealing with obstruction of the circulation, essentially the same in its results as is found in valvular disease. Death may result in the same way as from broken compensation. Pressure on the vena cava superior is by no means uncommon. We get venous stasis of the upper part of the chest, the head, and the upper extremities. Collateral circulation may lessen this by furnishing a channel to the vena cava inferior, by anastomosis with its branches, or to the vena azygos if the pressure be above the point of entrance of that vessel into the vena cava superior. Here, again, the brain is the important organ to consider, and death by coma from the obstructed venous circulation is reported. (Edema of the glottis has also been reported¹² as a cause of death from impeded venous circulation.

Case III. is an illustration of death caused by pressure interfering with the circulation. The point of application was the pulmonary artery.

Male, aged forty-two years, painter, came to the out-patient department of the Boston City Hospital in the latter part of September, 1900. He had diphtheria eleven years ago. Otherwise he had been in good health, except for syphilis ten years before. He had used alcoholic drinks and tobacco to excess.

His present illness began about one year ago. He began to get short of breath on exertion, and also had vertigo and palpitation. For some months he had had pain at times in the præcordial region, radiating into the shoulders. Of late the pain had been worse and more frequent, and he complained of increasing dyspnoea and weakness.

On physical examination the cardiac area of dulness was practically normal. At the base of the heart was to be heard a peculiar murmur. It was loud and harsh in quality. Its point of maximum intensity was to the left of the sternum just above the pulmonic area. In rhythm it began just after the first sound—that is, it was a somewhat late systolic murmur—and continued just a trifle after the second cardiac sound; yet it was clearly all one murmur, and not a systolic running into a diastolic murmur. There was also a loud, ringing, markedly accentuated second pulmonic sound.

This peculiar combination is characteristic of a persistent ductus arteriosus. But it seemed highly improbable that such a case should live to this age without a longer history of cardiac trouble. The only other explanation which seemed at all likely was the existence of an

aneurism which compressed the pulmonary artery, and thus raised the tension in the pulmonary circulation. There was, however, no dullness, no pulsation, no tracheal tug, no hoarseness, no inequality of pupils, or any other physical sign to confirm the suspicion of aneurism of the arch of the aorta. He was asked to return for X-ray examination, but did not return till December 7th, when he was admitted to the hospital.

He was now much worse. He was weaker, had much more pain, more dyspnoea, had been unable to do any work for three weeks, and had had orthopnoea for two weeks. He had also become hoarse at these times. He was pale and had a slight cyanotic hue. The pulse was 80, regular, and of fair volume and strength. There was no perceptible difference in the two wrists. A distinct tracheal tug could now be felt.

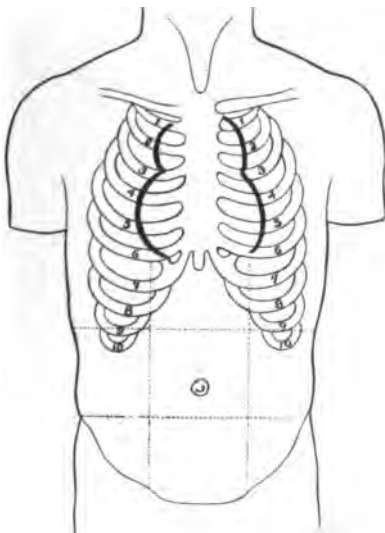
There was now a slight bulging of the chest just to the left of the sternum, from the second interspace nearly to the clavicle. Over this area a slight pulsation could be seen; palpation revealed a distinct, coarse, rough thrill; percussion showed marked dullness; and on auscultation a rough, coarse, rumbling systolic murmur was heard. The second pulmonic sound was still somewhat accentuated, but was not as loud or clear-cut as before. Moreover, it was followed by a diastolic murmur, with its maximum intensity in the pulmonic area, transmitted downward and diagonally across the sternum to the right — following the location of the right ventricle and not the left.

This murmur seemed to me to indicate pulmonary regurgitation. It was now perfectly evident that we had an aneurism pressing front to the chest wall to the left of the sternum. This had in September been too small to be detected, but so situated as to press on the pulmonary artery and greatly increase the resistance in the pulmonary circuit. This obstruction had now increased so much as to cause regurgitation through the pulmonary valve.

As a matter of interest an X-ray examination was made in this case also. It showed the heart slightly enlarged to the left, but markedly enlarged to the right. Above the base of the heart was an abnormally broad shadow, extending especially to the left of the sternum and very closely resembling the shadow of the aneurism of the descending arch, as shown in Fig. 1. The outline is shown in Fig. 2.

This patient gradually failed, and died December 17, 1900. Toward the end the attacks of dyspnoea were more frequent and severe. He had to be propped up in an upright sitting position for the last forty-

FIG. 2.



Fluoroscopic examination.
Sacculated aneurism of the ascending arch, growing forward and to the left, and compressing the pulmonary artery.

eight hours of life. During the last few hours he developed pulmonary œdema and his pulse gradually failed.

The autopsy showed a sacculated aneurism of the aorta compressing the pulmonary artery; hypertrophy and dilatation of the right ventricle; hypostatic congestion of the lower lobes of the lung, and atelectasis of an area of left upper lobe of lung, which was compressed by the aneurism; chronic passive congestion of liver, spleen, intestines, and kidneys; fatty degeneration of the heart and kidneys.

On removing the sternum the mediastinum presented a roughly spherical mass about 10 cm. in diameter, projecting forward and to the

FIG. 3.



Interior of left ventricle and aorta, showing opening into aneurismal sac just above the cusps of the aortic valve.

left. The mass is continuous with the heart and vessels of the base, and lies anterior and to the left of them. It projects 8.5 cm. to the left of the median line at about the level of the second rib. On dissection the mass is found to be an aneurismal sac 11 x 9 x 7.5 cm., which communicates with the aorta by an oval opening 2.75 x 2 cm. in diameter. This opening is situated in the anterior wall of the first portion of the aorta 3 mm. above the upper margin of the aortic cusps. The opening is on the left anterior aspect, above the junction of the anterior and the left posterior cusps of the valve. The edges of the opening are smooth and slightly raised. (See Fig. 3.)

Inside the sac a rounded mass of firm, organized clot fills the cavity to within $\frac{1}{2}$ cm. of the opening. This mass becomes firmly adherent to the inner wall of the aneurismal sac at $\frac{1}{4}$ to 1 cm. from the edge of the opening, except at the posterior side of the opening. Here a flattened pocket extends 3 cm. in depth between the rounded sac on the outside and the rounded mass of clot on the inside. The portion of this pocket farthest from the aortic opening is directly opposite the sinus of the right anterior cusp of the pulmonary valve. It is evident that this pocket is so situated as to have caught the greatest force of the blood

FIG. 4.



Aneurismal sac laid open, showing layered clot within.

current entering the sac, and this fact has prevented the formation of blood clot at this point. The cause of the peculiar murmur heard in this case was the eddying blood current in this pocket of the aneurismal sac.

Elsewhere the whole aneurismal sac is filled with firm clot. An incision from the anterior surface of the tumor shows a firm, fibrinous, dark red, layered clot, everywhere adherent to the inside of the sac. (See Fig. 4.) This aneurism not only had not ruptured, but it presented that condition, filled with firm clot, which is called "cured."

The general direction of the growth of the aneurism has been to the left and forward. It has also extended upward, as two-thirds of the mass is above the opening from the aorta. Still more important is the growth of the tumor a short distance backward, crowding in between the aorta and the pulmonary artery, so that where the transverse arch should cross close above the pulmonary artery it is separated from it by an aneurismal mass 5 cm. in thickness. The arch of the aorta has been apparently pushed upward, backward, and to the right to accommodate this growth; but there has been little, if any, diminution of the lumen of the artery.

FIG. 5.



Interior of right ventricle and pulmonary artery. Cusp of pulmonary valve stretched taut over bulging surface of aneurism.

In the pulmonary artery, however, a different condition obtains. There has been marked flattening from the encroachment of the tumor upon its lumen. The tumor presents as an irregular rounded mass, projecting downward and to the left into the pulmonary artery and the upper part of the right ventricle. (See Fig. 5.) The right anterior cusp of the pulmonary valve is stretched taut over the rounded surface of the tumor. The tumor projects into the right ventricle just below this valve, and the distance over its rounded surface is 4 cm. from the right ventricular wall to the upper edge of this cusp, and from this point to the end of the projecting mass in the pulmonary artery is 7 cm. The tumor mass as felt from the right ventricle or pulmonary artery is firm

everywhere except over the shallow pocket of the aneurismal sac already mentioned. This pocket lies behind the right anterior cusp of the pulmonary valve and part of the pulmonary artery just above it. Though yielding in the post-mortem specimen, it must have been firmly distended by the pressure of the aortic blood during life. The right anterior cusp of the valve must have lain flat over the surface, useless as far as its function was concerned.

The right ventricle was very much dilated, and the wall was hypertrophied to about four times its normal thickness. Death came from failure of the right ventricle, owing to the obstruction in the pulmonary artery and the regurgitation through the crippled pulmonary valve.

Pressure on the trachea or the primary bronchi is a frequent cause of death in aneurism of the aortic arch. This may act directly or indirectly. When it acts directly, death comes by suffocation, and the pressure is usually on the trachea just above its bifurcation. Death from this cause is rarely sudden; it is generally by slow asphyxiation, and the suffering is terrible. Sometimes the attack of asphyxiation is rather rapid in course, and unconsciousness ensues before there is much suffering, as in the following case:

CASE IV.—Male, aged thirty-nine years, laborer, entered the Boston City Hospital, December 6, 1900. Family history was negative. He had had typhoid fever when young, and remembered no serious illness since then. At twenty-seven he had syphilis. Used alcohol moderately, tobacco to some excess.

His present illness began in August, 1900, four months before, when he began to have occasional spells of difficult breathing. He felt an attack coming on, would then sit down, and soon lost consciousness. If severe, the attack lasted about one and one-half hour. The attacks were followed by great prostration. The attacks came on rather suddenly after exertion or excitement. He had one just before entrance to the hospital; another five days before, and another five weeks before that. The intervals between the attacks had been variable. In the attacks breathing was extremely difficult, and he got blue in the face.

In the intervals between attacks he had pain and discomfort in the præcordia. There was some cough, but little expectoration. The voice had been husky since September.

Physical examination showed the pupils equal, the left vocal cord immovable, and the radial pulse equal and of good volume and tension. Tracheal tug was present. There was no œdema. Examination of lungs was negative. Upper border of cardiac dulness was at the third rib. The right border was one and one-half inches to the right, and the left border five and one-half inches to the left of the median line. Apex-beat was in the fifth interspace. A soft, systolic murmur was heard at the base, loudest to the left of the sternum. The second pulmonary sound was accentuated. Physical examination was otherwise negative.

Up to January 21, 1901, a period of about six weeks, he had three attacks—all of them severe. The dyspnoea was especially marked on expiration. He became cyanotic and lost consciousness. He recovered from these attacks, but was exhausted and weak for hours afterward.

The hoarseness had continued, but varied in degree. At times he had a metallic, barking cough. There was dyspnoea on any marked exertion. He was able to be up and about the ward most of the time.

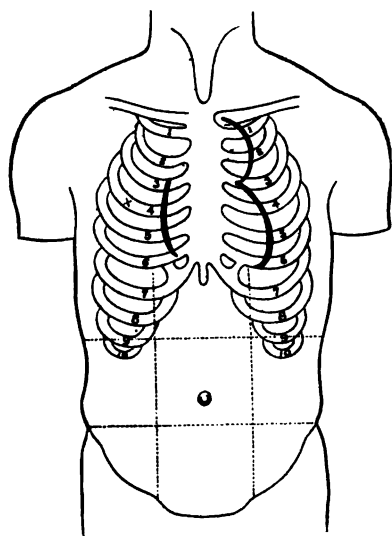
An X-ray examination had been made on December 21, 1900, and showed the following outline. (See Fig. 6.)

From January 21st to April 18th he had five attacks at varying intervals. On May 3d he had an attack. On May 5th and 6th he had four attacks. On the night of May 7th he had an attack during which he died.

The autopsy showed the following: sacculated aneurism of the transverse arch of aorta, and dilatation of the ascending arch; obstruction of trachea by pressure of aneurism; distention of lungs; chronic healed tuberculosis of lung; arterio-sclerosis; and hypertrophy of heart.

The heart was somewhat enlarged, weighing 480 grammes, the principal change being in the left ventricle. The muscular tissue was firm and dark red, but showed some fatty degeneration on microscopical examination. The aorta is apparently dilated, and a mass about the size of a lemon lay below the clavicular notch, extending slightly into the left pleural cavity. This was a sacculated aneurism of the transverse arch of the aorta, which had not ruptured.

The aortic orifice measured 8.5 cm. in circumference, and the ascending arch was somewhat dilated, measuring from 9 to 10.5 cm. in circumference. At a point 9.5 cm. from the aortic valve there is a rounded border forming part of the



Fluoroscopic Examination.
Sacculated aneurism of transverse arch,
pressing on trachea.

opening of an aneurismal sac. This opening measured 8 by 9 cm. in diameter. This sac arose almost entirely from the posterior and inferior part of the transverse arch, while the innominate, left carotid, and subclavian arteries arose in front from the apparently normal aortic wall, and were practically normal. The sac was hemispherical in shape, and the greatest circumference of it and the dilated aortic wall was 22 cm.

The aneurism lay directly anterior to the trachea just above its bifurcation. The trachea was flattened and curved back by the pressure, and its lumen was encroached upon from the anterior and left side by the bulging wall of the aneurismal sac. Even with the sac empty the lumen of the trachea was only one-third of its normal size at this point.

The lungs were very much distended with air. When the sternum was removed they did not collapse, but bulged forward, nearly covering the heart. On cutting the bronchi the lungs collapsed to less than one-half their former size from the escape of air. There was an area of

healed tuberculous tissue occupying about one-third of the right upper lobe. Elsewhere the lung tissue was normal. The bronchi were normal. The bronchial lymph nodes were normal.

It was clear that the distention of the lungs was due entirely to the pressure of the aneurism on the trachea. The pressure of the distended sac must have been sufficient to entirely prevent the exit of air, and the patient died of suffocation. It is not difficult to understand how the air passed by this obstruction on inspiration, but could not escape again. With the descent of the diaphragm on inspiration the heart and anterior part of the arch are drawn downward. This drew the aneurism away from the trachea enough to allow the entrance of air, but when this traction ceased at the end of inspiration the pressure was at once resumed, and the aneurism acted like a ball valve to prevent the exit of the air. The resulting dyspnoea caused more forcible inspiratory efforts, the entrance of more and more air which could not escape, and the extreme distention of the lungs resulted. With the complete distention of the lung no more air would enter, and respiration was brought to a standstill, and death ensued from suffocation.

Compression of one bronchus is not so likely to cause death from suffocation. It is rarely so sudden that the necessary supply of air cannot be obtained through the other lung. Death is more likely to come through secondary changes in the lung. How far these secondary changes may be the result of pressure upon the bloodvessels or nerves at the root of the lung has been a matter of dispute, and I do not think we are yet prepared to decide the question definitely. It is, however, certain that the mere mechanical occlusion of the bronchus may result in extensive and fatal changes in the lung tissue. This is due to the retention of secretions and the invasion of micro-organisms. Collapse and consolidation of lung tissue, a purulent bronchitis extending to the smaller divisions, and then to the lung tissue, and causing multiple foci of suppuration, and gangrene, are the manifestations of pulmonary disintegration which follow compression of the bronchus. Death in this case comes as an indirect result, not as the direct result of the pressure of the aneurism. Irvine¹⁰ has reported a case of this sort, and has called attention to the subject. Compression of one primary bronchus may, however, produce collapse and atelectasis of the corresponding lung without further secondary results, as is shown in Case V.

Finally, in considering the effects of pressure on the pulmonary tissue, we must note the occurrence of cases where the aneurism is so situated that it does not press on the main air passages, but upon the lung tissue itself. Here we get primarily a compression of the lung tissue, and may get secondary inflammatory processes in the tissue thus affected.

Pressure on the œsophagus may become so extreme as to prevent the passage of food and cause death by inanition. In less degree it may be

a more or less important factor in causing insufficient ingestion of food and imperfect nutrition, thus rendering the patient an easier victim to intercurrent disease of any kind. Compression of the thoracic duct may cause serious impairment of nutrition with similar relations to the fatal termination, but this is extremely rare.

The following case illustrates death by starvation from the pressure of an aneurism on the œsophagus.

CASE V. Female, aged fifty-seven years, seamstress, had always enjoyed good health before the present illness. In the fall of 1893 she first noticed a pain in the lower left axillary region, which also shot into the back and up to the left shoulder. This continued at intervals.

In the fall of 1895 she began to have partial aphonia and difficulty in swallowing. After a time solids could not be swallowed at all, but she could swallow liquids. By the last of January, 1896, a hacking cough had set in. Sibilant and sonorous râles were heard over both lungs. By March there was considerable expectoration, which was occasionally streaked with blood. By April dyspnoea was caused by even slight exertion. She was very weak. She had been losing flesh since fall, and was now greatly emaciated. Even liquids were now vomited or regurgitated, and nutritive enemata were resorted to after April 5th. She failed gradually, and died quietly on June 2, 1896. No new symptoms had developed, and all the old ones had persisted.

During the latter part of her illness it was noticed that the respiration was very faint on the right side of the chest. Tuberculosis of the lung was thought of, but the diagnosis would hardly account for all the symptoms, and no tubercle bacilli could be found in the sputum. The probable diagnosis was thought to be a malignant tumor of the œsophagus. This would account for the obstruction of the œsophagus, the wasting, and the pain, and, by extension and pressure on the left recurrent laryngeal nerve and right bronchus would account for the aphonia and diminished respiration of the right lung. This clinical diagnosis represented very closely the autopsy findings, except that the tumor was an aneurism of the aorta instead of a malignant tumor of the œsophagus.

The autopsy showed a sacculated aneurism of the thoracic aorta; atheroma of the aorta; pressure of aneurism on right primary bronchus and on œsophagus; atelectasis of right lung; bronchitis of left lung, and all other organs normal.

The aneurism was a sacculated one, about three and one-half inches in diameter, arising from the upper part of the descending thoracic aorta. It had grown forward and to the right, crowding in between the œsophagus behind and the air passages in front. The œsophagus was compressed flat against the vertebral column. The right primary bronchus was so completely occluded by the pressure toward the front that the right lung had collapsed and was in a state of atelectasis, but no inflammatory change had resulted. The fourth to sixth dorsal vertebrae (inclusive) were eroded on their right side from the pressure of the sac. The aneurism was partly filled with lamellated clot, and had not ruptured. The thoracic duct could not be found. The anatomical relations of the aneurism, the absence of other pathological cause, and the very extreme emaciation showed that death came from starvation, of which the aneurism was the immediate cause.

I wish to acknowledge my indebtedness to Drs. Withington, Williams, and Sears, of the staff of the Boston City Hospital, for permission to report the progress of Cases I., III., and IV. while under their charge in the wards; to the pathological department of the hospital for the excellent reports of autopsies; to Dr. H. C. Williams, of Boston, for the history of Case I., and to Drs. S. H. Ayer and W. H. Prescott for the clinical history and autopsy report of Case V.

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THE ASSOCIATION OF PULMONARY TUBERCULOSIS WITH
BOTH PRIMARY AND SECONDARY ENDOCARDITIS,
AND THE EFFECT OF VALVULAR DISEASE
UPON LUNG TUBERCULOSIS.*

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THE subject of the relationship existing between diseases of the endocardium and pulmonary tuberculosis is admittedly but imperfectly understood. The cases of endocarditis met with in tuberculosis of the lungs are clearly divisible into several classes, as follows: 1. Those due to the presence of tubercle bacilli within the heart—*endocarditis tuberculosa*. 2. Those that are secondary to tuberculosis or merely intercurrent, and caused by various organisms other than the tubercle bacillus. 3. Various forms of valvular heart disease that precede the tuberculous infection of the lung and are due to rheumatism and other etiological agencies.

A distinction between these different varieties has not, as a rule, been

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drawn by writers, hence the important practical question of the relative frequency of the occurrence of each in association with tuberculosis of the lungs has not been definitely settled. These varied classes of cases demand separate study if we would obtain positive and practical information on the general subject. Thus the mere detection of the co-existence of pulmonary tuberculosis with acute or chronic endocarditis at necropsy, without knowing anything of the special causation of the cardiac lesions, their nature, or whether a tuberculous lesion of the endocardium exists or not, is of no scientific and little if any practical value.

Although we are sadly in need of reliable and more elaborate statistics, it is my purpose, in connection with a general consideration of the practical phases of the subject, to tabulate all recorded cases according to the classification given above, satisfied that a collective investigation and a discriminating study made on these lines will lead to greater unanimity in establishing conclusions.

1. *Cases of endocarditis due to the tubercle bacilli.* In 1806 Corvisart¹ pointed out the existence of little tubercles situated on the mitral valve in an individual afflicted with tuberculosis of the lungs and the pericardium, coupled with caries of the ribs and vertebræ. It remained, however, for Wagner² to give the first description of tuberculosis of the endocardium. In 1886 Lancereaux observed a case of endopericarditis of a tuberculous nature. From the date of the observations and more complete studies of Letulle (1874) and of Perroud (1875) a proper classification of the lesions of tuberculous endocarditis was attempted. Later, Kundrat also found the tubercle bacillus in the endocardium, and the observation has been abundantly confirmed by others. It is interesting to note that while Ponfick, Weigert, Sticker, and others have found the tubercle bacilli commonly present in the blood, true tuberculous endocarditis is a truly rare condition. Perroud long since pointed out that small aortic vegetations sometimes occur in children dying of general miliary tuberculosis. Indeed, involvement of the endocardium is most apt to occur in acute miliary tuberculosis; but we are not especially concerned here with this clinical variety of tuberculosis. I shall also endeavor to exclude the cases of tuberculosis of the endocardium resulting from direct extension from adjacent parts, as in tubercular invasion of the mediastinal glands.

The fact is to be emphasized at the outset that the endocardium, like the interior of the bloodvessels, is to an unusual degree resistant to the tubercle bacillus. Infection of the endocardium most probably takes place through the blood-supply to the heart structures, or possibly through that which finds its way into the cavities from the endocardium, and not the blood circulating within the chambers of the organ. It is known that when the arteries in a tuberculous area become involved the adventitia, and not the intima, is first affected. The endocardium

then fulfils a decidedly protective function so far as invasion of its structure by tubercle bacilli is concerned. In this connection it is interesting to note that, as regards frequency of occurrence, Bollinger gives cardiac tuberculosis fourteenth place among the viscera. Here it should be recollected also that the pericardium and the myocardium are oftener the seat of tuberculosis than the endocardium.

Teissier,¹ in an extensive monograph on the subject of tuberculous endocarditis, states that during a period of three years he examined minutely the hearts of all cases of tuberculosis—chronic, acute, and miliary—and was unable to find a single example of true tuberculous endocarditis. He cites Potain's case of tuberculous myocarditis and Landouzy's numerous cases of infantile tuberculosis in which the endocardium was uniformly free from tuberculous lesions.

The figures in the accompanying Table I. (cited mostly from Teissier's monograph⁴) are derived from the earlier literature on the subject, and they serve to emphasize the then prevailing view that tuberculous endocarditis in the course of pulmonary tuberculosis practically never occurs.

TABLE I.

| Name. | Total number of necropsies. | Cases of tuberculous endocarditis. | Percentage of cases. |
|-------------------------------|-----------------------------|------------------------------------|----------------------|
| Sanger | 469 | 3 | 0.64 |
| Willig | 845 | None. | |
| Reisner | 152 | " | |
| Chambers | 566 | " | |
| Total number of cases | 2082 | 3 | 0.148 |

The figures gleaned from more recent available sources, however, and tabulated below (Table II.) convey a different idea as to the frequency of the occurrence of acute endocarditis in the course of pulmonary tuberculosis, although it is unquestioned that a close analysis of the cases recorded shows the great majority of them to have been due to excitants other than the tubercle bacilli. In cases in which the lesions were of the ulcerative variety, at all events secondary pyogenic infection probably existed.

TABLE II.

| Name. | Number autopsies in tuberculosis. | Recent endocarditis. | Per cent. | Remarks. |
|------------------------------|-----------------------------------|----------------------|-----------|--|
| Oster ⁶ | 216 | 12 | 5.5 | Mitral valves 8, aortic 3, a. and m. 1. |
| Bernard Schultz ⁶ | 6987 | 67 | 0.966 | In 42 other cases the endocarditis was primary. |
| Percy Kidd ⁷ . | 500 | 6 | 1.2 | In 21 other cases the endocarditis was probably due to other causes. |
| Teissier ⁸ . . . | 100 | 82 | 82 | In 12 the tubercle bacilli were present on the valves (cited cases). |

Additional statistics are to be found in the literature, but they bear upon the general subject of valvular disease associated with phthisis, or indicate no attempt to discern recent tuberculous endocarditis from other forms of endocarditis, hence they are not available for my present purpose. Thus Krygen⁹ autopsied 1100 cases of tuberculosis, and in 10 found valvular lesions associated.

H. Walsham¹⁰ met with 21 cases of endocarditis in 130 sections of cases of tuberculosis, but no mention is made of the particular varieties of endocarditis encountered.

As before mentioned, it is not improbable that the cases of recent endocarditis included in the above table were due to causes other than tuberculosis, for the reason that these cannot readily be excluded. Osler expresses doubt concerning the tuberculous nature of his own cases as well as of those reported by others. As opposed to this view it must be recollected that that eminent authority has favored us with the avowed opinion that more than one-half of the cases of endocarditis associated with chronic phthisis, even including the sclerotic variety, are secondary to the pulmonary lesions. This aspect of my subject is of great practical importance, and there is need for more extended study and investigation.

Clinically, the cases of tuberculous endocarditis are extremely difficult of recognition. The history of the case, however, may be of diagnostic significance. If it can be shown that the cardiac affection developed subsequent to undoubted pulmonary tuberculosis, and if rheumatic and other forms of infectious endocarditis can be eliminated, and especially if there have been neither previous arterio-sclerosis nor fibroid degeneration of the viscera, then a reasonably certain diagnosis of tuberculous endocarditis, given the usual signs and symptoms, can be made. The condition may also be overshadowed by the severer symptoms of the closing stage of pulmonary tuberculosis.

The clinical association of pulmonary tuberculosis with recent endocarditis is illustrated by a single case out of a total of ninety-two cases of tuberculosis of which I have satisfactory records.

A. T., aged thirty years; occupation, clerk; without vicious habits. Predisposition to tuberculosis was an inheritance from both sides of the ancestry. No previous rheumatism, although the patient had had scarlet fever and measles during childhood. Pulmonary tuberculosis developed at twenty-four years of age and pursued a comparatively rapid course, terminating fatally at the end of eighteen months amid the signs of extensive cavity formation. February 1, 1894, six weeks prior to death, a distinct soft bruit made its appearance at the apex of the heart; the area of transmission, however, was quite limited. The impulse was somewhat diffuse, stronger at first, then growing feeble, and the cardiac dulness extended to the left vertical mammary line. The second sounds were not accentuated, and there were no associated murmurs.

Although no necropsy was permitted after death, the case was regarded as one of probable tuberculous endocarditis after the exclusion of other causes to explain the condition.

Fenwick, Traube, and many others have made clinical reports of instances of tuberculous endocarditis; but I have found that they lack definiteness as well as all bacteriological evidence. It would appear from my studies that almost all of the rare instances of true tuberculosis of the endocardium are associated with other forms of cardiac tuberculosis, especially the pericarditic. In this connection I may mention the twenty-eight observations of tuberculosis of the myocardium (cited by Teissier) in which no lesions of any portion of the endocardium were found.

But though tuberculous endocarditis is so extremely rare as to be almost non-existent, and hence of slight practical import, tuberculous sclerotic endocarditis is not uncommon, as demonstrated by pathological investigation. Lebert has noted three cases of the sort. It is extremely difficult to eliminate the influence of antecedent rheumatism and other infections, as well as that of gout, syphilis, chronic rheumatism, occupation, and other recognized causes of sclerotic endocarditis. This can, however, be done, as a rule, in the cases occurring in young subjects; and Teissier found that sclerosis occurs in 40 per cent. of the instances, even after excluding other etiological factors, as rheumatism, etc. This sclerosis of the endocardium is probably ascribable to an intense and oftentimes protracted intoxication of the economy. Neither tubercle bacilli nor tuberculous changes have been detected in these thickened areas on microscopical examination. Teissier also affirms that progressive narrowing of the mitral orifice is due to tuberculosis. Sansom,¹¹ while not in entire accord with Teissier, states that there is some relation between mitral stenosis and pulmonary tuberculosis. Drs. Harris and Beale¹² met three cases of mitral stenosis in a series of 133 post-mortem examinations on cases of tuberculosis. On the other hand, Walsham remarks, "I suppose all who have made many post-mortem examinations on cases of pulmonary tuberculosis will admit that the endocardium is frequently found thickened in places, especially in the left ventricle in the neighborhood of the aortic segment of the mitral valve, but never to the extent of producing a narrowing of the mitral orifice." He concludes: "I think that mitral stenosis with pulmonary tuberculosis is so rare a condition that the one cannot be regarded as the cause of the other, although not antagonistic to each other."

Percy Kidd, in speaking of his cases of combined heart disease and phthisis, previously cited, observes: "It is uncertain whether endocarditis or phthisis was established first in more than half the total number of cases, though in the majority of these it is fair to assume that tuberculosis was the primary disease." The occurrence of rheumatism was

noted in twelve of Kidd's cases, "and it is remarkable that the rheumatic cases almost exactly corresponded to those in which old endocarditis and dilatation or hypertrophy were found. In the non-rheumatic cases it is difficult to account for the development of granulations on the cardiac valves in the course of phthisis. Whether or not there may be any etiological connection between the tuberculous process and endocarditis must remain at present uncertain." He did not succeed in discovering tubercle bacilli or any tuberculous structure in the granulations in his cases. Out of a total of forty-two cases of so-called tuberculous endocarditis collected by Teissier, only twelve were supported by bacteriological evidence. It has been questioned by some authors whether the detection of tubercle bacilli in the valvular vegetations warrants us in pronouncing positively in favor of tuberculous endocarditis, since the granulations may simply afford a resting-place for the organism.

Leyden,¹⁸ in writing on the subject of tuberculous endocarditis, alludes to four cases of acute endocarditis in his own experience in which tubercle bacilli were discovered in the vegetations. In his opinion the acute endocarditis was not due to the tubercle bacillus, their presence on the segments notwithstanding. In one of my cases the typical physical signs of both mitral stenosis and insufficiency were associated with the unequivocal signs and symptoms of pulmonary tuberculosis, including the presence of tubercle bacilli in the sputum, from the date of admission to the Medico-Chirurgical Hospital. The patient was a male, aged twenty-seven years, and gave no history of antecedent rheumatism. Such cases, and they are not very uncommon, cannot be reliably classified. To say that the chronic endocarditis was secondary to the lung infection and due to the same cause, would be a purely prophetic utterance.

2. *Cases of recent endocarditis and chronic endocarditis that are secondary to tuberculosis or merely intercurrent, and are caused by various non-tuberculous infections and other agencies.* That such cases do occur, due to invasion by the streptococcus, staphylococcus, etc., is beyond peradventure, and most of the instances that are ascribed to tuberculous infection have in reality a different etiology. As stated elsewhere, not less than one-half of the cases of chronic valvular disease are caused by rheumatism, and more than one-half of the total number occur between twenty and thirty years of age, a period of life during which pulmonary tuberculosis is also quite frequent. In the vast majority of the cases, then, chronic endocarditis originates in primary acute rheumatic endocarditis, which almost invariably terminates in the former affection. Acute rheumatism, however, rarely complicates chronic phthisis, according to my observation. Indeed, it may be affirmed that both the gouty and the rheumatic diathesis are largely exempt from pulmonary tubercu-

losis. In Pollock's¹⁴ experience, "no history of rheumatism is obtainable in a considerable proportion of the cases." Percy Kidd, however, noted rheumatism in twelve of his twenty-seven cases of endocarditis associated with pulmonary tuberculosis.

Other causes, such as syphilis, malaria, alcohol, lead, and even occupation may rarely produce intercurrent chronic endocarditis in the course of pulmonary tuberculosis. My notes probably furnish a case in point:

A young man, colored, aged thirty years, with atheromatous vessels due to acquired syphilis, contracted tuberculosis for which he applied to the out-patient department of the Episcopal Hospital of Philadelphia. Six months later he presented himself a second time with the signs of aortic regurgitation, with moderate left ventricle hypertrophy. The case progressed from bad to worse, with respect both to the tuberculosis and the valve complaint, and finally his visits ceased and the case was lost sight of.

Although developing after the onset of the pulmonary condition, the aortic regurgitation was, in view of the vascular changes and the previous history, believed to have been occasioned by the syphilitic rather than by the tubercular infection. Both acute and chronic endocarditis may be secondary in the course of pulmonary tuberculosis.

3. *Forms of chronic valvulitis that are primary and precede the tuberculous infection of the lung, and are ascribable to rheumatism and other recognized causes.* This is the most important group of cases to be considered. This class has manifestly a non-tuberculous etiology, and it is believed that the cases exert in the main a contrary effect according as they affect the right or the left side of the heart. Hence, this division of the subject falls naturally under two heads: 1, The influence of left-sided heart affections (mitral stenosis, mitral regurgitation, aortic stenosis, aortic regurgitation) upon the development and clinical course of pulmonary tuberculosis; and 2, the influence of right-sided chronic valvulitis, more especially stenosis of the pulmonary ostium upon the etiology and clinical course of chronic phthisis.

The subject has occasionally engaged the attention of clinicians from the time of Laennec down to the present, and for a detailed statement of the views of the older writers I must refer my auditors to the excellent article of J. E. Graham,¹⁵ of Toronto, read before the Canadian Medical Association at Montreal, 1896.

Rokitansky's law, that "Persons laboring under enlargement (dilatation, hypertrophy, and their complications) of the heart, whether primary or superinduced by mechanical obstruction at its orifices, do not contract tuberculosis," is untenable. Such noted authorities as Niemeyer, Traube, Ruehle, and Pollock, while not holding to an absolute antagonism, yet with one accord maintain by their writings that a

decided protection is offered against pulmonary tuberculosis by the occurrence of valvular heart disease.

Graham collected some statistics bearing upon the effects of valvular heart disease on the existence of pulmonary tuberculosis previous to the date of publication of his paper, 1896, and I have tabulated these below (see Table III.). Unfortunately, the figures may include cases in which it cannot be positively stated that the cardiac affection was primary and the tuberculosis secondary.

By this table the broad fact that pulmonary tuberculosis occurs less commonly secondary to valvular heart disease than as a primary disease under ordinary circumstances receives some support. But, as previously stated, this counteracting influence is less potent than formerly supposed, and, as shown in Table IV., is a somewhat variable quantity depending upon the particular orifice implicated. The subjoined Table III. sets forth the influence of valvular affections as a whole upon the development and course of pulmonary tuberculosis:

TABLE III.

| Name. | Number of cases of organic valvular disease. | Ante-mortem. | Post-mortem. | Number of cases of tuberculosis. | Percentage of cases of tuberculosis. |
|-----------------------------------|--|--------------|--------------|----------------------------------|--------------------------------------|
| Bernard Schultz ¹⁶ | 484 | | 484 | 22 | 4.5 |
| Alfred Eymann ¹⁷ | 4138 | 4138 | | 69 | 1.7 |
| Frommolt ¹⁸ | 276 | | 276 | 21 | 8 |
| Germain (see Hanot) ¹⁹ | 277 | 277 | | 22 | 8 |

In the majority of the cases in which the special lesions were given, combined forms were observed, hence it was not possible to collect a sufficient number of single lesions to establish the relative frequency of the association of pulmonary tuberculosis with the separate recognized valvular affections. On comparing the percentages given above with the well-known ratio of cases of pulmonary tuberculosis to the general population, one is not struck by the extreme rarity of this disease secondary to simple valvular heart lesions. If the cases that were autopsied (760) are considered alone, the percentage of instances complicated with phthisis rises to 16.1, while the clinical cases, 4405 in number, give a percentage of only 1.8. To account for these contrasted results in the ante-mortem and post-mortem findings, one is forced to conclude that in most cases of valvular disease presenting pulmonary tuberculosis as a complication, the latter goes unrecognized during life. Moreover, the tuberculosis is often latent.

Bollinger's²⁰ series of necropsies on subjects under fifteen years of age show that among 218 cases of pulmonary tuberculosis 68 were of this character. The association of phthisis with primary valvular

heart disease is probably more common than the usual clinical findings have thus far indicated.

In answer to a series of questions published in sundry periodicals as to the association of valve lesions with pulmonary tuberculosis, a few responses have been received, and these coupled with my own personal records are here tabulated :

TABLE IV.

| Name of reporter. | No. of cases of pulmonary tuberculosis. | Valve lesions. | Per cent. | Particular valve lesions. |
|---|---|----------------|-----------|---|
| Dr. L. B. Milliken . . . | 60 | 3 | 5 | { 1 mitral stenosis. |
| Dr. E. W. Huhner.. | 36 | 1 | 2 2/3 | { 2 mitral regurgitation. |
| | | | | { Cardiac hypertrophy. |
| Dr. A. Abrams . . . | 800 | 21 | 2 2/3 | { 1 pulmonary stenosis. |
| | | | | { 2 aortic stenosis. |
| | | | | { 3 mitral stenosis. |
| | | | | { 9 mitral regurgitation. |
| | | | | { 3 mitral stenosis and regurgitation combined. |
| | | | | { 3 unclassified. |
| Dr. J. M. Anders . . . | 94 | 6 | 6 2/3 | { 4 mitral regurgitation. |
| | | | | { 2 mitral stenosis. |
| Dr. E. Baldwin . . . | 1,200 | 9 | 3/4 | { 7 mitral regurgitation. |
| | | | | { 1 aortic regurgitation. |
| Dr. A. Eyman ²² . . . | 27,653 | 80 | 0.288 | { 1 mitral stenosis. |
| | | | | { Does not give individual lesions. |
| Dr. H. Walsham ²³ . . . | 1,000 | 1 | 0.001 | { 1 mitral stenosis. As to other lesions nothing is stated. |
| Total exclusive of Eyman's, Baldwin's and Walsham's cases | 990 | 81 | 8.13 | |

These figures indicate that the observations upon cases of phthisis give a comparatively low percentage of instances in which this disease occurs in primary valvular lesions. I confidently believe that a different result would have been obtained by those observers who report the fewest cases of primary valvular heart disease if their attention had previously been especially directed to the association under discussion. This is confirmed by my own results as well as those of Dr. Milliken, who found three cases among sixty patients at Loomis Sanitarium, Liberty, New York, at one and the same time, after his attention had been drawn to the subject by my inquiries.

A clinical diagnosis of valvular heart disease must be made with due reserve and caution, there being murmurs at various orifices and apparent hypertrophies met in pulmonary tuberculosis that are not dependent upon valve lesions; but to enter upon a discussion of their nature and discrimination from the latter here would lead me too far. Oppositely, I have observed two cases of mitral stenosis in which slight consolidation of the lung above the heart existed, but the usual crucial tests did not reveal pulmonary tuberculosis. The diagnosis of cases of combined cardiac disease and tuberculosis of the lungs demands either the presence

of tubercle bacilli in the sputum or a positive tuberculin test in connection with clear evidence of valvular heart disease. Sansom has called attention to the fact that many cases of mitral stenosis show signs which can be easily mistaken for those of pulmonary tuberculosis.

The question naturally arises here, What is the explanation of the immunity offered by the lesions of the valves in the left heart against pulmonary tuberculosis? The responses that I have received from leading members of the medical profession in answer to this question, with few exceptions, offered the opinion that the preventive effect is ascribable in the main to pulmonary congestion. Albert Abrams, however, ascribes the antagonism to cardiac hypertrophy. Brehmer first ably championed this theory: he stoutly maintained that the disposition to and the cause of phthisis was a small, weak heart. Commenting upon a series of eighteen patients in whom valvular disease and pulmonary tuberculosis co-existed, Abrams pertinently remarks: "These cases thrived admirably as long as compensation was maintained, but when failure of compensation supervened the phthisical signs were fulminating. The pulmonary symptoms seemed to be held in abeyance during the continuance of compensation."

Says Graham: "It seems probable that the immunity from pulmonary tuberculosis existing in mitral disease is due to the following conditions: 1, The increased amount of blood in the lungs; 2, the greater expansion of the apices; 3, the increase of the involuntary muscular fibres of the bronchi." Graham affirms that among the changes observed in brown induration, the increase of connective tissue and the deposit of pigmentary matter are of no value in preventing tuberculosis. Concerning the fibroid induration, it is but reasonable to suppose that it plays a rôle of prime importance, seeing that it is by the formation of new connective tissue that healed tuberculosis is made possible.

There ensues more or less enlargement of the lungs in consequence of passive congestion in mitral disease, but it is an error to assume that the expansive power of the lungs is noticeably augmented, as can be readily shown by the use of the tape in cases of diseases of the mitral segments. Opposed to the view that pulmonary congestion *per se* is the all-important factor in preventing the development of pulmonary tuberculosis is the fact that hæmoptysis which diminishes the hyperæmia often affords considerable relief from symptoms and even arrests temporarily the march of the disease.

Again, respecting the rôle played by vensosity of the blood, on which some writers, including Rokitsansky, have laid stress as a factor in the supposed antagonism existing between phthisis and heart disease, it is to be particularly remarked that this condition is a conspicuous feature in congenital pulmonary stenosis which is more commonly associated with phthisis than mitral disease (*vide infra*).

It must be conceded that passive congestion in the lesser circulation or the local anatomical changes it produces (brown induration) or both seemingly account for the comparative immunity enjoyed from phthisis by those who have primary chronic valvular disease; this probably implies an increased supply of nutritious material to the lungs rather than that the tubercular virus is opposed directly by the venosity of the blood. It is a question of lung-nutrition.

Left-sided valvular heart disease exerts an influence upon the symptomatology of phthisis. In reviewing the history of cases, as well as from personal experience, I have been struck with the similarity between the general course of some of the cases that are combined with heart lesions and uncomplicated pulmonary tuberculosis. Some of the leading symptoms, however, show noticeable variations. Thus, from an examination of the notes of the cases gleaned from the literature (including also six of my own cases) a prominent initial symptom is not uncommonly hæmoptysis. Pollock observes that in the chronic phthisis associated with chronic valvulitis, hæmoptysis is more frequent and more severe than under other circumstances, and Balfour²² also directs forcible attention to the same fact. I have records of six cases (see Table IV.), and in two of these hæmoptysis was the initial objective symptom for which relief was sought by the patients, and yet in both a physical examination revealed lung-involvement and the sputum-test the presence of tubercle bacilli directly after its occurrence.

The practical lesson to be learned from this bit of experience is that tuberculosis secondary to chronic valve lesions may remain latent for a longer or a shorter period, and is then apt to be announced by the supervention of blood-spitting. Once started, the hæmoptysis showed a greater tendency to recur in my cases than in non-cardiac cases, and in one instance the bleedings were so profuse, and that at a comparatively early period as to cause an almost fatal degree of shock. A change of air to the Adirondack region and the free use of digitalis produced a cure so far as the phthisis was concerned.

When not so copious as to be immediately serious, these hemorrhages have seemed in some instances to have been followed by an amelioration of the cough and expectoration and less commonly by an improved appetite and general condition.

The dyspnea is more pronounced in cases of phthisis combined with valvular heart disease than in uncomplicated phthisis, and is of the cardiac type. Again, the temperature pursued a lower range in my combined cases than in phthisis occurring independently of cardiac disease.

There is perfect agreement among writers that valvular heart disease has a retarding influence upon the progress of chronic phthisis. According to my experience and an examination of the literature, however,

this is true in a more limited sense than is usually supposed. While the development of phthisis is delayed in many cases combined with heart disease, it must be recollected that "when the disease attacks an individual after middle life, or when from bad habits or bad hygienic surroundings there is a low state of vitality the process is often rapid" (Graham). Excessive and frequent bleedings may prove an element of danger, and they may shorten the duration of a certain number of the cases. Moreover, the lung condition tends to aggravate the cardiac affection, and the error too often committed, I find, of sending these patients to resorts that have an elevation of more than 2000 feet produces an unhappy effect upon the cardiac lesions, and may undoubtedly hurry a fatal termination. In briefly summing up the effect of the heart lesions upon the course of the phthisis it may be safely assumed that in the main there is a distinct retarding effect manifested under judicious handling of the cases. The major treatment, however, has reference to the cardiac element no less than to the phthisis.

Finally, it is only in cases in which valve lesions and the compensatory hypertrophy are proportional that a prognosis for unusual length of days can be safely ventured. When the harmonious balance is disturbed in the slightest degree an early fatal termination may be expected principally from the cardiac complaint. I append here brief notes of my own cases:

CASE I.—C. C. R., aged seventeen years, at school, called first on March 4, 1899. One brother had had hæmoptysis. Patient had been complaining for a year of extreme nervousness and nocturnal emissions; also cough which for a long time had been unproductive, but later attended with expectoration containing tubercle bacilli.

Physical signs referable to lungs: weak vesicular murmur at the left apex with a few subcrepitant râles over bronchi and slight impairment of the percussion resonance. Heart: moderate hypertrophy of both ventricles, apical systolic murmur and accentuation of pulmonary artery second sound. August 15, 1901, patient apparently cured of the phthisis as the result of change of residence from the city to the country, forced feeding, and out-of-door life.

CASE II.—J. A. C., aged twenty-eight years, occupation glass decorator, temperate habits, brought to me by Dr. O'Reilly. The entire family, save father and one brother, died of phthisis. No memory of previous rheumatism, but subject to frequent "colds." His illness started as a heavy "cold" in April, 1900; there were cough and expectoration, the latter becoming abundant, and later containing tubercle bacilli.

Physical Signs. Both apices gave the signs of decided infiltration, while the heart presented those of mitral regurgitation. This patient removed to Denver, where, as I learn from Dr. O'Reilly, he died within one year.

CASE III.—A. L., aged eighteen years, male, at school. Family history negative. About three days prior to his first visit spat one-half pint of blood; this was followed by cough and a thick, yellowish expectoration. History of frequent attacks of hæmoptysis in the mean-

while. No sputum test was made by me, although from the well-marked physical signs referable to the left apex, no doubt would be entertained as to the correctness of the diagnosis of pulmonary tuberculosis. The heart presented the typical physical signs of mitral stenosis combined with regurgitation. The final outcome in this case could not be learned, but at the time of patient's second visit, about six months after his first, it was noted that the pulmonary lesions had made little progress, and the hæmoptysis, the patient declared, was followed by marked, though temporary relief from cough and expectoration.

CASE IV.—C. A., aged thirty-seven years, married, female, first fell under my care November 16, 1900.

Family History. Maternal relations all died of phthisis, otherwise negative.

Previous History. Childish diseases; at ten years of age had articular rheumatism; and six years ago had second attack, lasting six weeks. Malaria at twenty-five years of age for three months or more. Her present illness began January 1, 1900, with slight cough and expectoration; three months later gave birth to a child, after which the cough grew slightly worse. In July (1900) had hæmoptysis; five weeks later had another seizure, losing about a pint of blood. One month later hemorrhages recurred at frequent intervals until her life was despaired of. Since then has had lighter ones. Has not lost much weight; occasional night-sweats. The physical signs at first visit: Lungs: Evidence of slight infiltration of left apex region, clear and distinct. Heart presented the signs of typical mitral regurgitation. Tubercle bacilli have been repeatedly found in the sputum. The diagnosis of combined mitral regurgitation and phthisis was readily made in view of the history of previous rheumatism, slight extent of the pulmonary lesions on the one hand, and the well-marked signs of mitral incompetency (including decided ventricular hypertrophy) on the other. Doubtless the heart changes were primary. The course of the lung complaint since the occurrence of childbirth has been found much the same as in the absence of associated heart lesions, compensation remaining apparently good notwithstanding.

CASE V.—M. B., aged twenty-five years, married, occupation sewing machine operator; first came under observation September 3, 1898. No traces of any hereditary taint in the family, and his own health uniformly good until April, 1898, when the illness for which he consulted me began. He first developed a severe "cold," which improved from time to time, but never entirely disappeared. Three months previous to his first visit he expectorated blood-stained sputum. August 28th, had a recurrence of the hæmoptysis which lasted for three days. Since then has had a cough and slight expectoration, with occasional night-sweats, but thinks his bodily weight is maintained. The physical examination showed a paralytic thorax and the signs of commencing consolidation at the upper portion of the left lung. The heart presented the usual physical signs of mitral incompetency; the murmur, however, was more marked after muscular exercise. Compensation has been good. The progress of the phthisis was exceedingly slow and finally, perhaps, arrested. I have not been able to learn anything concerning his condition for the past year and a half.

CASE VI.—J. C. O., aged twenty-four years, single, occupation farmer, was brought to me for examination by Drs. Robin and Dugler,

on May 9, 1899. The family history was negative. The patient had had the usual child's diseases, and in 1889 contracted the grip, with which he was afterward frequently afflicted. In September, 1896, had developed a cough, which remained until the date of his first visit; this was accompanied by free mucopurulent expectoration. There had been a falling off in weight of not less than fifty pounds. The physical signs of a moderate-sized cavity at the left apex were present, combined with those of mitral stenosis. The course in this case had been quite protracted until the signs of failing compensation developed, after which a fatal termination amid the signs and symptoms of advanced cardiac disease was not long delayed.

I have not observed a single case of aortic valve disease associated with phthisis since my attention was especially directed to the subject of this paper, some five years since, and it will have been observed that Table IV. furnishes a single case of both aortic stenosis and aortic regurgitation out of a total of forty cases in which valvular heart disease and pulmonary tuberculosis existed together in the same individual. Traube and others state that they have often met with aortic disease in cases of advanced tuberculosis, but never or only exceptionally with mitral disease. Krygen's ten cases of valvular heart lesions met in 1100 sections in cases of tuberculosis furnished four instances of aortic incompetency, three of mitral incompetency, and two of pulmonary stenosis. For this discrepancy of opinion and experience of different observers there is no satisfactory explanation at present writing.

4. *The effect of right-sided valvular heart disease upon the development and progress of pulmonary tuberculosis.* A careful review of the literature of this aspect of my theme seems to indicate that stenosis of the pulmonary artery predisposes to pulmonary tuberculosis. Stölker²⁴ noted sixteen instances of congenital stenosis of the pulmonary ostium. He calculates that in the cases of pulmonary stenosis that live to the age of twenty, 14 per cent. succumb to pulmonary tuberculosis. Lebert's²⁵ estimate, however, is much higher, or 33 per cent. of all cases surviving the twentieth year. This observer²⁶ also gives the literature of twenty-four cases of stenosis of the conus arteriosus of the pulmonary ostium and of the pulmonary artery, all of which (fifteen males and nine females) were complicated with phthisis. Bernard Schultz²⁷ found six cases of stenosis, five of which revealed complicating pulmonary tuberculosis. Constantin Paul²⁸ has examined, post-mortem, thirty-three cases of congenital stenosis of the pulmonary artery, and has formed the conclusion that phthisis commonly develops as the result. Heftler²⁹ believes that congenital heart affections, particularly pulmonary stenosis, favor the coincidence of morbus cordis and pulmonary tuberculosis.

While congenital stenosis of the pulmonary artery is often followed by tuberculosis, it must be recollected that, independently of the latter disease, life is prolonged beyond the second decade only under the most

careful arrangement of all the sanitary details. The dangers that beset such subjects are numerous and often disastrous in their results. The conditions are not favorable to healthy nutritive processes, and the lungs must share disproportionately, owing to the insufficient blood-supply, in the general nutritive disturbance, and thus become unduly susceptible to tubercular invasion. Obviously, the lungs must remain more or less undersized and ill-nourished from the date of birth—a condition that explains in a satisfactory manner the increased tendency to tuberculous infection. The extreme dyspnoea and cough from which these unfortunates at last suffer upon attempting muscular exercise compels them to keep quiet, and, in consequence of this interference with ordinary movements, the strength declines and the general health becomes greatly impaired. Neither is it judicious in the treatment of these cases to advise any but the slowest and most cautious muscular exercise. The foregoing facts explain the rather remarkable tendency of the subjects of pulmonary stenosis to contract phthisis, but its influence as an indirect cause of the latter disease, in view of the available testimony at hand, has probably been somewhat overrated.

We are not concerned here especially with the question of the diagnosis of cases of pulmonary stenosis, but I desire to draw attention to the fact that a myocarditis of the conus of the ventricle, as first pointed out by Dittrich, may also induce narrowing of the pulmonary orifice; hence the diagnosis of valvular endocarditis cannot always be made with a feeling of absolute assurance.

Lebert²⁰ has collected five cases from the literature in which the pulmonary stenosis was located in the right conus arteriosus, and these cases manifest a similar tendency to consecutive pulmonary tuberculosis. On the other hand, it is interesting to note that in cases in which communication exists between the ventricles, without associated pulmonary stenosis, phthisis is rarely encountered.

The clinical differences between phthisis in the advanced stage and combined pulmonary stenosis and phthisis are not so obvious as at first sight appears. The dyspnoea, cyanosis, the right ventricle hypertrophy may be duplicated in pure phthisis. That considerable hypertrophy of the right chamber is generally present in pulmonary tuberculosis may be easily verified by fluoroscopical examination. Not to be overlooked in the discrimination of the hybrid from pure phthisis are the bellows murmur or sound, which is to be heard at the pulmonary artery valve, and is transmitted upward and to the left, and the previous history, which clearly points to the presence of the dyspnoea and cyanosis from the time of suckling.

I append here the literature of thirty-six cases (exclusive of the twenty-four given elsewhere by Lebert²¹) in which phthisis developed secondary to pulmonary stenosis.

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REPORT OF A CASE OF DEMENTIA PRÆCOX.

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THE following history is imperfect in many ways, but seemed to the writer to have sufficient points of interest to warrant publication. Dementia præcox is not well known in this country, and the present case shows very well the physical characteristics of the disease. The mental characteristics have not been noted as fully, partly because the writer was not sure of certain phases apparently shown by the patient and partly to avoid making this report too long.

Mrs. G. T. was admitted to the Sheppard and Enoch Pratt Hospital July 1, 1899. She was then thirty-one years of age, born in Norway and married, having three children, the youngest being eighteen months old. Family history is negative. There was no history of any serious illness excepting that after the birth of her last child she had chronic bronchitis, which lasted nearly all winter, but from which she recovered entirely.

The first mental change was noticed in the latter part of May, 1899, when she went to communion, and afterward she said that she had done wrong in attending the service. This seemed to worry her very much, and she accused herself as well as her husband of wrong-doing. She then began to neglect her household duties, to sit about and brood. She thought that people would kill her, burn her up, or cut her in pieces. She complained of the street noises and said she was frightened. She wanted her husband to stop the noises and said she could not endure them. Some days she was brighter than others, but there was little real change in her condition. A few days before admission she said that if she were left alone in the house she would burn it up. Her physical symptoms were as follows: Her periods had been regular, she had been sleepless, had lost flesh, although she had been eating fairly well, and had been constipated. On admission there was but little change in her symptoms until about a month after admission, the only change

being that her appetite began to fail. About August 1st she refused to answer questions and would merely shake her head. Early in August she began to cough, later had to be put to bed, to be fed and dressed, was very dull and stupid, would let saliva run out of her mouth, and seemed to have pain in her chest, though physical examination showed nothing. August 31st she was put on iodide of potash, 5 grains, t. i. d., and five days afterward seemed a little brighter, and lifted her head when spoken to. Later the iodide was increased to 10 grains, but was stopped after a time, as no improvement followed. Early in September she began to have an offensive discharge from the nose, apparently simply due to her not cleaning it.

In the middle of October she became rather resistive, began to pinch her mouth up tight (snouting), would not eat for a few days, and paid no attention to her friends when they came to see her. About this time the katatonic symptoms were first noticed. She would hold her arms for some time in any position in which they might be placed, but would gradually change the position until it became more comfortable. The symptoms did not change materially, and early in February of 1900 her condition was as follows: Patient antagonizes every attempted passive movement. The resistance includes the facial muscles, and when one attempts to separate the eyelids with the fingers there is great resistance, and if the lids are parted the eyeballs are found rolled upward. Slight percussion over facial nerves causes short, sharp contraction of orbicularis palpebrarum and well-marked twitching over whole area supplied by facial nerves. All muscles are in a state of slight but well-marked tonic contraction which relaxes on stimulation. Fingers and thumbs are tightly flexed. Flexion is not relaxed by irritation with point of needle on other parts of the body. Triceps reflex present, slightly subnormal, periosteal absent, knee jerks and masseter active. Plantar flexion of all toes relaxed, sometimes almost entirely, by tickling soles of feet, and when this is done she tries to draw her feet away. Facial innervation equal on both sides.

Cold baths were tried without any apparent benefit. Her hands, which were flexed constantly, became sore, and it was quite difficult to wash them and apply bandages, as she screamed with apparent pain when they were extended. When anyone talked to her she puckered and pursed out her lips. On February 14th it was noted that she laughed to herself, and also laughed when the nurses spoke to her.

February 22d. She had an attack in which she shook as though having a chill. The pulse was 144. During this attack her muscles were not contracted. In March she attacked a nurse, and then spoke for the first time in several months. Later in the day she again attacked a nurse and was very noisy. After this it was possible to have her up and dressed. She talked quite well at times, still had to be fed, and sat very quietly, rarely changing her position. In May she was well enough to be taken out walking, but generally had to be pushed forward while she took steps, which were about six inches long. At one time she jumped along like a toad. This condition continued, she having a few more excited attacks, and in the middle of October her condition was as follows: October 18, 1900. Patient sitting in chair, put to bed for examination, dorsal decubitus, with knees drawn up, eyes closed, and hands clenched. When addressed does not open eyes, and usually makes no sign of response, but sometimes laughs quietly. Resists any

attempt to open eyelids. Will not open mouth when asked. Does not object to a fly walking all over her face. When touched lightly with a camel's-hair brush about the eyes there is slight movement of the lids, but no attempt to move the brush with the hand and no change of expression. There is no reaction when the patient is touched about the corners of the mouth and no voluntary muscular movement, except at times a slight laugh. When alone the patient sometimes opens her eyes a little, but closes them again when spoken to or when anyone approaches her. At times cries out and has occasionally spoken the names of members

FIG. 1.



of her family, but is usually silent, not even mumbling. When tickled with a brush in the external ear there is slight contraction of the external ocular muscles and slight action of the zygomatici, invariably accompanied by a slight chuckle. Patient does not attend to bodily needs, soils herself if not watched. Is able to drink from a glass held in both hands without spilling the contents, and eats with a spoon held invariably in the right hand. Has slight muscular memory in distinguishing right from left. Strong pain stimuli do not seem to produce any reaction. Patient does not wince when the skin of the forearm is severely pinched. Auditory stimuli, on the contrary, are quickly

responded to. Head can be turned from side to side without resistance and the arms placed in different positions without opposition. The hands can be opened, but fingers cannot be fully extended. Thumb can be moved freely, but there is contracture of the interossei and lumbricales of both hands. When the terminal phalanges are forcibly extended there is a slight movement of the head which suggests the possibility that the forced movement causes the patient pain. There is a tendency for the arms, when placed in unnatural positions, to remain a considerable length of time as they are placed, and then they gradually drop. There is no tendency to grasp objects thrust into her palms. When tapped lightly on the face with percussion hammer there is marked movement of the eyelids, more so than when touched with the brush. Percussion of the regions supplied by the seventh nerve shows marked mechanical irritability of the muscles. No marked tenderness over skull on percussion. Eyeballs are apparently rotated upward, although it is almost impossible to separate the lids. Patient is not careful to be cleanly, and, when suffering from coryza, often allows mucus to appear and run down over her face.

Superficial circulation very poor. Passive movements of legs and feet are not resisted. Lies with toes turned in. Inner border of soles slightly elevated. When tickled on the sole of the foot there is no response. Knee-jerks exaggerated. When sole is scratched with a pin there is slight movement to withdraw the foot. Pin scratching causes plantar flexion of all the toes. Patient never dresses herself and has no initiative, but when very excited has sometimes taken off nearly all of her clothes. While sitting in bed scapulæ are rotated outward from mid-line, and inner border is raised from chest wall; both deltoids are very poorly developed, leaving prominences of shoulders plainly visible.

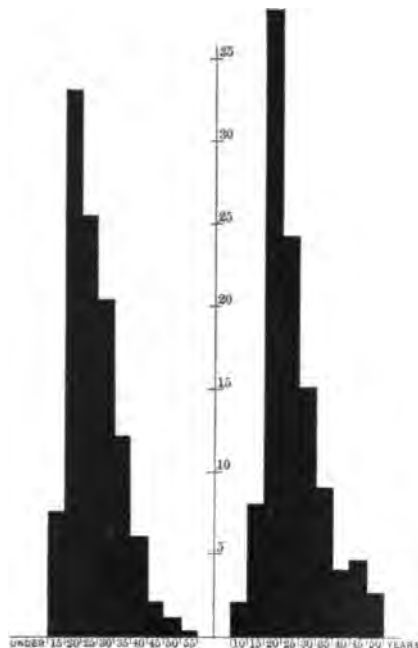
There is some atrophy of the supraspinous region. When uncovered patient begins to shiver as though cold, but when asked to nod her head if cold she replies by shaking her head. Pectoral muscles very poorly developed. Resisted having breast exposed.

With the galvanic current there is a short, sharp contraction of the right deltoid, left being somewhat sluggish. On stimulating the facial nerve there is a short, quick contraction with a current of three milliamperes. No increased mechanical irritability of muscles, and no marked response on Erb's point. With a faradic current of sufficient strength to give fairly strong contraction of muscles there is no evidence of sensation.

September 21, 1901. The patient was to-day discharged unimproved and taken to one of the State hospitals. Her condition had not changed since last note. She was in bed a great deal because she seemed more comfortable, was less apt to have sudden attacks of violence, and was more easily cared for. Menses were regular throughout her stay in the hospital. She weighed ninety pounds when discharged, a loss of thirty-eight and one-half pounds since admission. In the past year she had lost but three and one-half pounds. After being made ready for discharge the patient would not walk nor stand up, but after a few minutes walked when supported and pushed forward by two nurses, and later stood without support. She answered questions with nods and shakes of her head, but would not speak. Her hands were blue and fingers were immovably semiflexed.

Kraepelin gives the following symptoms as common to all forms: The tendon reflexes are increased; the pupils are dilated and may be different in size; saliva is increased; the activity of the heart is weakened; cyanosis and hyperidrosis are often present; the menses are skipped or irregular; sleep is disturbed; the capacity of taking nourishment varies, but the weight decreases.

FIG. 2.



Relative frequency of dementia præcox.

Relative frequency of the manic depressive insanities (mania, melancholia, stupor).

(After KRAEPELIN.)

The simple appreciative perception of external conditions is not interfered with, therefore orientation is undisturbed, but may be dimmed in stupor or influenced with insane ideas when excited.

Sensory perceptions are, however, frequently interfered with, and the order in which they are generally affected is: first, hearing; second, seeing; third, feeling. In the beginning the fallacious sense-perceptions are generally disagreeable, but later may be indifferently or pleasantly interpreted. For example, the patient often thinks he is in a theatre, and may ask questions about the performance. These questions are, of course, foolish or incoherent.

Fixation is poor. After stupor great curiosity may be shown. Negativism is frequently present. (Negativismus, or negativism, Kraepelin

defines as the senseless struggling against every external influence. It is shown in the mutism or the senseless dumbness, as well as in the complete inability to influence the patient.)

Time-sense is well marked. The power of retaining words, numbers, etc., is generally well preserved, but the power of putting together new words, or of using new words, is diminished, and that the train of thought suffers is shown by stereotypie, rhyming, or verbigeration.

Judgment is always impaired and the sense of right is lost. The will power is lost. There is disturbance of the emotional life. The patient is sometimes sad, and sometimes lively or excited, but the excitement is simply a motor one. They may have sudden attacks of boisterousness. Ideas of beauty are lost. There is no technical ability, and the power to work suffers, the patient being able to do old things, but nothing new. Epileptiform attacks occur, and are more common in men, occurring in 18 per cent. of all cases. Besides this there may be hysterical attacks, apoplectiform attacks, or paralyzes, or chorea-like movements.

Tromner says that the four most characteristic abnormalities are: 1, a peculiar confusion in speech, writing, and demeanor; 2, the tendency to the production of bizarre anomalies in gait, gesture, mien, and action; 3, weakness of judgment; 4, termination in the peculiar dementia.

Christian makes the following summary of symptoms: 1, a constant appearance at puberty; 2, various delirious symptoms at the beginning; 3, constant sudden impulses; 4, a rapid termination in a dementia more or less complete.

The majority of authors agree that dementia præcox is a degenerative psychosis.

A summary of the case shows that the patient was mentally depressed. There was an exaggeration of the tendon reflexes, a weakening of the heart's action, cyanosis, and a decrease of weight while taking nourishment well. At one time she refused food and had to be fed, later she took nourishment well. Simple perception of external ideas was not interfered with, but there was fallacious sense perception, as was evidenced by the early complaints of street noises, etc. Negativism, while present, was not especially marked. There was disturbance of her emotional life, as was shown by her periods of depression and attacks of boisterousness. Stereotypie and verbigeration were shown on several occasions. Katatonic rigidity was also present.

In view of these symptoms the case seems to be the katatonic form of dementia præcox. It is to be regretted that her previous history should be so meagre, but there was a good deal of difficulty in obtaining the little which is given. The age, thirty-one years, at which onset is noted is somewhat uncommon, being beyond the period of puberty,

which Christian has placed between the ages of fifteen and twenty-five. Kraepelin, in his *Text-book of Psychiatry*, gives a diagram showing the percentage of cases in reference to the age of occurrence. More than 60 per cent. occur before the twenty-fifth year, but over 10 per cent. occur before thirty-five years. Tromner, of Hamburg, in his monograph on dementia præcox, has placed this diagram beside one which shows the occurrence of the maniacal-depressive forms (mania, melancholia, stupor) in the same periods of life. It shows very strikingly that dementia præcox is not so essentially a puberty psychosis as was supposed, and that the maniacal-depressive forms are more common in early life than many of us have thought.

The condition of tonic muscular contraction shown by this patient has not been much studied. It occurs without accompanying mental symptoms, but the correlation between the two is practically unknown. It is hoped that physicians may become interested in this condition, and by reporting cases, either with or without accompanying mental symptoms, add to our knowledge of the subject.

The literature on dementia præcox is large, but unfortunately most of it is by foreigners, so that it is not easily reached by all physicians. A good deal has been translated, however, and appended is a list of a small part of the literature.

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ON SARCOMA OF THE RADIX LINGUÆ,¹ WITH REPORT OF A CASE.

BY GEORGE E. SHAMBAUGH, M.D.,
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In September, 1900, the following case came for treatment to the dispensary at the Woman's Medical College:

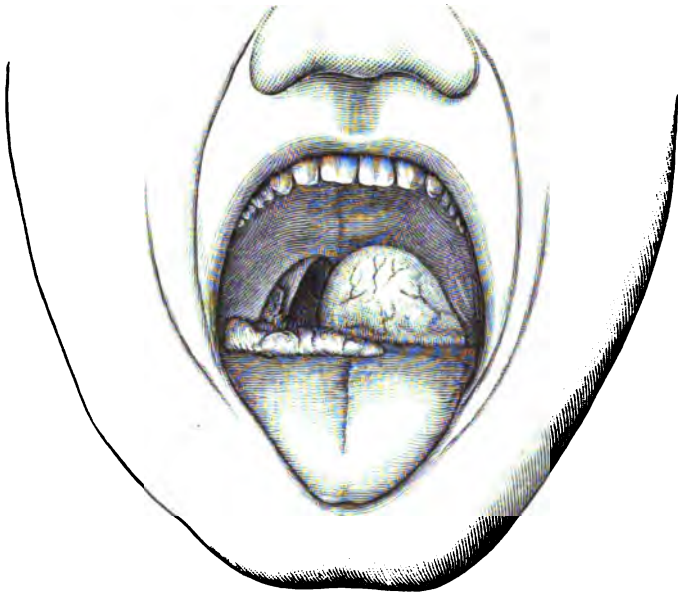
A. S., a laborer, aged thirty-eight years, was born and raised in Poland. Father and mother died at the ages of fifty-seven and fifty-

¹ When the mouth is opened as wide as possible and the tongue protruded one may see by direct inspection, in a favorable case, several of the circumvallate papillae lying near the border of the tongue. These papillae are arranged in the form of a V, with the apex pointing toward the back and terminating at the foramen cæcum. The part of the tongue in front of

five, respectively; causes not known. Patient had been married sixteen years, and had had five children. One child died in infancy, and the other four are living and well. Previous to his present trouble he had always been well and strong. He was a strongly-built man, of medium height. When first seen, on September 8, 1900, he showed a well-marked cachexia. His face appeared somewhat shrunken, his muscles were flabby, and his skin sallow. His facial expression was strained and anxious, his chin was slightly elevated, allowing the throat in front to protrude, frog-like, as when the base of the tongue is strongly depressed.

His throat trouble began eight months previous, with slight pain on swallowing, in the region of the hyoid bone. With the exception of this trouble on swallowing, he had never suffered any pain, but had been troubled a great deal with mucus accumulating in his throat.

FIG. 1.



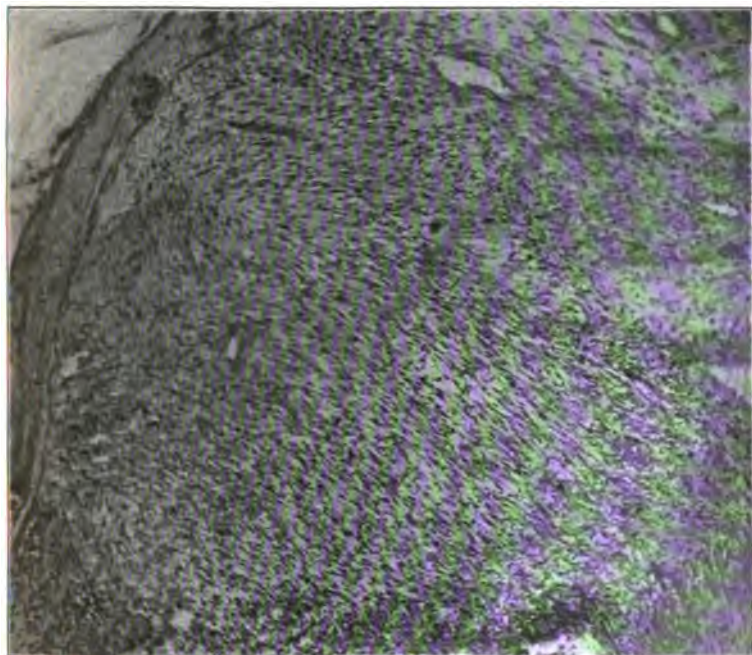
This he had great trouble in dislodging. He first experienced difficulty in swallowing food, two months previous. After a time he could swallow solid food only after he had pushed it past the swelling with his finger. Later he was unable to swallow solid food at all, and for the past two weeks he had had trouble in swallowing liquid food on account of the regurgitation which took place through the nose. His respira-

the circumvallate papillæ is developed (according to His) from the tuberculum impar, which springs from the median line of the floor of the pharynx between the first and second arches. The part of the tongue back of these papillæ results from the growing together of two lateral halves, which spring from the antero-lateral pharyngeal walls in the location of the second and third visceral arches. This posterior part of the tongue, having an origin quite distinct from the anterior part, is called the radix linguæ (Anatomische Nomenclature, Basll, 1895). The term "base of the tongue" has sometimes been applied to it also.

tion had been interfered with somewhat, and his voice was greatly altered by the mechanical obstruction in the oropharynx.

Examination of the neck disclosed no glandular enlargement. Inspection of the oral cavity showed the front part of the tongue normal. The posterior part of the oral cavity and the oropharynx were occupied by a tumor the size of a small hen's egg, lodged on the left side, hiding the pillars of the fauces and the soft palate on that side. (Fig. 1.) At times the soft palate was seen stretched over the rounded top of the tumor, which then projected into the postnasal space. This tumor had an oval outline, its surface was smooth and of a red flesh color, with dilated veins showing at several places. It felt soft, and bled readily when disturbed

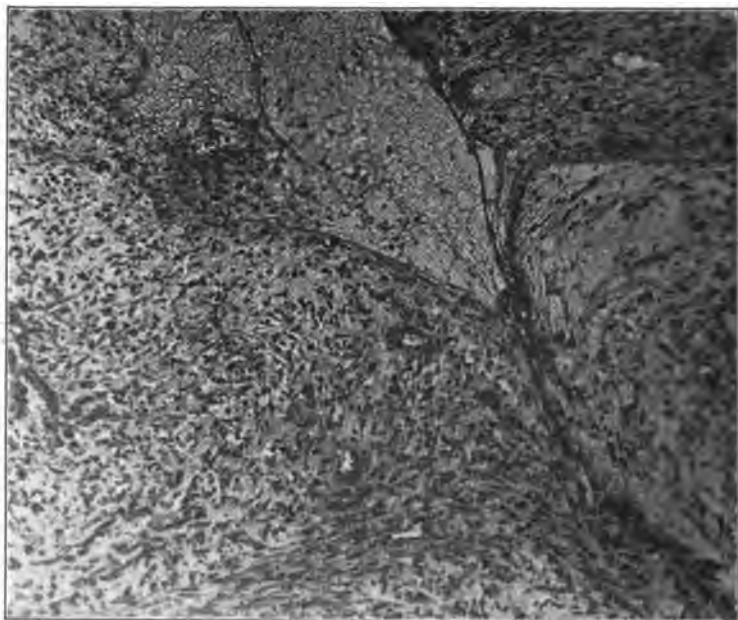
FIG. 2.

Showing spindle-cells and bloodvessels. $\times 60$.

with a probe. Its attachment appeared, on palpation, to be deep down on the radix linguæ. Occupying the middle and right thirds of the radix linguæ was a dense, firm, elastic mass, elevated about one-quarter of an inch above the surface of the tongue, extending as far forward as the circumvallate papillæ, and with its anterior edge somewhat curled over its attachment. Its nodular surface was covered with smooth mucous membrane. It did not bleed so readily as the tumor on the left. The presence of ulceration was not detected on any part of the growth.

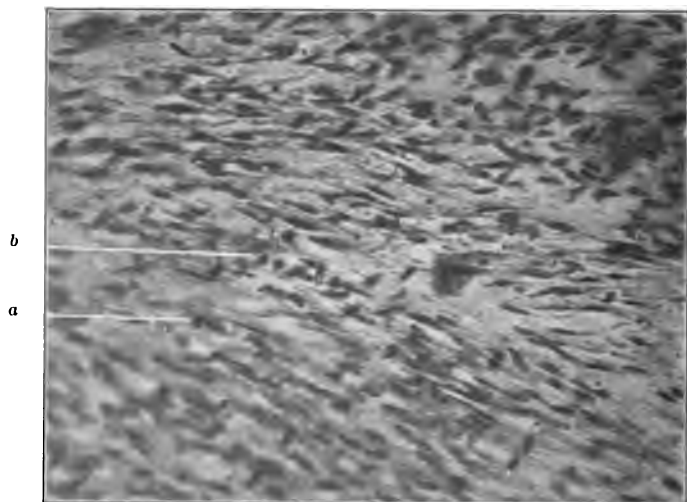
The patient was seen again on September 30th, when he said that a week previous he had suffered a severe hemorrhage from his throat,

FIG. 3.



Showing spindle-cells cut in cross section, bloodvessels, and extravasation of the blood into the tissue. $\times 90$.

FIG. 4.

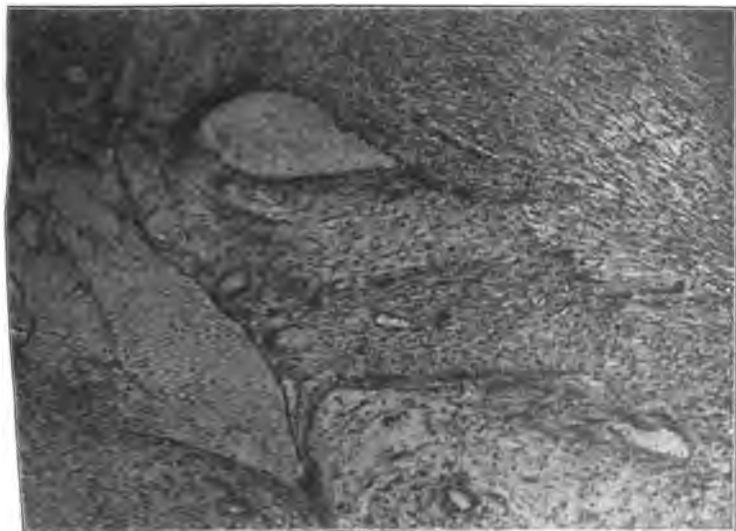


Showing large spindle-cells separated by intercellular substance; also karyokinetic figures (a, a, and b, b). $\times 200$.

when he had lost two quarts of blood. He had been very weak and dizzy and had tinnitus aurium following the hemorrhage. He appeared much paler than when seen before. The large tumor on the left was slightly shrivelled. It had partially lost its red flesh-color and was somewhat yellowish. At this time I removed from the mass on the right a small piece for microscopical examination.

This piece was made up of large spindle-cells, arranged in broad currents streaming in different directions through the tumor. (See Fig. 5.) No distinct epithelial covering could be made out, nor was there a distinct capsule. At one place only was there a structure that resembled a capsule. (See Fig. 2.) In several places large areas of necrosis were found. Scattered through the middle of the section were a number of large, open spaces filled with blood, evidently extravasa-

FIG. 5.



Showing streams of spindle-cells, numerous bloodvessels, and extravasation of blood. $\times 60$.

tion of blood into the tissue. (See Figs. 3 and 5.) The section was quite rich in bloodvessels. The cells were large, fusiform, or spindle-like, many having elongated thread-like ends. (See Fig. 4.) In many of the cells, karyokinetic changes could be seen. (See Fig. 4.) The cells, for the most part, were not packed closely together, but were separated by a considerable amount of fibrous intercellular substance.

The tumors which involve the radix linguæ are rare, if we exclude the not uncommon hypertrophy of the lingual tonsil. The benign tumors that have been found in this locality include fibroma, fibrolipoma, fibromyoma, chondroma, cysts, adenoma, accessory thyroids, dermoids, gumma, and cavernoma. Of the malignant tumors, primary carcinoma appears to be the most rare, while secondary carcinoma is

relatively not uncommon. Sarcoma of this region, while not so rare as primary carcinoma, is of exceedingly rare occurrence. I have been able to collect but nine cases from the literature on the subject. The first case was reported by Fiedler as early as 1864. No other case was reported until 1885, twenty-one years later, when Albert reported his case of round-celled sarcoma of the basis linguæ. The following year, 1886, Eve examined microscopically and described an old museum specimen of the Hunterian collection, which proved to be a round-celled sarcoma. The next year Beregszaszy reported a case of lymphosarcoma. In 1888 Schulten reported a case of small-celled sarcoma. In 1892 Scheier reported one of small round-cells, and in 1894 Perman reported one of round-cells. In 1895 Onodi published the case of a round-celled fibrosarcoma occurring in a girl, aged seventeen years. Michael, in 1895, reported the only case of secondary sarcoma of the radix linguæ that has been published.

In this list I have not included the following cases, where doubt exists as to the involvement of the radix linguæ.

A case reported by Gross,¹ in 1872, of spindle-celled sarcoma, occurring probably congenitally in a child, where the tumor is described as occupying the posterior half of the dorsum of the tongue, but it is doubtful whether the radix linguæ itself was involved.

A case reported by Butlin,² in 1887, where the tumor occupied the attachment of the tongue (root of the tongue—Butlin), causing a bulging both above and below this organ. He describes it as extending from the junction of the anterior and middle thirds to the back of the tongue, but is not clear as to the involvement of the radix linguæ.

Regarding the etiology of sarcoma of the radix linguæ, nothing definite can be formulated from the cases reported. In one case the irritation of a broken tooth and of excessive cigarette-smoking was given as a possible cause. In Scheier's case it looked as if the sarcoma might have developed in the base of a previously existing ulcer. Cases of sarcoma in other parts of the tongue, developing in the seat of an ulcer, have been reported by Dunham³ and by Littlewood.⁴ Heredity does not seem to play any part in its occurrence. In one case only was the sarcoma secondary. In this case the primary growth appeared in the tonsil. It occurs with equal frequency in either sex. Of the nine cases where the sex has been given, four were female and five were male. It appears to occur at any age. Of the nine cases where the age has been given, one occurred at the age of seventeen, and one at the age of fifty-six. All the other cases occurred between the ages of

¹ Philadelphia Medical Times, 1887, vol. ii. p. 272.

² Lancet, March 26, 1887, p. 632.

³ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, Philadelphia, 1895, n. s., vol. cx. p. 259.

⁴ Transactions Pathological Society, London, 1897-98, vol. xlix. p. 60.

twenty-eight and forty-two. In the case reported by Gross¹ the tumor appeared to be congenital. Marion,² who collected twenty-one cases of sarcoma of the tongue, in which the ages of the patients were given, found that twelve occurred below thirty years, three between thirty and forty, three between forty and sixty, and three over sixty.

Sarcomata of the radix linguæ have been found from the size of a hazelnut to that of a hen's egg. They may be distinctly encapsulated or appear as diffuse infiltration of the tissue from which they take their origin, or they may appear pedicled. The surface is usually smooth, but may appear nodular, and is usually covered with normal mucous membrane. In two cases only was ulceration detected. Its consistency varies from the firm elastic tumor of the fibrosarcoma to that of semi-fluctuation. The tumor may be of one piece, or it may be distinctly lobulated. In no case was there a definite history of the existence of more than one tumor. In the case reported by me, it looked as if we had to do with two distinct tumors.

Histologically, all of the cases where the type of cells was specified are cases of round-celled sarcoma, except Fiedler's case and the one which I report, which are cases of spindle-celled sarcoma.

The symptoms caused by sarcoma of the radix linguæ are very similar to those caused by any benign tumor in this locality, symptoms referable chiefly to the mechanical disturbance caused by the size of the growth. In the beginning, sarcoma often causes but an unpleasant sensation in the throat, associated with an increase in the amount of mucus secreted. As the tumor develops, pain and difficulty in swallowing solid food are noted. Later the voice is impaired, and respiration interfered with. Ulceration is not common. In only two of the cases was it noted. Glandular involvement is rare, as is the case with sarcoma in other localities. When it has been found, it was referable, as a rule, to local infection rather than to the extension of the sarcomatous growth to the glands. Metastasis to the peritoneum was noted in one case. Pain, which is such a prominent symptom in carcinoma, is not pronounced in sarcoma, except where ulceration exists. Hemorrhage is also rare, owing to the absence of ulceration. The course of the disease is usually quite rapid. In only three of the cases had the tumor existed more than one year after the first symptoms appeared, while in four of the cases it had lasted less than six months.

The diagnosis of sarcoma of the radix linguæ from the benign tumors which are found here is not, as a rule, difficult. The wide insertion of the sarcoma and its rapid growth marks it as in all probability malignant. Its color more nearly resembles that of the surrounding tissue than is the case with benign growths. When the sarcoma is large its

¹ Loc. cit.

² Rev. de Chir., Paris, 1897, vol. xvii.

growth is such as to alter the contour of the surrounding parts, while a benign tumor is more apt to conform to its surroundings and to cease growing when it meets an obstruction. From carcinoma the diagnosis is not always easy to make. When we have to deal with the non-ulcerating variety of sarcoma this clinical fact alone is strong evidence that the tumor is not carcinoma, for the latter almost always breaks down quite early. With the ulcerating variety, on the other hand, the question is more difficult. The absence of marked pain and glandular enlargement would distinguish the sarcoma from carcinoma where both of these symptoms are usually marked. It should be noted here that with an ulcerating sarcoma pain is much more apt to be present than in the non-ulcerating variety, and that in this type of sarcoma, glandular enlargement from infection is quite apt to be present. Clinically the diagnosis cannot always be made between the two types of malignant tumors. In Beregzaszy's case a diagnosis of cancer was made until a post-mortem examination showed the case to be sarcoma. In these cases of ulceration the histological diagnosis is not always easy to make, as we may repeatedly succeed in getting only a piece of the granulating tissue about the sarcoma for examination, as happened with Scheier before he finally was fortunate enough to get a piece of the sarcoma.

Syphilis may readily be mistaken for sarcoma, especially in the pre-ulcerative stage. Esmarch¹ expressed his belief that most of the cases diagnosed as sarcoma of the tongue were cases of syphilis, and would clear up under the proper treatment.

After ulceration has occurred the diagnosis may still be difficult, for sarcoma may also ulcerate, and it is right here again where the histological examination is likely to fail one. For these reasons, where any doubt exists as to the nature of the growth the diagnosis should be reserved until the case had been subjected to vigorous anti-syphilitic treatment. It should be remembered that sarcoma, too, may show a temporary improvement under the administration of potassium iodide.

The prognosis is not so unfavorable as one might at first suppose. Of the nine cases where the clinical history is given, four received no treatment. Two of these were lost sight of, and the other two were considered inoperable cases, and died from exhaustion. Of the remaining five cases, in two, where the tumor was removed, recurrence and death took place; in two cases where the tongue was removed one died of pneumonia eight days later, the other got well, and three years later there had been no recurrence. The fifth case was cured by injections of pyoktanin and subsequent removal of the tumor with the galvanocautery snare. In the case reported by Butlin,² where the sarcoma

¹ Langenbeck's Archives, Bd. xxxix., Heft 2.

² Loc. cit.

occupied the left side of the posterior half of the tongue, the left half of the tongue was removed. The case recovered, and four years later there had been no recurrence.

| Surgeon. | Age | Sex | Duration. | Histology | Pain. | Ulceration. | Involvement of glands. | Treatment. | Result. |
|--------------------------|-----|-----|----------------|-------------------------------|-------|-------------|------------------------|---|--|
| Fiedler. ¹ | 40 | M. | Several years. | Soft spindle-celled sarcoma. | Yes. | | Yes. | Extirpation with galvano-cautery snare | Recurrence and death from exhaustion. |
| Albert. ² | 56 | F. | 3 yrs. | Round-celled sarcoma. | No. | No. | No. | Tongue extirpated. | Died of pneumonia eight days later. |
| Eve. ³ | ad. | ... | | Round-celled sarcoma. | | | | | |
| Beregnaszy. ⁴ | 42 | M. | 2 mos. | Lympho-sarcoma. | Yes. | Yes. | Yes. | No treatment | Developed metastasis in peritoneum eight days later. |
| Schulten. ⁵ | 32 | F. | ½ yr. | Small celled sarcoma. | No. | No. | No. | Extirpation of tumor; recurrence; tongue extirpated by supraglottic pharyngotomy. | Three years later no recurrence. |
| Scheier. ⁶ | 28 | M. | 2 yrs. | Small round-celled sarcoma. | Yes. | Yes. | Only after operation. | Tumor removed. | Recurrence and death. |
| Onodi. ⁷ | 17 | F. | 7 mos. | Round-celled fibro-sarcoma. | No. | No. | No. | No treatment | Patient lost sight of. |
| Perman. ⁸ | 30 | F. | 6 mos. | Sarcoma. | No. | No. | No. | Injections of pyoktanin and subsequent removal with snare. | One year after removal no recurrence. |
| Michael. ⁹ | 30 | M. | Few weeks. | Lympho-sarcoma. | ... | | Yes. | Inoperable. | Died of cachexia. |
| The author. | 88 | M. | 8 mos. | Large spindle-celled sarcoma. | No. | No. | No. | No treatment | Patient lost sight of. |

¹ Ztschr. f. med. Chir. u. Geburtsh., Leipzig, 1864, n. f. iii. pp. 305-312.

² Wien. med. Presse, 1885, p. 171.

³ Transactions Pathological Society of London, 1886, vol. xxxvii. p. 223.

⁴ Krankheiten der Zunge von Butlin. Wien, 1887, p. 226.

⁵ Pínska. Läkarsällskapets handlingar, 1888, p. 689. Cited in Deutsche Ztschr. f. Chir., Bd. xxxv. p. 417.

⁶ Berl. klin. Wochenschr., 1892, p. 584.

⁷ Monatschr. f. Ohrenh., 1895, vol. xxix. p. 75.

⁸ Hygiea, 1894, vol. i. p. 367; also, Buffalo Medical and Surgical Journal, 1894, p. 148.

⁹ Handbuch der Laryngol. u. Rhinol. Helmann. Wien, 1899, vol. ii. p. 634.

A REVIEW OF ECHINOCOCCUS DISEASE IN NORTH AMERICA.

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THE writer's intent in the present article is to present the statistics of hydatid (echinococcus) disease in the United States and Canada, based upon the cases and references collected from the literature and other sources to date—July 1, 1901—and to add thereto certain observations suggested by the facts shown, with the hope that such a study may contribute to the interest and knowledge of this rare disease in America, and make available for reference the cases that are scattered through the mass of medical literature.

Two previous reviews of hydatid disease in America have been published, the first by Osler,¹ in 1882, collecting sixty-one cases from the United States and Canada, and the second by Sommer,² in 1895 and 1896, bringing together 100 cases from the United States. Osler³ states also that Alfred Mann collected for him from the literature in the period—1882, to July, 1891—twenty-four in addition to his previous sixty-one cases. Mann's summary of cases was not published, but Dr. Osler has informed us that all of Mann's cases were later included in Sommer's collection. The statistics of Osler and Sommer together include 110 cases. To these cases we have been able to add 135 other cases, so that our statistics embrace in all 241⁴ cases occurring in the United States and Canada.

The statistics, in brief, of the 241 cases are given in Table I., as follows :

TABLE I.—SUMMARY OF CASES OF ECHINOCOCCUS DISEASE IN
NORTH AMERICA.

Case 1.—Low, 1822. N. Y. Med. and Phys. Journal, i. 287-289. Male, aged twenty-nine years, white, Albany, N. Y. Hydatids in bladder.

Case 2.—Low, 1822. N. Y. Med. and Phys. Journal, i. 287-289. Female, aged seventy-three years, white, Albany, N. Y. Hydatids in liver and ovary.

Case 3.—Alexander, 1838. Boston Medical and Surgical Journal, xviii. 37-42. Male, aged thirty-five years, foreigner, Danville, Vt. Cysts in liver (about 200).

Case 4.—Polk, 1841. West. Journ. Med. and Surg., Louisville, 2 S., iv. 34-35. Male, aged sixty-five years, white, Perryville, Ky. Liver.

¹ Read at the annual meeting of the New York State Medical Association, held at New York, October 21-24, 1901.

² Osler. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES. October, 1882, N. S., vol. lxxxiv. pp. 475-480.

³ Sommer. New York Medical Journal, November, 1895, vol. lxii. pp. 656-659, and August, 1896, vol. lxiv. pp. 262-265.

⁴ Osler. Practice of Medicine, New York, 1895, second edition, p. 1102.

⁵ We have omitted from our statistics four of Osler's and Sommer's cases, as they seemed of doubtful validity.

- Case 5.—Gross, 1845. *Path. Anatomy*, Phil., 2 Ed., 662. Male, aged thirty-five years, Cincinnati, O. Large cyst in liver; rupture into peritoneal cavity.
- Case 6.—Moses, 1846-47. *Annalist*, N. Y., i. 258-255. Male, adult, Governor's Island, N. Y. Hydatids in liver.
- Case 7.—Grainger, 1849-50. *Buffalo Med. Journal*, v. 80-82. Male, aged sixty years, Lawrenceville, N. Y. Hydatids in ventricles of brain.
- Case 8.—Weber, 1852. *N. Y. Med. Wochenchr*, April. Male, aged twenty-six years, German, New York City. Cyst in liver.
- Case 9.—Ellis, 1856. *Boston Med. and Surg. Journ.*, liv. 344, and *Extract Rec. Boston Soc. Med. Improv.*, ii. 332. Female, aged twenty-eight years, Italian, Boston, Mass. Liver.
- Case 10.—Ellis, 1856. *Extract. Rec. Boston Soc. Med. Imp.*, ii. 332. Male, aged twenty-four years, Irish, Boston, Mass. Liver.
- Case 11.—Evans, 1856. *West. Lancet*, Cincinnati, xvii. 680-694. Female, aged thirty-four years, Welsh, Newport, Ky. Liver.
- Case 12.—Finnel, 1856. *N. Y. Journ. of Med.*, N. S., i. 216, and *Trans. N. Y. Path. Soc.*, iii. Male, aged twenty-seven years, English, New York City. Cyst in liver; no hooklets.
- Case 13.—Gross, 1857. *Path. Anatomy*, Phil., 3d Ed., 609. Fifteen small cysts of liver.
- Case 14.—Smith, F. G., 1858. *N. Amer. Med.-Chir. Rev.* (*Proc. Path. Soc. Phil.*), ii. 333-334. Female, aged thirty-four years, Philadelphia, Pa. Liver; cysts expectorated; discharge of fluid by bowels.
- Case 15.—Park, 1858. *Nashville Journ. Med. and Surg.*, xv. 120-125. Female, aged seventy-three years, white, Franklin, Tenn. Hydatids in abdomen.
- Case 16.—Gay, 1858. *Boston Med. and Surg. Journ.*, lvii. 218-220. Male, aged seventy-one years, Boston, Mass. Echinococcus in lungs.
- Case 17.—Gay, 1858. *N. Amer. Med.-Chir. Rev.*, ii. 506. Female, Philadelphia, Pa. Echinococcus in lung.
- Case 18.—Minot, 1859. *Boston Med. and Surg. Journ.*, lxi. 279. Female, aged thirty-five years, Marblehead, Mass. Liver; echinococci expectorated, and passed per rectum.
- Case 19.—Sands, 1861. *Amer. Med. Times*, ii. 376. Female, adult, New York City. Cyst in fascia of neck.
- Case 20.—Gross, 1862. *System of Surgery*, 2 Ed., ii. 968. Hydatid of breast.
- Case 21.—Gross, 1862. *System of Surgery*, 2 Ed., ii. 968. Hydatid of breast.
- Case 22.—Simmons, 1864. *Pacific Med. and Surg. Journ.*, San Francisco, ii. 264-266. Male, aged ten years, Sacramento, Cal. Abdominal tumor; echinococci passed per rectum.
- Case 23.—Marsh, 1869. *Cincin Lancet and Obstet.*, N. S., xii. 539-543. Male, aged fifty-three years, Indian Hill, O. Kidney.
- Case 24.—Nancrede, 1871. *Trans. Path. Soc., Phil.*, iii. 1866-1876, 146, and *Amer. Journ. Med. Sci.*, N. S., lix. 417-421. Male, adult, Philadelphia, Pa. Hydatid cyst of liver.
- Case 25.—Sherard, 1872. *Med. and Surg. Rep.*, Phil., xxvii. 143-145. Female, aged twenty-nine years, Mobile, Ala. Hydatid passed per rectum.
- Case 26.—Hutchinson, J. H., 1874. *Trans. Phil. Path. Soc.*, iv. 38-40. Male, aged thirty two years, French, Philadelphia, Pa. Large cyst of liver containing hundreds of daughter cysts; small cyst of liver; large puckered cicatrix in liver; large cyst from connective tissue of pelvis.
- Case 27.—Tyson, J., 1874. *Trans. Phil. Path. Soc.*, iv. 98-99. Male, aged thirty-two years, Philadelphia, Pa. Small cyst of liver; scolices. (*Phil. Hosp.*)
- Case 28.—McKinnon, 1874. *Amer. Med. Weekly*, i. 79-81. Female, Selma, Ala. Bladder.
- Case 29.—Ellis, 1874. *Boston Medical and Surgical Journal*, xc. 568-559. Male, aged forty-three years, Asorian, Boston, Mass.
- Case 30.—Osgood, H. R., 1876. *Transactions of the Philadelphia Pathological Society*. Female, aged eighty-one years, Philadelphia, Pa. Small degenerated cyst of liver; hooklets, cholesterol.
- Case 31.—Pierce, Bartholow, Campbell, 1877. *Clinic*, Cincinnati, xii. 157, 169, 181. Male, aged forty-five years, Indianapolis, Ind. Liver.
- Case 32.—Dean, 1877. *St. Louis Medical and Surgical Journal*, xiv. 420-421, and *Transactions of the New York Pathological Society*, iii. Female, negro, St. Louis, Mo. Echinococcus multilocularis of liver.
- Case 33.—Dean, 1877. *St. Louis Medical and Surgical Journal*, xiv. 420-424. Male, aged thirty-nine years, Bavarian, St. Louis, Mo. Echinococcus multilocularis of liver.
- Case 34.—Fauntleroy, 1878. *Virginia Medical Monthly*, Richmond, iv. 282-284. Aged forty-five years, Staunton, Va. Hydatids in kidney.
- Case 35.—Drude, 1878. *Virginia Medical Monthly*, Richmond, iv. 666-670. Male, aged forty-four years, German, Quincy, Ill. Liver.
- Case 36.—Delafield, 1878. *Philadelphia Medical Times*, viii. 609-611. Male, aged forty-six years, German, New York City. Hydatids of omentum.
- Case 37.—Loomis, 1878. *Medical Record*, New York, xiv. 281-283. Male, aged forty-three years, Irish, New York City. Hydatids of lung.
- Case 38.—Jacobi, A., 1879. *Transactions of the New York Pathological Society*, iii. New York City. Cyst in liver, degenerated; hooklets and cholesterol. (Bellevue Hospital).
- Case 39.—Metcalfe, J. T., 1879. *Transactions of the New York Pathological Society*, iii. New York City. Degenerated cyst in liver; hooklets and cholesterol. (Bellevue Hospital).
- Case 40.—McCreedy, 1879. *Transactions of the New York Pathological Society*, iii. Male, old. New York City. Echinococcus in common bile duct; three daughter cysts.
- Case 41.—Loomis, A. L., 1879. *Transactions of the New York Pathological Society*, iii. Male, aged thirty-eight years. New York City. Large cyst in liver; daughter cysts and hooklets.
- Case 42.—Keyes, E. L., 1879. *Transactions of the New York Pathological Society*, iii. Male, English, New York City. Cyst in liver; cyst-shreds vomited and passed per rectum.
- Case 43.—Jacobi, A., 1879. *Transactions of the New York Pathological Society*, iii. Female, aged twenty-nine years. New York City. Large cyst in liver; daughter cysts and scolices.
- Case 44.—Cory, D. M., 1879. *Transactions of the New York Pathological Society*, iii. Male, German, New York City. Cysts in liver; scolices.
- Case 45.—Van Buren, 1879. *Transactions of the New York Pathological Society*, iii. New York City. Cyst in liver.

- Case 46.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. New York City. Cyst in liver; daughter cysts.
- Case 47.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. Female, adult, New York City. Echinococci vomited from liver or omentum.
- Case 48.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. Male, old, New York City. Tumor in common bile-duct; obsolete cysts in liver; no hooklets.
- Case 49.—Clark, A., 1879. Transactions of the New York Pathological Society, iii. New York City. Liver.
- Case 50.—McCready, 1879. Transactions of the New York Pathological Society, iii. New York City. Large cyst in liver; hooklets.
- Case 51.—Alnsworth, 1880. Medical Record, New York, xviii, 346-347. Male, aged forty years, Polish, Fort Vancouver, Wash. Cyst in lung, spleen, and bladder.
- Case 52.—Polk, 1880. Medical and Surgical Reporter, Philadelphia, xlii, 290-291. Female, adult, New York City. Cysts in gastro-hepatic omentum.
- Case 53.—Hart, 1880. Cincinnati Lancet and Clinic, N. S. iv, 495. Male, German, Cincinnati, O. Cyst in liver.
- Case 54.—Osler, W., 1880. Montreal General Hospital Reports, i, 314. Male, Montreal, Quebec. Cyst in liver; scolices.
- Case 55.—Eldridge, 1881. American Journal of Obstetrics, New York, xiv. Female, aged twenty years, Japanese, Washington, D. C. Bladder.
- Case 56.—Fenger, 1881. American Journal of the Medical Sciences, Oct., 377. Male, aged thirty-seven years, Italian, Chicago, Ill. Cyst in lung.
- Case 57.—Pollak, 1881. St. Louis Medical and Surgical Journal, xli, 492-493. Female, aged twenty-six years, St. Louis, Mo. Bladder.
- Case 58.—Smith, D. F., 1882. Canada Medical and Surgical Journal, xi, 195-197. Female, aged twenty years, Walkerton, Ont. Lung; discharging through sinus in chest wall behind.
- Case 59.—Bernays, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Male, adult, English (from Honolulu), St. Louis, Mo. Echinococci expectorated from lungs.
- Case 60.—Bernays, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Female, adult, German, St. Louis, Mo. Cyst in liver, rupturing into bowel.
- Case 61.—Hyndman, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Cincinnati, O. Echinococcus of liver.
- Case 62.—Hyndman, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Cincinnati, O. Echinococcus of brain.
- Case 63.—Ogden, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Female, foreigner, New Orleans, La. Cyst in liver.
- Case 64.—Buchan, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Female, adult, Icelandic, Toronto, Ont. Cyst in liver; scolices.
- Case 65.—Cameron, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Female, Toronto, Ont. Cyst in walls of pelvis; two cysts in liver, rupturing into intestine.
- Case 66.—Cameron, 1882. American Journal of the Medical Sciences, lxxxiv, 475-480. Female, young adult, English, Toronto, Ont. Cyst in liver.
- Case 67.—Black, 1882. Canada Medical and Surgical Journal, xi, 140-144. Male, aged twenty-nine years, English (6 years in Canada), Uxbridge, Ont. Cyst in spleen; suppurating cyst in liver, rupturing into lung; cysts expectorated.
- Case 68.—Anglin, W. G., 1882. Canada Journal of the Medical Sciences, Toronto, vii, 260-262. Male, aged sixty-two years, English, Kingston, Ont. Obsolete cyst of liver; hooklets and cholesterolin.
- Case 69.—Helm, 1883-1884. New England Medical Monthly, Sandy Hook, Ct., iii, 106-111. Female, aged twenty-six years, English, Sing Sing, New York. Hydatids of liver; rupture into intestine.
- Case 70.—Helm, 1883-1884. New England Medical Monthly, Sandy Hook, Ct., iii, 106-111. Female, aged forty years, German, Sing Sing, N. Y. Hydatids of liver; rupture into intestine.
- Case 71.—Scheaffer, 1884. Transactions of the Medical Society of Penna., xvi, 474. Male, aged fifty-four years, Lewistown, Pa. Hydatids of liver.
- Case 72.—Carson, 1884. St. Louis Courier of Medicine, xli, 455. Male, adult, St. Louis, Mo. Hydatids of liver.
- Case 73.—Mayer, 1885. Medical Record, N. Y., xxvii, 261. Female, aged thirty-eight years, Hartford, Conn. Liver.
- Case 74.—Osler, W., 1885. Transactions of the Pathological Society, Phila., xli, 217-218. Male, aged fifty-eight years, English, Ontario, Can. Cysts passed in urine; hooklets.
- Case 75.—Weir, R. F., 1885. N. Y. Medical Journal, xli, 311-312. Male, aged twenty-six years, English, New Jersey. Bilobed cyst of liver, containing hundreds of daughter cysts; cyst of mesentery.
- Case 76.—Kerr, 1886. Canada Medical and Surgical Journal, xv, 120. Icelandic, Winnipeg, Man. Cyst of liver. (Winnipeg Gen. Hosp.)
- Case 77.—Whittaker, 1886. Cincinnati Lancet and Clinic, N. S., xvi, 704-716. Male, aged sixty-nine years, German, Cincinnati, O. Echinococcus in liver.
- Case 78.—Bauer, 1886. Philadelphia Medical Times, xvii, 180. Male, aged thirty-five years, German, Philadelphia, Pa. Liver.
- Case 79.—Van Gieson, I. J., 1887. Proceedings of the New York Pathological Society, 160-161. Female, adult, German, New York City. Multiple cysts: liver, omentum, abdominal wall, peritoneum, fascia of psoas muscle (4 cysts) mesentery, and uterus; daughter cysts, lamellated membrane, cholesterolin; no scolices or hooklets found.
- Case 80.—Stavely, A. L., 1889. Montreal Medical Journal, xviii, 148-150. Male, aged forty-three years, Russian (Pole), Reading, Pa. Enormous cyst of right kidney; hooklets.
- Case 81.—Ferguson, F., 1890. Proceedings of the New York Pathological Society, 78. New York City. Cyst of liver; daughter cysts and hooklets.

¹ Specimens (Nos. 1342-44) in the Army Medical Museum, Washington, D. C.

² Specimens in the museum of McGill Medical School, Montreal.

³ Age stated by Osler (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1882, lxxxiv, 476).

- Case 82.—Chown, H. H., 1890. Medical Record, New York, xxxviii. 469, and personal communication. Male, aged thirty years, Iceland, Winnipeg, Man. Sixteen cysts of different parts of abdominal cavity (first operation); two cysts in peritoneal cavity and a group of cysts in and below liver (second operation); suppurating cyst of spleen with daughter cysts (third operation).
- Case 83.—Freeman, R. G., 1890. Proceedings of the New York Pathological Society, 7. Female, aged forty-four years, Sicilian (3 years in U. S.), New York City. Two small cysts of liver; scolices, hooklets, lamellated membrane, daughter, granddaughter, and great-granddaughter cysts. (Roosevelt Hospital, January, 1889.)
- Case 84.—Kinyoun, 1890-1891. U. S. Marine Hospital Report, xlix. 147-149. Male, aged thirty-eight years, Swede, New York City. Cysts in liver, kidneys, and bladder.
- Case 85.—Allaben, 1891. North American Practitioner, Chicago, iii. 612-617. Female, aged fifty-two years, German, Argyle, Ill. Multiple cysts in bladder, pelvis, liver, spleen, kidney, omentum, peritoneum, diaphragm, and pericardium; had passed cysts per rectum; hydatids removed from abdominal cavity in 1879.
- Case 86.—Shattuck, F. C., 1891. Boston Medical and Surgical Journal, cxxiv. 3-5 and 11. Female, aged twenty-three years, Iceland (American parents), Sandwich, Mass. Large suppurating cyst of liver; daughter cysts.
- Case 87.—Knapp, P. C., 1891. Boston Medical and Surgical Journal, cxxiv. 10. Female, aged twenty to thirty years, English, Boston, Mass. Two small cheesy cysts of liver. (Boston City Hospital.)
- Case 88.—Shattuck and Bradford, 1891. Boston Medical and Surgical Journal, cxxiv. 5. Boston, Mass. Large suppurating hydatid cyst of liver; daughter cysts. (Boston City Hosp.)
- Case 89.—Musser, J. H., 1891. Transactions of the Philadelphia Pathological Society, xiv. 88. Female, old, Philadelphia, Pa. Small cyst of liver; hooklets, cholesterol.
- Case 90.—de Schweinitz, G. E., 1891. Transactions of the Philadelphia Pathological Society, xiv. 202. Male, aged twenty years, English, Philadelphia, Pa. Cyst in abdominal muscles of right iliac region; brood-sacs, scolices.
- Case 91.—Hatch, J. L., 1891. Transactions of the Philadelphia Pathological Society, xv. 389. Old, Philadelphia, Pa. Cyst in liver and spleen. (Philadelphia Hospital.)
- Case 92.—Biggs, G. P., 1891. Proceedings of the New York Pathological Society, 47. Male, aged sixty-two years, New York City. Two small cysts of liver; scolices, hooklets, cholesterol, and lamellated membrane.
- Case 93.—Lafleur, 1892. Montreal Medical Journal, xx. 694-695. Male, foreigner, Montreal, Quebec. Cyst of liver; hooklets.
- Case 94.—Richardson, 1892. American Journal of the Medical Sciences, Oct., 485-486. Cyst in liver.
- Case 95.—Mudd, 1892. American Journal of the Medical Sciences, Oct., 412-422. Female, aged twelve years, St. Louis, Mo. Echinococcus of brain. (Surgically a remarkable case.)
- Case 96.—Solis-Cohen, 1892. *Sajous' Annual*, i., c. 33, and New York Medical Journal (1895), xlii. 763. Male, adult, German, Philadelphia, Pa. Liver.
- Case 97.—Solis-Cohen, 1892. *Sajous' Annual*, i., c. 33, and New York Medical Journal (1895), xlii. 763. Male, adult, German, Philadelphia, Pa. Liver.
- Case 98.—Solis-Cohen, 1892. *Sajous' Annual*, i., c. 33, and New York Medical Journal (1895), xlii. 763. Male, adult, Italian, Philadelphia, Pa. Liver.
- Case 99.—Haggard, G. D., 1893. Northwestern Lancet, St. Paul, xlii. 8:5. Male, aged twenty-six years, Iceland, Minneapolis, Minn. Cysts in liver, spleen, mesentery, and pelvis. (Minneapolis City Hospital.)
- Case 100.—Stamm, 1893. Medical News, i. 365-369. Female, aged forty-nine years, German, Cleveland, O. Abdominal cavity.
- Case 101.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Female, adult, Iceland, Winnipeg, Man. Cyst in abdomen, connecting with liver.
- Case 102.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Male, aged twenty-five years, Iceland, Winnipeg, Man. Cyst in liver, rupturing into hepatic duct.
- Case 103.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Female, aged ten years, Iceland, Winnipeg, Man. Five cysts in liver.
- Case 104.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Male, aged twenty-four years, Iceland, Winnipeg, Man. Two large cysts in liver, containing "five common wash-basinsful of cysts and fluid."
- Case 105.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Male, aged thirty-nine years, Iceland, Winnipeg, Man. Three large calcified cysts in liver.
- Case 106.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Male, aged thirty-nine years, Iceland, Winnipeg, Man. Two cysts in liver; vomited cysts.
- Case 107.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Iceland, Winnipeg, Man. Cyst in lung.
- Case 108.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Iceland, Winnipeg, Man. Kidney.
- Case 109.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Iceland, Winnipeg, Man. Kidney.
- Case 110.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Iceland, Winnipeg, Man. Peritoneum.
- Case 111.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Iceland, Winnipeg, Man. Peritoneum.
- Case 112.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Male, Iceland, Winnipeg, Man. Testicle.
- Cases 113-127.—Ferguson, A. H., 1893. Northwestern Lancet, xlii. 41-48. Icelanders, Winnipeg, Man. Liver. (Fifteen cases in addition to those above given; no details.)
- Case 128.—Biggs, G. P., 1893. Proceedings of the New York Pathological Society, 95. Male, aged thirty-five years, Italian, New York City. Large cyst of liver; daughter cysts, scolices and hooklets.
- Case 129.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). Child, San Antonio, Texas. A great many echinococci in bladder; hooklets.
- Case 130.—Herff, 1894. Texas Medical Journal, Austin, ix. 613-616, and xv. 237-245 (1899). Female, adult, San Antonio, Texas. Passed per rectum, probably from liver; hooklets.

- Case 131.—Herff, 1891. *Texas Medical Journal*, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Bulbus of eye; hooklets.
- Case 132.—Herff, 1891. *Texas Medical Journal*, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Extensor muscles of thigh; hooklets.
- Case 133.—Herff, 1891. *Texas Medical Journal*, Austin, ix. 612-616, and xv. 237-245 (1899). San Antonio, Texas. Liver; one quart of fluid; hooklets.
- Case 134.—Herff, 1891. *Texas Medical Journal*, Austin, ix. 613-616, and xv. 237-245 (1899). San Antonio, Texas. Liver; two quarts of fluid; hooklets.
- Case 135.—Hartwig, M. 1894. *Medical Record*, N. Y., xli. 167. Male, Italian, Buffalo, N. Y. Cyst of abdomen, probably connecting with liver.
- Case 136.—Page, 1895. *Medical Record*, N. Y., xlviii. (September). Female, aged fifty-one years, German, Hiram, O. Liver, pleura, omentum, mesentery, peritoneum, and bladder.
- Case 137.—Van Cott, 1895. *New York Medical Journal*, lxii. 656-659. Mexican, Brooklyn.
- Case 138.—Frank, J., 1896. *American Journal of the Medical Sciences*, cxii. N. S., 437-443. Female, aged thirty-two years, Russian, Chicago, Ill. Large cyst of liver.
- Case 139.—Frank, J., 1896. *American Journal of the Medical Sciences*, cxii. N. S., 437-443. Male, aged twenty-eight years, German, Chicago, Ill. Large cyst of liver, containing hundreds of daughter cysts.
- Case 140.—Hughes, W. E., 1896. *Transactions of the Philadelphia Pathological Society*, xvii. 53. Male, aged thirty years, negro, Philadelphia, Pa. Small cyst of liver.
- Case 141.—Custis, 1896. *New York Medical Journal*, lxiv. 363-365. Adult, Washington, D. C. Vomited (or expectorated?).
- Case 142.———, 1896. *Philadelphia Evening Star*, June 13. Male, Muskegon, Mich. Liver.
- Case 143.—Keyes and Busch, 1896. *Buffalo Medical Journal*, xxxv. 25-29. Male, aged fifty-three years, German, Buffalo, N. Y. Multiple cysts in lung, omentum, mesentery, subdiaphragmatic, left kidney, and sac of left inguinal hernia; scolices and hooklets.
- Case 144.—Cordier, 1896. *Lancet*, Kansas City, i. 180-182. Female, aged forty-six years, Kansas City, Mo. Liver; sterile.
- Case 145.—Lloyd, 1896. *American Med.-Surg. Bull.*, N. Y., x. 659-661. Male, aged fifty-seven years, Dane, New York City. Muscles of back; about 250 cysts.
- Case 146.—Cheney, W. F., 1897. *Archives of Pediatrics*, N. Y., xiv. 851-855. Male, aged seven years, Italian, San Francisco, Cal. Two large cysts in liver; daughter cysts.
- Case 147.—McNamara, S. J., 1897. *Medical News*, New York, lxxi. 694-695. Female, aged thirty-one years, Italian (9 years in U. S.), Brooklyn, N. Y. Large cyst in upper part of abdominal cavity, probably liver, containing 26 pounds of contents and thousands of daughter cysts; omentum studded with "secondary deposits."
- Case 148.—Larkin, J. H., 1897. *Proceedings of the New York Pathological Society*, 62-63. Male, aged eighty-three years, New York City. Large calcareous cyst of liver, with diaphragm adherent and forming upper part of cyst wall; cholesterolin; scolices and hooklets absent.
- Case 149.—PHELPS, C., 1898. *New York Medical Journal*, lxvii. 218-219. Male, aged fifty-one years, New York City. Three large cysts attached to greater curvature of stomach and six to the colon; daughter cysts; no hooklets.
- Case 150.—Packard, F. A., 1898. *Transactions of the Philadelphia Pathological Society*, xviii. 101. Female, aged thirty-four years, negro, Philadelphia, Pa. Small cyst of liver; hooklets, cholesterolin, lamellated cyst wall. (Pennsylvania Hospital, 1896.)
- Case 151.—Steele, J. D., 1898. *Transactions of the Philadelphia Pathological Society*, N. S., i. 106. Adult (Western town). Cyst of liver; scolices, hooklets, and lamellated cyst wall.
- Case 152.—Mason, A. L., and Burrell, H. L., 1898. *Medical and Surgical Rep., Boston City Hospital*, Ser. ix. 108-115. Boston, Mass. Cyst of liver; no hooklets.
- Case 153.—Mason, A. L., and Burrell, H. L., 1898. *Medical and Surgical Rep., Boston City Hospital*, Ser. ix. 108-115. Male, American, Boston, Mass. Large purulent cyst of liver, with secondary involvement of right pleural cavity; daughter cysts.
- Case 154.—Beck, C., 1898. *Journal of the American Medical Association*, xxxi. 1238-1239. Male, aged thirty-eight years, Austrian (7 years in U. S.), New York City. Purulent cyst in right lung; expectorated pus containing hooklets. (St. Mark's Hospital, 1897.)
- Case 155.—Beck, C., 1898. *Journal of the American Medical Association*, xxxi. 1238-1239. Male, aged thirty-four years, German (8 years in U. S.), New York City. *Echinococcus* of liver. (St. Mark's Hospital, 1892.)
- Case 156.—Lane, 1899. *Pacific Record of Medicine and Surgery*, San Francisco, N. S., i. 314. San Rafael, Cal. Cyst of liver.
- Case 157.—Berg, 1899. *Medical Record*, New York, lv. 26. Male, aged thirty years, New York City. Large cyst of liver; rupture into common bile-duct; passed per rectum.
- Case 158.—Lillenthal, 1899. *Medical Record*, New York, lv. 26. Male, young adult, New York City. Cyst in left breast.
- Case 159.—Willen, 1899. *Journal of the American Medical Association*, xxxii. 114-115. Male, aged fifty years, Fontanet, Ind. Large cyst in liver.
- Case 160.—Weir, 1899. *Medical Record*, New York, lv. 149-151. Male, aged thirty-nine years, German (?), New York City. Large cyst in liver; scolices.
- Case 161.—Woods, G. W., 1899. *Proceedings Assoc. Military Surg. of the U. S.*, viii. 157-160. Male, Brooklyn, N. Y. Large cyst of liver extending through diaphragm into right pleural cavity; daughter cysts, scolices, hooklets; also small cyst in head of left humerus. (U. S. Naval Hospital, Brooklyn, N. Y.)
- Case 162.—Cary, C., and Lyon, I. P., 1900. *Transactions of the Association of American Physicians*, xv. 367-379, and *American Journal of the Medical Sciences*, cxx. 492-413. Male, aged thirty-one years, Negro (Va.), Buffalo, N. Y. Primary exogenous cysts of right pleura; hyaline degeneration of cuticle, without gross degeneration; hooklets and cholesterolin.

¹ Sommer (New York Medical Journal, 1896, lxiv. 265) says that this case was in a Mexican, N. Y. Liver.

² Specimens in the museum of the University of Buffalo

³ Ibid.

- Case 163.—Henry, F. P., 1900. Philadelphia Medical Journal, v. 880, and Personal Communication. Male, aged forty-nine years, negro (Va.), Philadelphia, Pa. Two cysts in liver; hooklets.
- Case 164.—Porter, C. A., 1900. Boston Medical and Surgical Journal, cxiii. 302. Male, aged seventeen years, Italian, Boston, Mass. Large cyst of liver; lamellated cyst membrane; scolices and hooklets not found. (Massachusetts General Hospital).
- Case 165.—Rogers, W. K., 1900. Journal of the American Medical Association, xxiv. 276, 277. Female, aged thirty-four years, Newark, O. Cysts of nasal septum; hooklets.
- Case 166.—Odendahl, F. H., 1900. Northwestern Lancet, St. Paul, xx. 29-30. Female, aged thirty-two years, St. Paul, Minn. Large cyst of liver, containing one gallon of fluid and several hundred small cysts.
- Case 167.—Odendahl, F. H., 1900. Northwestern Lancet, St. Paul, xx. 29-30. Female, aged thirty-five years, St. Paul, Minn. Liver; cysts passed per rectum.
- Case 168.—Vickery, H. F., 1900. Transactions of the Association of American Physicians, xv. 573, and Personal Communication. Male, aged twenty-nine years, French, Boston, Mass. Purulent mass in right pleural cavity; daughter cysts. (Mass. Gen. Hosp., 1896).
- Case 169.—Shorb, J. D. B., 1901. Southern Cal. Practitioner, xv. 472-474. Female, aged fifty-eight years, German (20 years in U. S.), Los Angeles, Cal. Large degenerated cyst cavity in liver; daughter cysts; cysts passed per rectum.
- Case 170.—Bartholow, 1900. Annals of Surgery, xxxi. 721-722. Male. Large single cyst on posterior surface of liver; gallon of fluid.
- Case 171.—Da Costa, J. C., 1900. Annals of Surgery, xxxii. 721. Female, aged twenty-nine years. Suppurative cyst of liver.
- Case 172.—Loux, H. R., 1900. Annals of Surgery, xxxii. 714-728. Male, aged thirty-one years, German, Philadelphia, Pa. Single cyst attached to lower surface of liver; weight, 197 grammes; numerous daughter cysts; hooklets, scolices, brood-capsule, cholesterol.
- Case 173.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Female, aged twenty-eight years, Italian (3 years in U. S.), Brooklyn, N. Y. Single cyst arising from under surface of liver.
- Cases 174-178.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Italians, Brooklyn, N. Y. Liver. (Five cases).
- Case 179.—Fowler, R. S., 1900. Annals of Surgery, xxxii. 812-822. Brooklyn, N. Y. Large cyst in left lobe of liver.
- Case 180.—Bolton, P. R., 1900. Annals of Surgery, xxxi. 253-254. Male, aged eighteen years, Italian (3 years in U. S.), New York City. Single cyst in under surface of liver; lamellated cyst wall; no hooklets.
- Case 181.—Mercier, O. F., 1900. L'Union méd du Canada, Montreal, xxix. (N. S., v.), 427-438. Male, aged fifty-five years, Canadian, Montreal, Quebec. Large cyst in the region of the right kidney; daughter cysts. (Hôpital Notre-Dame, 1900).
- Case 182.—Mercier, O. F., 1900. L'Union méd du Canada, Montreal, xxix. (N. S., v.), 427-438. Male, aged thirty-four years, Syrian, Montreal, Quebec. Large cyst of liver; daughter cysts. (Hôpital Notre-Dame, 1900).
- Case 183.—Park, R., 1901. Personal communication. Male, aged thirty-five years, Buffalo, N. Y. Multilocular cyst of liver, degenerated; lamellated cyst wall; no scolices or hooklets.
- Case 184.—Park, R., 1901. Personal communication. Male, aged twenty-six years, English (2 years in U. S.), Lyndonville, N. Y. Two cysts in liver, one calcified and one, as large as an infant's head, protruding into abdominal cavity.
- Case 185.—Fronczak, F. E., 1901. Personal communication. Female, aged forty-five years, Polish, Buffalo, N. Y. Single cyst of liver; hooklets. (Erie Co. Hospital, Buffalo, 1897).
- Case 186.—Kenerson, V., 1901. Personal communication. Male, aged thirty-five years, Irish, New York City. Cyst of liver, purulent; daughter cysts and hooklets. (New York Hospital, 1896).
- Case 187.—Packard, F. A., 1901. Personal communication. Male, negro, Philadelphia, Pa. Hydatid cyst in liver; hooklets and scolices. (Philadelphia Hospital.)
- Case 188.—Packard, F. A., 1901. Personal communication. Female, aged fifty-nine years, negro, Philadelphia, Pa. Small cyst in liver; hooklets, cholesterol, and lamellated cyst wall. (Pennsylvania Hospital, 1887.)
- Case 189.—Packard, F. A., 1901. Personal communication. Female, aged twenty-seven years, negro, Philadelphia, Pa. Multilocular hydatid cyst of right breast; hooklets (Pennsylvania Hospital, 1889).
- Case 190.—Packard, F. A., 1901. Personal communication. Female, aged forty years, Syrian, Philadelphia, Pa. Cyst of liver. (Pennsylvania Hospital, 1889.)
- Case 191.—Chown, H. H., 1901. Personal communication. Female, aged thirty-two years, Iceland, Winnipeg, Man. Neck; hooklets.
- Case 192.—Chown, H. H., 1901. Personal communication. Male, aged thirty-six years, Iceland, Winnipeg, Man. Spleen; daughter cysts.
- Case 193.—Chown, H. H., 1901. Personal communication. Male, aged eighteen years, Iceland, Winnipeg, Man. Arm, above elbow; hooklets.
- Case 194.—Chown, H. H., 1901. Personal communication. Female, aged twenty-five years, Iceland, Winnipeg, Man. Cyst of liver, resembling a dilated gall bladder; hooklets; subsequently operated on in Boston for a second cyst.
- Case 195.—Chown, H. H., 1901. Personal communication. Male, aged twenty-four years, Iceland, Winnipeg, Man. Left iliac region, apparently post-peritoneal; daughter cysts.
- Case 196.—Chown, H. H., 1901. Personal communication. Male, aged fifty-six years, Iceland, Winnipeg, Man. Lung; expectorated collapsed daughter cysts for months.
- Case 197.—Chown, H. H., 1901. Personal communication. Female, aged thirty-four years, Iceland, Winnipeg, Man. Pelvis; rupture into bladder; cysts passed per urethram.
- Case 198.—Chown, H. H., 1901. Personal communication. Female, aged forty-six years, Iceland, Winnipeg, Man. Calcareous cyst of liver; death from intestinal hemorrhage.

¹ Specimens in the museum of the University of Buffalo.

² Ibid.

³ Specimens in the museum of the New York Hospital.

- Case 199.—Chown, H. H., 1901. Personal communication. Male, aged thirty-seven years. Iceland, Winnipeg, Man. Liver; hooklets.
- Case 200.—Chown, H. H., 1901. Personal communication. Female, aged twenty-three years, Iceland, Winnipeg, Man. Cyst of peritoneal cavity.
- Case 201.—Chown, H. H., 1901. Personal communication. Female, aged seventeen years, Iceland, Winnipeg, Man. Liver; hooklets.
- Case 202.—Chown, H. H., 1901. Personal communication. Male, aged twenty-seven years, Iceland, Winnipeg, Man. Cyst of liver; daughter cysts.
- Case 203.—Chown, H. H., 1901. Personal communication. Female, aged twenty-three years, Canadian (both parents Icelanders), Winnipeg, Man. Cyst of liver; daughter cysts.
- Case 204.—Chown, H. H., 1901. Personal communication. Female, aged twenty-six years, Iceland, Winnipeg, Man. Peritoneal cavity, cyst floating free; patient was previously operated on in London for large hydatid of left lumbar region.
- Cases 205-217.—Chown, H. H., 1901. Personal communication. Thirteen cases (in addition to the above), eight in females and five in males; ages, 23, 26, 31, 33, 33, 36, 37, 39, 42, 43, 44, 45, and 51 years. Icelanders, Winnipeg, Man. All cases had cysts of the liver, with daughter cysts, and varying only in size.
- Case 218.—Bangs, L. B., 1901. *Annals of Surgery*, xxxiii. 565-573. Male, aged thirty-nine years, German, New York City. Cyst of prostate; daughter cysts and hooklets. (Bellevue Hospital, 1900.)
- Case 219.—Gay, W. F., 1901. *Boston Medical and Surgical Journal*, cxliv. 492-493. Male, aged twenty-five years, Italian (4 years in U. S.), Boston, Mass. Hydatid of liver, with rupture into lung and expectoration of cysts; sputum contained scolices, lamellated cyst membrane, etc.
- Case 220.—Peters, G. A., 1901. *Canadian Practitioner and Review*, xxvi. 75-78. Male, aged twenty years, Argentine Republic, Guelph, Ont. Large cyst in tail of pancreas [clinical diagnosis of site]; brood capsules and hooklets.

MUSEUM SPECIMENS¹ (NOT REPORTED).

- Case 221.—McGill Medical School Museum, Montreal. Male, aged thirty-five years, Italian (4 years in America), Montreal, Quebec. Multiple cysts in liver, spleen, stomach, omentum, mesentery and pelvis; perforation of stomach and duodenum (1880).
- Case 222.—McGill Medical School Museum, Montreal. Female, aged forty years, English, Montreal, Quebec. Obsolete cyst in liver; hooklets.
- Case 223.—McGill Medical School Museum, Montreal. Montreal, Quebec. Cyst in liver.
- Case 224.—Miner. United States Army Medical Museum, Washington, No. 8014. Male, adult, colored, Alexandria, Va. Echinococcus of brain.
- Case 225.—Bond. United States Army Medical Museum, Washington, No. 8089. Male, aged fifty-one years, light mulatto, Washington, D. C. Cyst in liver.
- Case 226.—Warren Anatomical Museum, Harvard, Boston, No. 2381. Cyst in liver.
- Case 227.—Warren Anatomical Museum, Harvard, Boston, No. 2156. Cavity at apex of left lung, containing echinococci.
- Case 228.—Warren Anatomical Museum, Harvard, Boston, No. 3773. Echinococci passed per rectum.
- Case 229.—Warren Anatomical Museum, Harvard, Boston, No. 3871. Male, adult. Cyst in liver.
- Case 240.—Bellevue Hospital Museum, New York, No. 865. Cyst in liver.
- Case 281.—Bellevue Hospital Museum, New York, No. 866. Cyst in liver.
- Case 282.—Bellevue Hospital Museum, New York, No. 867. Suppurating cyst of liver.
- Case 233.—New York Hospital Museum, No. 932. New York City. Cyst in liver.
- Case 284.—New York Hospital Museum, Nos. 933-937. New York City. Multiple echinococci: one in abdominal wall, one on surface of liver, a third loose in peritoneal cavity, and a fourth in pelvis.
- Case 235.—Loomis. Museum of University of New York. Large cyst of liver.
- Case 236.—Museum of Pennsylvania Hospital, Philadelphia, No. 1882²⁰. Cyst in liver.
- Case 287.—Museum of Pennsylvania Hospital, Philadelphia, No. 1882²⁴. Male, lad, French. Multiple cysts in liver.
- Case 238.—Museum of Pennsylvania Hospital, Philadelphia. Aged fifty-five years, Italian. Philadelphia, Pa. Cyst in liver.
- Case 289.¹—Museum of University of Pennsylvania, G. B. Wood Cabinet, P. C., 46, vol. i. Several cysts from liver.
- Case 240.²—Museum of University of Pennsylvania, G. B. Wood Cabinet. Hydatid cyst of spleen.
- Case 241.⁴—Museum of University of Pennsylvania, G. B. Wood Cabinet. Liver with a cyst, probably hydatid.

AGE. The occurrence of the disease according to age is shown in Table II. by decades, and graphically in the accompanying age-chart:

¹ Most of these museum specimens were originally summarized by Osler (loc. cit.).

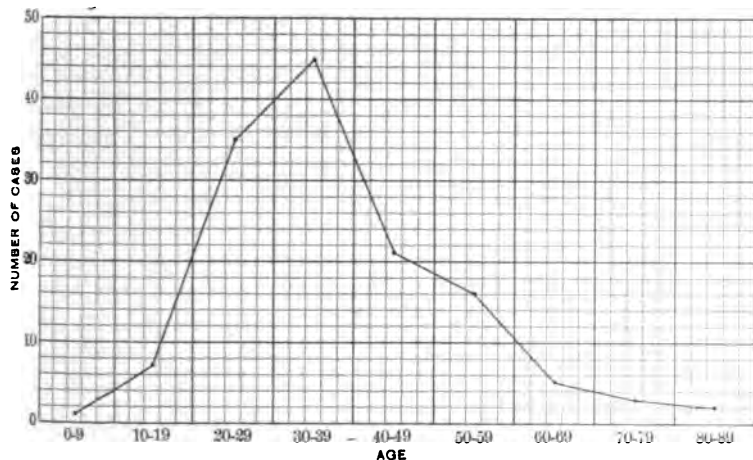
² Osler states that this case is perhaps not from American sources.

³ Ibid.

⁴ Ibid.

TABLE II.—AGE.

| | | | | | | | | | |
|-------------|-------|---|---|---|---|----------------|---------|--------------|--------------|
| 0-9 | years | . | . | . | . | 1 ¹ | | | |
| 10-19 | " | . | . | . | . | 7 | | | |
| 20-29 | " | . | . | . | . | 35 = 26 | per ct. | } 59 per ct. | } 74 per ct. |
| 30-39 | " | . | . | . | . | 45 = 33 | " | | |
| 40-49 | " | . | . | . | . | 21 = 15 | " | | |
| 50-59 | " | . | . | . | . | 16 | | | |
| 60-69 | " | . | . | . | . | 5 | | | |
| 70-79 | " | . | . | . | . | 3 | | | |
| 80-89 | " | . | . | . | . | 2 | | | |
| | | | | | | — | 135 | | |
| Child. | . | . | . | . | . | 1 | | | |
| Lad. | . | . | . | . | . | 1 | | | |
| Young adult | . | . | . | . | . | 2 | | | |
| Adult | . | . | . | . | . | 19 | | | |
| Old | . | . | . | . | . | 4 | | | |
| Unstated | . | . | . | . | . | 79 | | | |
| | | | | | | — | 106 | | |
| Total | . | . | . | . | . | | 241 | | |



A large majority (74 per cent.) of the cases whose ages were given occurred in young and middle-aged adults. In the age-period—twenty to thirty-nine years—59 per cent. of the cases occurred. Jonassen² found 55 per cent., and Finsen³ 42 per cent. in Iceland in this period of life. Under the age of ten years the disease is rare, in spite of the fact that young children are apparently more exposed than adults to infection. As shown by the table, no period of life is exempt.

SEX. The cases were divided by sex, as follows :

¹ Ferguson (vide infra) saw three cases in children under eight years of age, who had been brought to Winnipeg by Icelandic emigrants. These cases were not described individually, and have therefore been omitted from this enumeration.

² Jonassen. Echinokokkensygdomen belyst ved Islandske Lægers Erfaring, Kjøbenhavn, 1892.

³ Finsen. Jagttagelser angaaende sygdoms-fortsoldener, x., i. Iceland, Kjøbenhavn, 1874.

TABLE III.—SEX.

| | | | | | | | |
|-------------|---|---|---|---|---|---|--------------------|
| Males | : | : | : | : | : | : | 104 = 60 per cent. |
| Females | : | : | : | : | : | : | 69 = 40 " |
| | | | | | | | <hr/> 173 |
| Not stated. | : | : | : | : | : | : | 68 |
| | | | | | | | <hr/> 241 |

The preponderance of males over females (3 to 2) in the American cases is about reversed in the statistics of most authorities. Neisser¹ gives 34.8 per cent. in males and 65.2 per cent. in females; Finsen² found 29.1 per cent. in males and 70.9 per cent. in females, and Jonassen's³ figures show 40.6 per cent. in males and 59.4 per cent. in females. According to Davaine,⁴ the occurrence of the disease in the two sexes is equal. The fact that a large part of the American cases occurred among foreign immigrants, among whom young adult males predominate, may explain in part the peculiarly high proportion of males in these cases.

NATIONALITY. The representation of the cases by nationality or race is shown in Table IV., as follows:

TABLE IV.—NATIONALITY OR RACE.

| Birthplace. | | | |
|-------------------------|----|---|-----|
| Iceland | . | . | 58 |
| Germany | . | . | 23 |
| Italy | 18 | } | 19 |
| Sicily | 1 | | |
| England | 13 | } | 14 |
| Wales | 1 | | |
| Ireland | . | . | 3 |
| France | . | . | 3 |
| Poland | . | . | 2 |
| Russia | . | . | 2 |
| Syria | . | . | 2 |
| Argentine Republic | . | . | 1 |
| Azores | . | . | 1 |
| Austria | . | . | 1 |
| Denmark | . | . | 1 |
| Japan | . | . | 1 |
| Mexico | . | . | 1 |
| Sweden | . | . | 1 |
| Foreign (not specified) | . | . | 3 |
| | | | 136 |
| America | 1 | } | 13 |
| Canada | 2 | | |
| Negro | 6 | | |
| " (U. S.) | 4 | | |
| Total stated | . | . | 149 |
| White | 4 | } | 92 |
| Not stated | 88 | | |
| | | | 241 |

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| | | | | Foreign-born, 91 per cent. |
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Foreign-born, 91 per cent.

Native-born, 9 per cent.

¹ Neisser. Die Echinococcen-Krankheit., Berlin, 1877.² Finsen. Loc. cit.³ Jonassen. Loc. cit.⁴ Davaine. Traite des Entozoaires, Paris, 1877, second edition.

From this table it is seen that 136 cases were stated to be among foreigners; in 92 cases the nationality was not stated, and the remaining 13 cases were distributed as follows: Negro, 10; Canadian, 2; American, 1. Mention of American birth is conspicuously rare, though it may be inferred from the context of the reports of some cases that the subjects were native-born.

If the proportion of foreigners shown among the cases whose birth-place was stated be assumed to obtain also in the cases of unstated nationality, it is seen that 91 per cent. of the American cases occurred in foreigners. The negro has been assumed to be native-born, and, in fact, it was so stated in at least four instances. The disease seems to have been directly imported into America in the great majority of cases by emigrants from foreign countries. The relative immunity of Americans is seen even among the American-born children of the Icelandic emigrants of Manitoba, though among the latter the disease is very common. The disease prevails chiefly in the lower classes and particularly among the ignorant and slovenly, but not exclusively, for instances of the disease are recorded in persons of rank and culture.

The large representation of Germans in the table is explained by the large German emigration to America and the well-known fact that hydatid disease is very common in certain parts of Germany. The occurrence of nineteen cases among Italians is not remarkable considering the habits of this people, and the disease is said to be not uncommon in England.

The following facts, quoted from Ferguson,¹ of Winnipeg, are of interest in reference to the large number of cases of hydatid disease in Icelanders shown by the table:

"First, in 1874, and at different times since, the hydatid disease was brought to Manitoba by the influx of Icelandic immigrants.

"So far as I have been able to ascertain, the disease is limited to the Icelandic population and to those of them born in Iceland.

"I have met with it in three children under eight years of age, but upon inquiry it was found that they were born in Iceland.

"The total number of my cases has been twenty-seven, but this does not include several of doubtful diagnosis, in which objection was taken to aspiration or exploratory incision.

"From the reports of our hospitals, and from conversations with the city physicians, I have traced some eighteen or twenty² other cases treated by different medical men.

"I think that I am justified in concluding that between forty-five and fifty persons affected with the echinococcus disease have been

¹ Ferguson. *Northwestern Lancet*, St. Paul, 1893, vol. xiii. pp. 41-48.

² These 18 or 20 cases have not been included in our tables, because they were merely estimated and no details were given.

treated in Winnipeg since 1874. I feel that this number is rather below the proper reckoning."

Ferguson gave the Icelandic population of Winnipeg as 3000, and, accepting his estimate of forty-five to fifty cases of hydatid disease seen in this population, the relative frequency of occurrence is found to be about identical with that of the disease in Iceland, where, according to Jonassen, whose estimate is probably the most accurate, one out of every sixty-one persons (1.6 per cent.) is affected with hydatid disease. Ferguson's estimate, however, is probably too conservative, judging from the number of cases shown in our statistics.

It is interesting to note that Ferguson saw no cases of the disease in the Canadian-born offspring of Icelandic emigrants. Chown, however, observed one such case in a young woman, aged twenty-three years. This case, therefore, gives ground for the apprehension that an endemic focus of the disease has already been established in Winnipeg by the Icelandic colony, and that further cases may occur in the children of Icelanders as they reach maturity, the period of life in which the disease is most commonly observed.

GEOGRAPHICAL DISTRIBUTION. The geographical distribution of the American cases is shown in Table V., as follows:

TABLE V.—GEOGRAPHICAL DISTRIBUTION.

| | | | |
|--------------------------------|----|-------------------------|-----------------|
| New York | 59 | Connecticut | 1 |
| Manitoba | 56 | Louisiana | 1 |
| Pennsylvania | 24 | Michigan | 1 |
| Massachusetts | 13 | New Jersey | 1 |
| Ohio | 9 | Tennessee | 1 |
| Missouri | 8 | Vermont | 1 |
| Ontario | 8 | Washington | 1 |
| Quebec | 7 | | |
| Texas | 6 | | 220 |
| Illinois | 5 | Not specified | 21 ¹ |
| California | 4 | | |
| Minnesota | 3 | Total | 241 |
| District of Columbia | 3 | | |
| Alabama | 2 | United States | 150 |
| Indiana | 2 | Canada | 71 |
| Kentucky | 2 | Not stated | 20 |
| Virginia | 2 | | |
| | | | 241 |

There seems to be nothing noteworthy in the geographical distribution shown, except the fact that concentration of the disease in certain States is coincident with concentration of population, and especially foreign immigrant population in such States. Attention has already been called to the prevalence of the disease among the Icelandic immigrants of Manitoba. It is noticeable that so few cases are furnished by the great grazing and cattle-raising States of the West, where it would

¹ Includes one case from a "Western town" in the United States.

seem, *a priori*, that the conditions favorable to the spread of the disease might prevail.

ANATOMICAL LOCATION. The distribution of the disease by organs in the body is shown in Table VI., as follows:

TABLE VI.—ANATOMICAL LOCATION.

| | | |
|---|-----|--------------------------|
| Liver | 177 | = 73.7 per cent. |
| Omentum | 8 | |
| Peritoneal cavity | 8 | |
| Peritoneum | 5 | |
| Mesentery | 5 | |
| Lung | 11 | = 4.5 " |
| Spleen | 9 | = 3.7 " |
| Kidney | 9 | = 3.7 " |
| Bladder | 8 | = 3.3 " |
| Pelvis | 5 | |
| Walls of pelvis | 1 | |
| Connective tissue of pelvis | 1 | |
| Pleura | 5 | = 2.0 " |
| Brain | 4 | = 1.6 " |
| Breast | 4 | = 1.6 " |
| Abdominal wall | 4 | = 1.6 " |
| Stomach | 2 | = 0.8 " |
| Common bile-duct | 2 | = 0.8 " |
| Neck | 2 | = 0.8 " |
| Arm | 1 | = 0.4 " |
| Humerus | 1 | = 0.4 " |
| Pancreas | 1 | = 0.4 " |
| Eye ("bulbus of") | 1 | = 0.4 " |
| Diaphragm | 1 | = 0.4 " |
| Sub-diaphragmatic | 1 | = 0.4 " |
| Intestine | 1 | = 0.4 " |
| Muscles of back | 1 | = 0.4 " |
| Ovary | 1 | = 0.4 " |
| Pericardium | 1 | = 0.4 " |
| Prostate | 1 | = 0.4 " |
| Sac of inguinal hernia | 1 | = 0.4 " |
| Testicle | 1 | = 0.4 " |
| Uterus | 1 | = 0.4 " |
| Psoas muscle | 1 | = 0.4 " |
| Muscles of thigh | 1 | = 0.4 " |
| Nasal septum | 1 | = 0.4 " |
| | | 287 organs in 240 cases. |
| Not stated | | 1 case. |
| | | 241 cases. |
| Cysts passed per rectum | 10 | |
| Cyst membrane passed per rectum | 1 | |
| Cysts expectorated | 6 | |
| Hooklets expectorated | 1 | |
| Cysts vomited | 3 | |
| Cyst membrane vomited | 1 | |
| Cysts passed per urethram | 2 | |
| | 11 | = 4.5 per cent. |
| | 7 | = 2.9 " |
| | 4 | = 1.6 " |
| | 2 | = 0.8 " |

The liver is seen to be the seat of election of the parasite, being involved in 73.7 per cent. of the cases—a high rate compared with other statistics. Davaine¹ gives for the liver 44 per cent.; Neisser² 50

¹ Davaine. Loc. cit.

² Neisser. Loc. cit.

per cent., and Finsen¹ 69 per cent. Next to the liver the order of frequency of the organs involved by the disease is as follows: omentum, peritoneal cavity, peritoneum, and mesentery (combined), 10.8 per cent.; lung, 4.5 per cent.; spleen and kidney, each, 3.7 per cent.; bladder, 3.3 per cent., etc. Very unusual sites of the parasite are shown in our statistics in the sac of an inguinal hernia, nasal septum, pericardium, head of the humerus, brain, eye, prostate, testicle, pancreas, and pleura (primary), in one case each. The multilocular type of cysts (*echinococcus multilocularis*) is mentioned in four cases—three times in the liver and once in the female breast. Two of these cases were in negroes, in the breast and in the liver. Rupture of cysts into the following organs is mentioned: intestine, stomach, bile-ducts, bladder, peritoneal cavity, and lung. Rupture through the chest wall externally took place in one case. Discharge of cysts, cyst membrane, or hooklets by the various natural outlets of the body was mentioned as follows: Passed per rectum, 11 cases (4.5 per cent.); expectorated, 7 cases (2.9 per cent.); vomited, 4 cases (1.6 per cent.), and passed per urethram, 2 cases (0.8 per cent.).

DIAGNOSIS. The diagnosis of echinococcus disease in the American cases seems to have been based on the gross appearances of the cysts, the presence of daughter cysts, the site of the cysts, etc., in a majority of the cases. However, in a fair proportion of the more recently reported cases, such characteristic microscopical elements as brood capsules, scolices, hooklets, calcareous corpuscles, lamellated cyst membrane, etc., were mentioned in support of the diagnosis. The chemical tests of the cyst fluid for the absence of albumin and the presence of grape-sugar were commonly applied, and the specific gravity was tested in many cases. Succinic acid, said by Leuckart to be "found in hardly any other living organism," and pathognomonic of hydatid fluid, was looked for in only a few cases. The physical characteristic of the outward curling of the elastic cyst wall, when incised, was only rarely mentioned. In doubtful cases none of these points of diagnosis should be neglected, though any or all of them may be wanting in atypical or degenerated cysts.²

It has been a difficult matter, in reviewing the literature, to decide what cases ought to be excluded because of insufficient evidence of the true cestode nature of the cysts, and perhaps an occasional case has been included in the summary of cases that might better have been omitted. Without being hypercritical, we have tried to be conservative in judging of cases. It is possible, also, that an occasional case

¹ Finsen. Loc. cit.

² Cary and Lyon. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, October, 1900, vol. cxx. pp. 402-413; and Transactions of the Association of American Physicians, 1900, vol. xv. pp. 867-879.

may have been reported independently by different observers, and thus included more than once in our summary, though we have been on our guard against this source of error.

IS THE DISEASE INCREASING? This important question, we believe, cannot be answered authoritatively at present. It is true that the great majority of cases in our statistics are accredited to recent years. This fact, however, cannot be accepted as evidence that the disease is more common now than formerly. It is only within recent years that the habit of reporting rare and interesting cases in the medical journals has become general. Probably more cases of pneumonia, typhoid fever, or any other disease that might be mentioned, have been reported in the medical literature in the last quarter of a century than in all previous time. This fact plainly does not argue that these diseases are increasing in prevalence, but merely that they are being better recognized, studied, and discussed than formerly. So we are not able to affirm that hydatid disease is increasing in America. Still, one of the best authorities, Dr. C. W. Stiles,¹ zoölogist of the Bureau of Animal Industry, has stated that the disease in the domesticated animals in the United States is undoubtedly on the increase. If this be true, it is probably true also that the disease in man is on the increase in this country. Probably only a small portion of the cases occurring in the country are recognized and reported. However, the disease is still a rarity, and is considered a curiosity in medical circles.

HYDATIDS IN ANIMALS. Table VII. shows what is at present known of the occurrence of echinococcus cysts in animals² in the United States and Canada. With the exception of the records of the United States meat-inspection service, published by Dr. D. E. Salmon, chief of the Bureau of Animal Industry, the evidence seems to indicate that hydatid cysts in our domesticated animals are not uncommon, different observers finding them in from 1 to 10 per cent. of animals examined. It is difficult to reconcile these various observations with the enormous statistics of the government meat-inspection service, which indicate that the disease is a great rarity in the live-stock of the United States. In

¹ Stiles, C. W. Bull. No. 19, United States Department of Agriculture, Bureau of Animal Industry, Washington, 1898, p. 122.

Doctor Stiles, in a personal communication, writes as follows: "It is largely on the base of our unpublished statistics that I have insisted so frequently upon the necessity of guarding against hydatid disease, which is undoubtedly on the increase among the domesticated animals."

² The cysts described in the muscles of prairie jack-rabbits by Herff (Texas Medical Journal, 1904, vol. ix. pp. 613-616) and by Menger (Ibid., 1899, vol. xv. pp. 237-45; and Ibid., 1900, vol. xvi. pp. 415-20; and American Journal of Dermatology and Genito-urinary Diseases, St. Louis, 1900, vol. iv. pp. 155-163), although containing hooklets, cannot be accepted as *Echinococcus polymorphus*. They were probably *Cœnurus serialis*, the larval cysts of *Tenia serialis*, a well-known bladder-worm of rabbits in this country. The writer has occasionally encountered these cysts in white rabbits, in Buffalo, in making autopsies on experimental rabbits dead of various infections.

the two years ending June 30, 1899, only six out of 8,831,927 cattle were condemned in whole or in part on account of hydatid disease, or one in 1,471,987; 209 out of 11,110,776 sheep, or one in 53,161, and 1994 out of 44,841,779 hogs, or one in 22,488. The official authority of these figures, as well as their enormous scope, support them against the evidence of the other scattered observations recorded in Table VII., and we are, therefore, bound to assume that at present echinococcus disease is a very rare affection in the native herds and flocks of this country, as it is also among the native population.

TABLE VII.—ECHINOCOCCUS IN ANIMALS IN NORTH AMERICA.

- Gross, S. D., 1845. *Pathological Anatomy*, 2d ed., 118. Hogs. Ohio, Indiana, and Kentucky. "In Cincinnati, where there are annually slaughtered upward of two hundred thousand hogs, probably not a tenth part are free from this disease. Whole droves, consisting of three or four hundred head, are sometimes thus affected. These animals, most of which are young, are raised in the prairie districts of Ohio, Indiana, and Kentucky."
- Osler, W., 1882. *American Journal of the Medical Sciences*, lxxiv. 475-480. Montreal, Can. "In casual visits to butcher stalls and to the shambles I have obtained six or eight large echinococci."
- Osler, W., 1882. *American Journal of the Medical Sciences*, lxxiv. 475-480. Cat. Montreal, Can. Liver of a cat with two large cysts.
- Dean, 1882. *American Journal of the Medical Sciences*, lxxiv. 475-480. Hogs. St. Louis, Mo. "Considerable proportion of the hogs slaughtered at St. Louis are infected."
- Osler and Clement, 1883. *Canada Medical and Surgical Journal*, xi. 325-326. Hogs. Montreal, Canada. "In the 1087 hogs examined, echinococci were found in the livers of 31, or 1 in 33.4."
- Day.¹ Bureau of Animal Industry Records, Washington (unpublished). Pigs. St. Louis, Mo.
- Moore.² Bureau of Animal Industry Records, Washington (unpublished). Washington, D. C. Hydatids, 2 cases.
- Wheeler.³ Bureau of Animal Industry Records, Washington (unpublished). Hogs. New Orleans, La. Livers of 117 out of 2000.
- Buckingham.⁴ Bureau of Animal Industry Records, Washington (unpublished). Camel. Washington, D. C. Liver.
- Welch.⁵ Hogs. Maryland. Hydatids, 3 cases.
- Stiles, C. W., 1898. Bureau of Animal Industry, Washington, D. C. Cattle, hogs, and camel. District of Columbia, Missouri, and Nebraska. "I have seen cases of hydatids in this country in cattle, hogs, the camel, and man, but as yet have seen no cases in sheep."
- Stiles, C. W., 1900. Private communication. Hogs, Kansas City, Mo., and Ottumna, Iowa; Cattle, Chicago. "About 1 per cent. of the hogs I examined at Kansas City some five years ago contained echinococci. I have also found it in cattle at the Chicago abattoirs, and in hogs at the Ottumna (Iowa) abattoir, besides the cases published in Bull. 19 and by Sommer."
- Salmon, D. E., 1901. *Journal of the American Medical Association*, xxxvi. 867-871. Sheep. United States. Hydatids ("echinococci") found in 209 out of 11,110,776 slaughtered sheep inspected by the United States meat inspection service during the two years ending June 30, 1899.
- Salmon, D. E., 1901. *Journal of the American Medical Association*, xxxvi. 867-871. Cattle. United States. Hydatids found in 6 out of 8,831,927 slaughtered cattle inspected by the United States meat inspection service during the two years ending June 30, 1899.
- Salmon, D. E., 1901. *Journal of the American Medical Association*, xxxvi. 867-871. Hogs. United States. Hydatids found in 1994 slaughtered hogs inspected by the United States meat inspection service during the two years ending June 30, 1899.

The United States meat-inspection service, however, gives only the aggregate for the whole country, and furnishes no facts on the special distribution of the disease in different parts of the country. It is quite likely that the disease is not evenly distributed throughout the country, but prevails in certain districts in greater proportion than in other districts, and that endemic foci of the disease exist, perhaps, here and there. Such a presumption is favored by the known facts of the distribution of the disease in Germany and other countries, and would help to explain the greater frequency of the disease in animals reported from certain parts of the country by various individual observers.

¹ Cited by Sommer, *New York Medical Journal*, 1895, lxii. 656-659.² *Ibid.*³ *Ibid.*⁴ *Ibid.*⁵ *Ibid.*

OCCURRENCE OF THE ADULT TAPEWORM (*TÆNIA ECHINOCOCCUS*) IN DOGS. It would, therefore, appear, *a priori*, that the adult *Tænia echinococcus*, from whose eggs when introduced into the gastro-intestinal tract are developed the larval hydatid cysts, must be an exceedingly rare tapeworm in the dogs of North America. And this is shown to be the case by the fact that this tapeworm in dogs has been discovered and confirmed by competent authority¹ in only a single instance in the United States, by Curtice² in Washington, D. C. Osler³ failed to find it in "some scores of dogs" examined in Canada from 1867 to 1882. Osler and Clement,⁴ in 1883, from Montreal, wrote: "We have never met with a specimen in numerous dissections." Sommer⁵ examined fifty dogs in Washington, D. C., in 1896, without finding it. Ward⁶ did not discover it among twenty dogs examined in Lincoln, Neb., in 1897. Stiles⁷ and Hassall,⁸ of the Bureau of Animal Industry, Washington, D. C., have never in their large experience seen it from any American source, except the single specimen discovered by Curtice. Sommer,⁹ in 1900, wrote: "In numerous dogs examined since 1896 at Blackwell's Island, New York, and in the State of New York I have never succeeded in finding the *Tænia echinococcus*." More extensive investigations than heretofore made, however, are required for determining the distribution and rate of occurrence of this parasite in dogs on this Continent. The minute size of the tapeworm makes its detection difficult, and throws the burden of investigating its occurrence on the few scientific zoölogists, veterinarians, and physicians who have given special attention to the much-neglected study of helminthology.

PROPHYLAXIS. Echinococcus disease in man and animals is one and the same disease, derived from a common source, namely, the ingestion with food or drink of the eggs of the *Tænia echinococcus*—a tiny tapeworm inhabiting the intestinal tract of dogs and wolves. The dog or wolf represents the determinate host of the parasite, and man and various animals the intermediary host in which the larval or bladder stage of the parasite in its cycle of development is undergone. For human beings or animals to become infected with the larval stage or hydatid cysts, they must in some way receive the eggs of the adult tapeworm of dogs into their stomachs, where the capsule or shell of

¹ Herf (Texas Medical Journal, 1894, vol. ix. pp. 613-616), who claims to have found the *Tænia echinococcus* in his district (San Antonio, Texas) in all dogs dissected, was probably dealing with some other variety of tapeworm, as already suggested by Sommer (loc. cit.).

² Curtice. Records of the Bureau of Animal Industry, Washington, D. C.

³ Osler. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1882, vol. lxxxiv. p. 475.

⁴ Osler and Clement. Canada Medical and Surgical Journal, 1883, vol. xi. p. 825.

⁵ Sommer. Veterinary Magazine, August, 1896.

⁶ Ward. Report of Nebraska State Board of Agriculture for 1896, p. 173.

⁷ Stiles. Personal communication.

⁸ Hassall. Bull. No. 19, United States Department of Agriculture, Bureau of Animal Industry, Washington, D. C., 1898.

⁹ Sommer. Personal communication.

the egg is digested and the embryo is released and enabled to penetrate the wall of the stomach or intestine, and be carried to its destination in the liver or elsewhere in the body, where it undergoes its metamorphosis into a bladder or hydatid cyst. For dogs to become infected with the adult tapeworm it is necessary that they ingest the live scolices contained in hydatid cysts; in other words, that they eat the cysts or cyst contents of animals infected with the disease. The full cycle of development in the life-history of the parasite is thus established.

This leads us to a consideration of the means of controlling the spread of the disease, which can be effected in two ways, viz., first, by carefully destroying, by burning, the larval cysts in slaughtered animals and thus preventing the infection of dogs; and, second, by guarding ourselves against infection from dogs by "recalling that the dog is not a human being and should not be treated as one. Too intimate association with dogs is sure to breed the disease in man." (Stiles.) Stray and ownerless dogs should be killed. But, though we can in a large measure protect ourselves against infection from dogs, we cannot equally guard our domesticated animals against such infection, except by first protecting our dogs against infection from diseased cattle, sheep, and swine. And this is the *key to the whole problem*. We can *exterminate the disease by enforcing proper sanitary regulations at the slaughter-houses*. Compel the destruction by burning of hydatid cysts found in slaughtered animals, and the infection of dogs and the subsequent infection of man or animals from dogs are prevented, except by remote possibilities hardly worthy of consideration. The practice of throwing out the offal of slaughtered animals should be interdicted by law, and dogs should be prohibited from entering the premises of slaughter-houses. The worst offender against these plain rules of public hygiene is the *country slaughter-house*, and against it our first and chief efforts for reform must be directed, as already insisted upon by Stiles.¹

"Hydatid disease is at present comparatively rare in this country, and now is the time to attack it. By proper precautions at the abattoirs and slaughter-houses this dangerous parasite can be totally eradicated from the country. If these precautions are not carried out it will be only a question of time when this country will take its place with Germany and Australia in respect to the number of human lives sacrificed to a disease which has not yet gained much ground with us and can now be easily controlled."²

COMMERCIAL CONSIDERATIONS. Finally, it may not be amiss to mention the possible commercial importance that this disease in our

¹ Stiles, C. W. The Country Slaughter-house a Factor in the Spread of Disease. Year Book of the Department of Agriculture for 1896, pp. 155-166.

² Stiles, C. W. Bull. No. 19, United States Department of Agriculture, Bureau of Animal Industry, Washington, D. C., 1898, p. 4.

live-stock may in the future assume if it is now neglected and allowed to gain a permanent place on this Continent. In view of our past experiences in the matter of international commercial jealousies and reprisals against American cattle by certain European nations, under the pretense of sanitary safeguards, the United States Government may well give serious heed to the possibility of furnishing to countries commercially hostile to us a plausible pretext for the exclusion of American animals, if by its present negligence it allows hydatid disease to become endemic and widespread in this country, as it is in Germany, Australia, the Argentine Republic, and many other parts of the civilized world.

In conclusion, we wish to acknowledge our indebtedness for notes of unpublished cases to various gentlemen mentioned in the summary of cases, and, in particular, to Dr. H. H. Chown, of Winnipeg, for his private notes of a large number of cases in Icelanders, and to Dr. Frederick A. Packard, of Philadelphia, for his generosity in placing at our disposal his references to cases which he had been systematically collecting from the literature with a view to ultimate publication.

RECENT VIEWS OF THE ORIGIN AND NATURE OF HERPES ZOSTER.

BY ARTHUR VAN HARLINGEN, M.D.,
OF PHILADELPHIA.

SOME years ago my friend Dr. Curtin called my attention to some cases observed by him, which seemed to point to the occurrence of herpes zoster on the pleura, the peritoneum, and in the articulations. He asked me whether I knew of any similar cases. I had not had any personal experience, but determined to examine the literature of herpes zoster with this point in view. It is only recently that I have had the opportunity of doing this, but, although references to the subject are scanty, quite a number of cases have been published which go to confirm Dr. Curtin's observations.

At the same time I have found so much of interest in scattered monographs of recent date that I have thought well to give some account of the views generally prevalent among dermatologists and neurologists regarding the nature of herpes zoster.¹ In order to do this satisfactorily, it seems desirable to give a hasty sketch of what is known regarding the pathology of the disease and of the various theories as to its origin.

¹ Dr. Curtin's views are stated in several monographs. "Is Herpes Zoster a Cause of Pleurisy and Peritonitis?" *Sanitarian*, December, 1890, and in other publications. Some valuable observations will be found in his latest contribution to the subject, to appear in the next number of the *JOURNAL*.

Barth, in an account of herpes zoster, or, as I shall call it for convenience, zoster, says that Parrot, in 1857, advanced the theory of zoster as a secondary disease, subordinate to a neuralgia, and Charcot, in 1859, called attention to a case, the first recorded, where zoster followed an injury. In 1861 Bärensprung, in his classical paper, developed the idea of the nervous origin of zoster, and attributed it to a disease of the ganglionic system, an irritation of the spinal ganglia, or of the Gasserian ganglia, while admitting that peripheral irritation of a nerve might result in a limited outbreak of the eruption.

In 1864 Mitchell, Morehouse, and Keen showed that irritation and not section of a nerve was the cause of zoster and similar eruptions. Later it was shown that compression by tumors, etc., might result in zoster on the distribution of the implicated nerve.

In 1872 Charcot reported a case of double zoster of the trunk following acute myelitis of the anterior cornua, and later reported cases of zoster occurring in connection with locomotor ataxia.

The mode of action of the morbid influence through the nerve on the skin was early made the subject of speculation, and several theories have been brought forward. What was known as the *vasomotor theory* assumed that the contraction, expansion, or paralysis of bloodvessels, and the consequent increase or decrease in the amount of nourishment brought to the cells, might lead to nutritive changes. This failed of experimental proof.

Samuel suggested a theory of *trophic nerve fibres* which was ingenious, but the existence of such fibres never having been proved, the theory fell to the ground.

The theory of the *trophic action of sensory nerves* attributes to the ordinary sensory and motor nerve fibres those functions for which Samuel had provided a special group of nerves. The nervous system acts on the nutrition of the anatomical elements by maintaining their functional activity. Trophic influence is thus transmitted to the skin and mucous membranes by the sensory nerves. Any alteration in the sensory fibres would result in the enfeeblement of the trophic influence of spinal and Gasserian ganglia upon these fibres and probably through their mediation on certain elements of the skin itself.

If this is the case we must admit that the weakening of this trophic influence would create a state of least resistance, or of greater vulnerability in those parts of the skin connected with the peripheral extremities of the affected sensory nerves.

Under these conditions, an irritant local cause, such as pressure, rubbing of the skin by clothing, or even contact of the air, might determine the appearance of the herpetic eruption, and, as would appear from recent observations, a microbic invasion could also occur.

Although this theory is attractive, and, if well founded, would

explain the pathology of zoster better than any yet brought forward, it has not as yet been proved, and remains a theory like the others.

The simplest and most plausible theory of the production of zoster, and one, indeed, which must account for a certain number of cases, is that of *propagated neuritis*. This supposes that zoster is simply the result of an inflammatory irritation transmitted by continuity of tissue from a nerve twig, or branch, to the cutaneous elements among which it is distributed.

I may say here in passing, that these theories take for granted that all zosteriform eruptions are of the same nature, and that each belongs to a specific affection: herpes zoster. In the present state of our knowledge, however, this is by no means universally admitted. We may have a specific herpes zoster, as we believe there is a specific pemphigus, while at the same time we may have various zosteroid eruptions, as we have various bullar or pemphigoid eruptions, which may arise from various causes.

Such, at least, is the generally prevalent belief among the dermatologists of to-day.

At the time Barth wrote, discussions upon the etiology and pathology of zoster seemed premature. Zoster was to be recognized as the cutaneous expression of a nervous disturbance, irritative in character, affecting, sometimes the peripheral sensory nerves, and sometimes the spinal and cranial ganglia. Sometimes resulting from an injury, as contusion or compression of a nerve, sometimes as the result of cold, or under the influence of a general perturbation of the system.

The nerve disturbance might at times be so slight as not to affect perceptibly the anatomical integrity or the functions of the nerve fibres, and might show itself only by the cutaneous eruption.

At other times, however, and especially in the old, this slight initial disturbance might prelude a destructive neuritis, which might give rise to anæsthesia, rebellious neuralgias, and various other disorders of sensation so common as the result of severe zoster.

Although, most commonly, the morbid process produces only slight skin lesions, the irritation might extend to other organs innervated by the same nerves, and might determine grave and irreparable disorders. Ulceration of the cornea, as observed by Hutchinson and Hybord many years ago, and which is an occasional accompaniment of ophthalmic zoster, was one of the first of these complications to attract attention. Joffroy pointed out that the neuritis of zoster may be propagated to neighboring nerves, and might cause atrophy of certain muscles. Such was the position of the zoster question as stated by Barth in his article, to which reference has been made. He hazarded, however, the suggestion that visceral organs might be attacked under the influence of the same cause as that producing the zoster in a given case, and referred

to the opinion of Fernet, that certain pneumonias may be the expression of a sort of zoster of the pneumogastric nerve.

This prophesy, put forth by Barth twenty years ago, has been verified by subsequent clinical observations. A large number of cases of visceral disorders of various kinds in connection with zoster have been reported of late years. Pitres and Vaillard, Leroux, Potain, Huchard, Chandeaux, Mongour and Michel, Cantrell, Curtin, and others have shown the concurrence of zoster with tuberculosis, pleurisy, peritonitis, etc. Palm reported the case of an infant with bilateral zoster of the third branch of the trigeminal, which developed on the third day of a follicular angina. Hervonet reported a case of cervicobrachial zoster, accompanied by paresis of the arm, and, in addition, incomplete facial paralysis and ptosis. The various symptoms occurred simultaneously and, as will be observed, in different nerve tracts.

While these and similar cases continued to appear, other observations were published, going to show a close connection between zoster and various infective diseases. Gaucher and Barbe wrote on zoniform syphilides, Julien on zona and syphilis, and Spillman and Etienne published observations of a similar character. In the cases reported by the first of these writers, the distribution, as I understand, rather than the character of the lesions, lead to its denomination of zoniform, but the point of interest lies in the suggestion of the authors that the lesions had been produced, in all probability, under the influence of the pathogenic agent of syphilis or of its toxin upon the spinal axis.

Cancer, otherwise than through the direct influence of pressure from the new growth, has been suggested as a cause of zoster. Carrière, in a case of femoro-cutaneous zoster following cancer of the uterus, found parenchymatous neuritis of the nerve involved. There was no cancerous infiltration of the nerve, and Carrière attributed the neuritis to the local and direct action of cancer toxins. One of Dr. Curtin's cases was also connected with cancer of the oesophagus.

The occurrence of zoster in connection with coal-gas poisoning and as a result of the administration of arsenic presents certain problems which have not been studied with sufficient care to enable us to express any opinion. The fact that zoster or zosteriform eruptions may occur under such circumstances is established, but more cannot be said.

Wasilewsky, in his monograph on zoster as an infectious disease, cites reports of epidemics of the affection. He regards the question of immunity as settled, but with the numerous cases of recurrent zoster reported of late years, it is difficult to agree with him on this question, unless, indeed, we come to recognize the recurrent cases as zosteroid in character, rather than as true zoster.

In typical cases, prodromata, as gastric disturbance, and fever appearing several days before the eruption, with a temperature often reaching

104° F., and ceasing with the outbreak of the eruption, point, in Wasilewsky's opinion, to an infectious origin for the disease, and this view is supported by the observations of Head and Campbell, which I shall cite later.

A point in favor of the infectious character of zoster is the occurrence of enlarged lymphatic glands. Grindon and Hay have shown that true zoster is always preceded by adenopathy in the neighborhood of the eruption, or even by generalized involvement of the lymphatic glands. Hay discovered cocci in the glands.

The infectious nature of zoster having been so strongly indicated by the observations of numerous writers, it is not surprising that facts should now begin to appear in favor of its contagious character. Ferré has reported some very interesting cases in this connection. He thinks that in some cases zoster may be the consequence of a meningeal or spinal affection, and in the epidemic form may be an indication (*symptôme révélateur*) of an attenuated form of cerebro-spinal meningitis. As numerous instances of the recurrence of zoster have been reported of late years, these must be taken into consideration before the infective theory is generally accepted. The view that there are several varieties of zoster, or that there is one specific zoster, an infective disease, and, therefore, not recurrent, and various zosteroid eruptions due to different causes, is one that seems to have gained considerable adherence of late years, and in the light of recent observations is certainly a plausible explanation of numerous otherwise anomalous phenomena.

The anatomy of the lesions of zoster themselves has been made the subject of investigation in the light of more recent knowledge and with the aid of modern methods. Peffer maintains that the true cause of zoster is in all cases a protozoid, which he has found in the affected skin and has described. Hartzel, who has made similar investigations, says that these protozooids are not present in ordinary inflammations of the skin, but are peculiar to zoster, variola, vaccinia, and varicella. Gilchrist, after an elaborate investigation of the subject, concludes, however, that the so-called protozooids are really the nuclei of epithelial cells.

Up to within the past year or two, Bärensprung's observations and conclusions have never been questioned, and zoster has everywhere been received as an affection closely connected with the sensory nerves, and following in its occurrence the distribution of these nerves in the skin.

It cannot have failed, however, to strike the careful observer that the eruption of zoster does not invariably follow the line of distribution of the nerves, this circumstance being particularly noticeable in thoracic zoster. Brissaud, a recent writer, goes so far as to assert that in a large number of cases the concordance of the eruption of zoster with a nerve path is a fiction.

Having made the same observation with regard to other diseases besides zoster, Brissaud sought for an explanation and found it in the theory of "metamerism."

Concluding from our knowledge of embryology that the spinal cord is composed of a series of segments superposed and relatively independent (metamers), Brissaud attempted to prove that the eruption of zoster should appear in the territory of a region innervated by a medullary metamer. This territory of innervation, however, is sometimes entirely different from that which corresponds simply to the course and distribution of the sensory nerve fibres.

Another and quite different theory of the relation of the zoster eruption to the nervous system has been proposed by Abadie. He maintains that the skin affection does not result from an alteration of the peripheral sensitive nerve fibres, nor from a medullary lesion, but from a pathological condition of the arteries and of the vasomotor nerves which regulate their dilatation in the region where the eruption is situated.

In a general way the sensory nerves accompany the arteries, at least during a portion of their course, and, therefore, the zoster eruption, which really occupies, the territory of vascular distribution, has heretofore been supposed to occupy the nerve territory. As, however, in certain localities, these two territories are neither identical nor superposable, apparent anomalies supervene, anomalies which Brissaud tries to explain by his "metameric" theory above mentioned. This hypothesis is not necessary, however, to explain the topographical anomalies of the eruption, as the cutaneous lesions occur along the bloodvessels and not along the nerve filaments.

Taking ophthalmic zoster as an example, Abadie asks how it is, if zoster is due to inflammation of the trigeminal nerve, that it only occurs (save in the very rarest cases) in the first or ophthalmic branch. As a fact, maintains Abadie, the eruption extends over the area supplied by the *supra-orbital*, *frontal*, and sometimes the *nasal* arteries.

The lesions of zoster, says Abadie, are due to a trophic influence proceeding from the *sympathetic nerve fibres*, which, in the case of ophthalmic zoster, follow the course of the arteries just mentioned. The pain and other nervous symptoms, which may or may not accompany the eruptions, are due to some involvement of the sensory nerves, which may be implicated in one case and may not in another.

This theory is supported by the form of the eruption in thoracic zoster. This does not occur above the line of the third intercostal space. The arteries supplying the three upper intercostal regions, instead of proceeding from the aorta, are derived from the subclavian artery, and consequently their vascular origin and that of the vasomotors which accompany them are different from the others which come directly from the aorta.

This theory of Abadie's makes clear, so he says, the apparent anomalies of the eruption which are inexplicable otherwise, but it takes away from the sensitive nerves of relation a part which does not belong to them and assigns it to the great sympathetic. It proves that the latter really commands and regulates nutrition in all the regions to which it is distributed, whether visceral or cutaneous.

We may thus understand, also, that certain forms of ophthalmic zoster may present particularly grave cerebral complications, and may be accompanied by hemiplegia, as Brissaud has shown, in some cases. The mechanism of these lesions has been difficult to explain by the nerve theory, but it is easily understood by accepting the excessive vasomotor dilatation of certain cerebral arterioles which may provoke rupture in their territories of irrigation.

Finally, Abadie says that quinine is a specific in ophthalmic zoster. Now, quinine is essentially a vasoconstrictor medicine acting on the sympathetic and producing a constriction of the arterial vessels.

These views of Abadie have not been generally accepted, although there is certainly something in them.

But, as Hollochau says, the character of the concomitant and consecutive pains appear necessarily to imply a participation of the sensory nerves, and also the localizations can only be explained by a metameric action.

The blood examinations which have been made in zoster are worthy of mention, although their significance is not apparent. Like the microscopical examinations of the lesions, they are materials of possible future structures, but have not yet been fitted into their place.

Leredde says the white globules may fall to 3500. The polynuclears are diminished to 45 per cent. on an average; they may fall to 37 per cent. On the tenth to the twelfth day an eosinophilia of 6 to 8 per cent. has been noted.

Head and Campbell, in a recent and extremely elaborate paper, give a large number of facts regarding the origin and distribution of zoster. These investigators have been able to map out the affected areas in zoster, and have subsequently examined the spinal cord and nerves implicated. The cases in which they have been able to make post-mortem examinations number more than the sum total of those previously recorded by all other observers.

Head and Campbell find inflammatory and destructive changes in the posterior ganglia of the cord and in the Gasserian ganglion, and degeneration in the nerves leading to the skin. In mild cases the effects of these changes gradually pass away, but in severe cases the nerve fibres that have degenerated are replaced by fibrous tissue, and whole bundles of the nerve may be sclerosed.

Degeneration in the spinal cord occurs secondary to the herpetic lesion of the ganglion.

It may be noted, referring to the brain symptoms in connection with ophthalmic zoster, that the third division of the trigeminal is connected with cells in the Gasserian ganglion which send fibres centralward to lie in the so-called ascending or spinal roots.

Head and Campbell conclude that some agent, the nature of which we are ignorant, chooses the substance of the posterior root ganglion for its selected activity, producing profound inflammatory changes. These destructive changes in the ganglion give rise to irritation of its nerve elements, and to this irritation is due the eruption on the skin.

Acute inflammation of a posterior root ganglion, due to its implication in some local inflammatory process, can apparently produce an herpetic eruption indistinguishable from an eruption which arises as a manifestation of the acute specific disease (as, for instance, a case of zoster reported by Head and Campbell, where lymphosarcoma invaded the posterior root ganglion directly).

Zoster may appear in the course of gross organic disease of the spinal cord and roots. In some such cases Head thinks the original disease simply predisposes to an attack by the specific agent supposed to be the chief etiological factor in the production of the zoster.

Head has traced nerve twigs up to the portion of skin excised for examination, and found they showed marked signs of degeneration. He did not succeed in demonstrating degenerated fibres in the actual portion of skin examined.

A typical attack of zoster arises without any obvious peripheral or cerebral cause, and, as has been said, must be considered an acute specific disease of the nervous system, for it starts with a prodromal period of varying length, during which the temperature is raised. During this period the patient feels ill and has more or less pain.

If a child, he may be supposed to be sickening from one of the ordinary specific diseases. Then the rash appears, the febrile period lasting three to five days.

Head's experience with recurrent zoster is worth noting. He met with but four cases of relapse in four hundred of zoster.

In connection with the occurrence of zoster as a specific infectious disease we must consider the large number of zoster-like eruptions evidently due to various causes, as injuries, poisoning by arsenic, coal gas, etc. All these must be ruled out from the division of true zoster.

On the other hand, the occurrence of zoster as a specific fever running a regular course and characterized by a typical eruption, prepares the mind for the reception of Curtin's theory as to the involvement of various membranes and viscera.

If the cause of zoster lies in a disorganization of certain nerve cells or nerve fibres by the toxin of a specific germ, or by an unknown

agent of any kind, there is some reason for admitting the possibility that the same agent may give rise to inflammatory symptoms in the pleura or in the articular cavities.

As Osler and others have described the various visceral complications of erythema multiforme, so we may regard the cases observed by Curtin as those of visceral complications of herpes zoster, and I have no doubt that when the attention of observers has been drawn to this aspect of the case we shall find numerous cases of such complications reported.

CONCLUSIONS. From what has preceded, the following conclusions may be drawn:

1. Under the designation zoster or herpes zoster is to be understood a specific infectious and possibly contagious exanthem, characterized in its invasion by lassitude, general malaise, chills, increased temperature, and more or less digestive disturbance.

Following this, in most cases, neuralgic pains develop along certain nerve-paths or metameric areas, together with the development of enlarged lymphatic glands.

After a period of several days, more or less, during which the symptoms mentioned, or some of them, have manifested themselves, the characteristic exanthem shows itself, and runs through a fixed cycle of development, acme, and decrudescence. The general symptoms, particularly the neuralgic pains, may continue during this period, or, in many cases, may diminish, usually disappearing with the eruption. In other instances, and particularly in older persons, the neuralgia may form a prolonged succedaneum to the regular course of the disease.

In a certain number of cases, probably, however, rare, various visceral complications may accompany the affection. Such are paralysis of sensory or motor nerves, inflammations of the pleura, peritoneum, articulations, or viscera.

2. The infection attacks chiefly the posterior (sensory) ganglia of the cord and the Gasserian ganglion. From thence inflammation and degeneration may extend along the nerve trunks and fibres.

No other lesions have as yet been discovered post-mortem, although it is probable that further observation will result in tracing the disease in the various membranes and viscera when its presence has been clinically noted.

The numerous examinations of the skin lesions and such blood examinations as have been made have not as yet thrown any light upon the nature of the disease.

3. Zosteroid eruptions are not infrequently observed in cases of poisoning from coal gas, after the ingestion of arsenic, following injuries of the nerves, as a result of moral shock, as grief, or in hysteria, and probably under other conditions. These, however, are to be distinguished from the true herpes zoster as defined above.

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SOME EXPERIMENTS ON THE INTERMEDIARY CIRCULATION
 OF THE BILE ACIDS: A CONTRIBUTION TO OUR
 KNOWLEDGE OF ICTERUS.

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A LARGE proportion of the bile-acid salts that are poured into the intestine from the liver are reabsorbed. This is proved by the following four series of experiments:

1. According to different authors (Bidder and Smith, and Leyden), a dog weighing 8 kg. excretes about 4 g. of bile acids in the twenty-four hours; of this amount only 0.5 to 0.7 g. will be found in the feces (Hoppe-Seyler) and unweighable traces in the urine (Naunyn, Vogel, Höne, and Dragendorff). Leyden injected 1½ g. of one of the bile-acid salts (sodium glycocholate) into the blood of an animal and found only 0.227 g. in the twenty-four hours' urine. As the bile acids are very resistant to putrefaction in the urine and to digestive destruction in the stomach and intestine (Leyden), it cannot be assumed that they are destroyed in the bladder or the digestive tract.

2. Tappeiner placed two ligatures around a loop of intestine without disturbing its connection with its mesentery; he then placed bile-acid salts into the jejunum and the ileum at different times, and found that

the salts were completely absorbed (the loop of jejunum absorbing only the glycocholate, the ileum both the glycocholate and the taurocholate).

3. Neisser showed that nearly all the bile-acid salts that are administered by the mouth can be recovered a short time after from the bile flowing from a biliary fistula.

4. There are two principal bile acids : the one contains sulphur (taurocholic) the other does not (glycocholic). Dogs excrete the former almost exclusively, nearly 99 per cent. of the bile acids found in their bile being of the sulphur-containing variety. If, now, dogs are fed with large quantities of the sulphur-free variety it will be found that their bile (removed through a fistula) contains a large proportion of the latter, as much as from 20 to 30 per cent., showing conclusively that at least that much of the sulphur-free salt had been absorbed from the intestine to be re-excreted in the bile (Weiss). (Prevost and Binet.)

Whereas the fact, therefore, is established that the bile acids are in great part absorbed from the intestine to be re-excreted through the common duct of the liver, the path that they follow in this intermediary circulation and the function they perform *en route* are obscure.

Tappeiner succeeded in finding appreciable quantities of the bile acids in the lymph of the thoracic duct of a dog, but no one so far has found them in the blood under normal conditions. As the bile acids contained in the thoracic lymph must necessarily be poured into the systemic blood, and as they seem to disappear there only to reappear in the bile, we are confronted with the interesting problem, *What becomes of the bile acids while they are circulating from the thoracic duct to the liver?*

The following two considerations led us to assume that possibly the bile acids were present in the blood in a form that rendered them undiscoverable by known methods :

1. When isolating the bile acids from the bile it is necessary to remove the mucin by precipitation. This precipitate always contains a large proportion of the bile acids. The mucin and the bile acids are combined so firmly that it is impossible to separate the two by repeated washings or any of the other manipulations commonly employed for the separation of bile acids from organic substances ; and still this combination is purely a mechanical one, for by a very long and very tedious process of dialysis it is possible to finally separate the two.

2. Bile acids are very toxic. When they are poured into the blood from the thoracic duct it is probable that they suffer the fate of other toxic substances, and are taken up by the phagocytes, which carry them through the blood-stream to the liver. Here, like many other poisons, they are liberated, arrested, and finally excreted in the bile.

These two possibilities must, therefore, be taken into consideration. Either the bile acids form compounds with some of the proteids of the serum similar to those formed with the proteids of the bile (they may

exist during life or be formed, as in the case of the bile, when the proteids are precipitated) or they enter the body of the leucocytes. In either case they would evade detection by ordinary methods.

The task set is, therefore, to isolate the bile acids from either hypothetical combination by methods other than those ordinarily employed. Here two possible manipulations suggest themselves:

1. To submit large quantities of blood to artificial digestion. This method would be based on the fact that the bile acids are more or less resistant to the action of the digestive ferments. The proteid compound would be digested away, leaving the bile acids free in the solution.

2. To oversaturate large quantities of blood with many volumes of absolute alcohol. This method would be based on the fact that the bile acids are readily soluble in alcohol, whereas all proteids (whether in or of the serum or the leucocytes) are coagulated, precipitated, and rendered insoluble by contact with alcohol.

EXPERIMENT I. Fifty c.c. of dog's blood were slightly acidulated with acetic acid and boiled. The voluminous coagulate of albumin was filtered off; the clear filtrate was examined for bile acids and found to contain none; the coagulate was divided into two equal parts, and one portion was placed into a sufficient quantity of artificial gastric juice, the other into a sufficient quantity of pancreatic juice; both mixtures were allowed to remain in the incubator at body temperature for forty-eight hours, and the flasks frequently agitated during this time. Each of the two fluids was then examined for bile acids; the result was negative.

EXPERIMENT II. Fifty c.c. of blood were allowed to flow directly from the vein of a dog into a sterile flask containing 300 c.c. of absolute alcohol warmed to 40° C. The mixture was rapidly shaken for about a minute, and the thick precipitate that formed filtered off; this coagulate was repeatedly washed with alcohol, expressed, and finally dried in the vacuum. The dry, amorphous mass was powdered and extracted with boiling water; this extract contained no bile acids. The alcoholic filtrate and the alcoholic washings were united and the bulk of the alcohol distilled off until the volume of the fluid was reduced to about 50 c.c. This fluid contained bile acids.

The last experiment shows that bile acids are present in the blood of a normal dog. Similar experiments repeated with human blood obtained from volunteers by venesection, and, in one instance, from the operating-table, showed that they are also present in human blood in normal subjects.

It was necessary to determine, however, whether the bile acids are bound to some proteid substance of the serum or whether they are carried by the leucocytes. Serum and blood-corpuscles were examined separately by the alcohol method, and it was found that the bile acids were present in the corpuscles and not in the serum. In order to determine whether the white or the red cells contained these bile acids the vein of a cow was ligated in two places, excised, and suspended for twenty-four hours in the ice-chest; a separation of the corpuscles by gravity

can be brought about in this way. If an aspirating needle is inserted into the lowest portion of this vein only red blood-corpuscles will be aspirated. These were examined by the alcohol method, but no bile acids were found. We did not succeed very well in aspirating only white cells from the higher layers, and the quantities obtained were too small to justify any conclusions; the *absence* of bile acids in the red corpuscles and the serum permits the conclusion that the bile acids are, in fact, normally carried in the leucocytes. This question cannot be definitely decided until a method is discovered of procuring large quantities of leucocytes from the blood.

If the bile acids were an excretory product pure and simple, with no further function to perform, they would not be poured into the uppermost portion of the intestine, but would probably enter the lower portion of the bowel. As a matter of fact, it is known that the bile acids perform a variety of important functions in the intestine that are fairly well understood; these do not concern us here. We are occupied with the question, *What function do they perform in the systemic blood during their intermediary circulation from the intestine back to the liver?*

The following toxic effects of the bile acids are known:

1. They have a powerful cytolytic action. Injected even in small doses they produce a wide-spread disintegration of the red blood-corpuscles, with a liberation of their hæmoglobin; brought in contact with other cells of the body they cause their disintegration.

2. They have a distinct cholagogue action—are, in fact, the only substances known to possess this power, and actually cause an increased flow of bile.

3. They aid coagulation in small doses (1 : 500).

4. They stop coagulation in large doses (1 : 250 and over).

5. They slow the heart-beat by a direct action on the heart muscle and the cardiac ganglia.

6. They act as vasodilators in very small doses.

7. They act as vasoconstrictors in large doses.

8. They reduce motor and sensory irritability.

9. They act on the higher cerebral centres, causing coma, stupor, and death from "gentle enslumbering."

Of all these possible effects only those can normally take place in the body that can be exercised in doses as small as those normally found in the blood, namely, those described under 1, 2, 3, and 6.

We are justified, therefore, in assuming that, aside from their intra-intestinal functions, the bile acids are concerned in the normal destruction of cells, chiefly red blood-corpuscles; that they exercise an influence on the flow of bile; that they aid coagulation and play a rôle in vasodilatation; in pathological states, when large quantities of the bile acids enter the blood, their other powers are developed and become clinically manifest.

The finer mechanism of these various activities has so far not been made the subject of experimentation.

CLINICAL EPICRITICISM. The discovery of the bile acids in normal blood has a definite and important bearing on the origin of icterus.

Notwithstanding the fact that under certain pathological conditions (hemorrhagic exudates and extravasations, subcutaneous injections of red blood-corpuscles or hæmoglobin, pyæmia without occlusion of the bile-ducts or other impediment to the flow of bile), bile-pigment is found in the tissues, enters the blood, stains the tissues diffusely, and is excreted in the urine, the existence of extrahepatic "hæmatogenous," or, better, "anhepatogenous" icterus is almost universally denied. It is claimed that every icterus is an icterus from absorption of bile from the liver; that blood-pigment is converted into bile-pigment in the liver alone, and that even in excessive hæmolysis or after injection of hæmoglobin, icterus (hæmato-hepatogenous) is due to the increased viscosity of the bile causing occlusion of bile-channels, stasis, and absorption, or to some injury of the hepatic cells that permits diapedesis of bile-constituents from the bile-channels through the liver cells to the blood-channels.

The chief argument adduced in favor of this view is the presence under these conditions of bile acids in the blood and urine, for, it is claimed, these are a specific liver product formed only in the liver and by the liver, and *never normally present in the blood*. Aside from the fact that a series of experiments that I have recently published show that bile acids are not a specific product of the liver cells, and make it highly probable that they can be and are normally, and to a greater degree pathologically, formed outside of the liver, the investigations reported above show that they are normally present in the blood; so that this argument and the conclusions that are based on it collapses.

If, *per contra*, the adherents of the theory of hæmatogenous icterus claim that every icterus in which bile acids are absent from the blood and urine is hæmatogenous ("Finden sich keine Gallensäuren—dann haben wir hematogenen Icterus vor uns," Leyden) they, too, occupy an extreme position, for we know that in some cases of the most pronounced obstructive icterus in which there is occlusion of ducts, stasis of bile, absorption of bile constituents, etc., ordinary methods may fail to reveal the presence of bile acids in the blood and urine. *They are, however, never absent from the blood.*

All arguments, therefore, that are based on their "presence" or "absence" from the blood are essentially fictitious and invalid.

From the bile-acid findings in any given case we are no longer justified in categorically asserting or denying the existence of pure hæmatogenous icterus, nor are we aided by these findings in formulating any definite diagnostic conclusions in regard to the participation of the liver or its ducts in any disease-process complicated by icterus.

REVIEWS.

A MANUAL OF SURGICAL TREATMENT. By W. WATSON CHEYNE, C.B., M.B., F.R.C.S., F.R.S., Professor of Surgery in King's College, London, and F. F. BURGHARD, M.D., M.S. (Lond.), F.R.C.S., Teacher of Practical Surgery in King's College, London. In seven volumes. Vo's. I. to V. Philadelphia and New York: Lea Brothers & Co.

THE original intention was to issue this book in six volumes, but the subject grew in the writing, and it became necessary to obtain enlarged space. The fifth volume of this valuable series is now before us, and is equal in merit to its predecessors. It deals with the head, face, jaws, lips, larynx, and trachea, and the intrinsic diseases of the nose, ear, and larynx, the latter subjects being discussed by H. Lambert Lack, M.D.

An extensive work upon surgical treatment was greatly needed, and the manual by Cheyne and Burghard fills a long-realized want. In the five volumes so far issued the authors have considered thoroughly, carefully, and sometimes even elaborately, the treatment of injuries and of surgical diseases. The constant effort has been to furnish clear descriptions and detailed information, and the result is singularly gratifying. Often when dealing with a difficult case—a case in which ordinary methods of treatment have proved futile—have we wished for a book like this, because the information upon treatment contained in the ordinary text-book is usually scanty and incomplete, and is sometimes so meagre as to be practically useless. The authors of this manual assume that the reader is familiar with the nature and diagnosis of the disease, and “refer to pathology and symptoms” only so far as is necessary to render “intelligible the principles on which treatment is based and the various stages of the disease to which each particular method is applicable.” They do not set forth all methods or even many methods of treating each condition, but prefer to describe the treatment which has been most successful in their hands.

In treating acute inflammation the authors do not particularly advocate general blood-letting except in diseases of the lungs and brain with enlargement of the right heart. They use cold only early in an inflammation, but fail to caution us against the intermittent use of cold. They point out dangers in the employment of Leiter's tubes, emphasize the fact that heat is used to treat inflammation after cold has been abandoned, and that heat is not applicable to a skin inflammation. Chronic inflammation is discussed in a separate chapter, and in this article the value of free incision is especially dwelt upon.

In the section upon Acute Abscess, Hilton's method is advocated for reaching deep-seated collections of pus in certain regions, and we are told that when Hilton's method is used a probe must be inserted before the forceps are withdrawn, so that the opening in the deep fascia will

not be lost. Particularly judicious is the direction to avoid violent squeezing of an abscess after opening, because squeezing may injure the abscess wall and lead to dissemination of the infection. Washing out an acute abscess after incising it is strongly condemned on the grounds that irrigation cannot satisfactorily disinfect, and may injure the abscess wall, lead to diffusion of the infection or the production of a weak spot in which micro organisms "which would otherwise die can spread."

In the section on Ulcers, skin grafting by the methods of Reverdin and Thiersch is carefully described, but the transplantation of pieces of the entire thickness of the skin is not discussed.

We are advised to cover the grafted area with strips of protective, one inch wide (the strips having been sterilized in 1 : 20 carbolic-acid solution and washed in boric-acid solution), and to apply over the protective, first, cyanide gauze wrung out of a solution of corrosive sublimate (1 : 4000), and then salicylic wool. In from five to seven days the dressing should be removed and a fresh dressing applied. At the end of another week dressing can be dispensed with.

In senile gangrene of the foot, Cheyne and Burghard believe that usually amputation through the knee-joint is the proper procedure.

In diabetic gangrene they advocate early amputation high up, and they do not believe that coma is more apt to arise if amputation is performed than if the gangrene is allowed to go on undisturbed.

The article upon Anæsthetics is most excellent and practical. The author is J. Fred. Silk. He doubts the value of morphine given before the administration of an anæsthetic, and believes the drug may be actually dangerous by masking the symptoms of overnarcosis.

In Silk's article the Subsection upon Special Cases is of decided practical use.

The article upon Local Anæsthesia is short and unsatisfactory, and does not do justice to a most valuable expedient.

In the section dealing with wounds the authors point out that fibrin ferment is a useful agent to check capillary oozing. They advocate the use, in certain regions, in order to avoid scars, of buried interrupted sutures passing through the derm but not the epiderm. Such sutures seem to us clumsy and troublesome of application when compared to Halsted's admirable subcuticular stitch.

In the article upon Nævi the clearest description we know of is given of the application of electrolysis.

In treating of aneurisms of large vessels we are told that the stay knot is reliable and satisfactory. The authors have evidently little faith in wiring aortic aneurisms, and prefer distal ligation to Macewen's method of needling.

The sections on the Skull and Brain are notably good, and we are glad to find that this book does not advocate the treatment of microcephalic idiocy by the ridiculous operation of linear craniotomy.

In cleft palate the authors adopt the modern view of Owen, that comparatively early operation is advisable. They do not delay operation after the third year.

In the section on Fractures no attempt at complete enumeration of methods is attempted, as the authors only speak of those which have proved useful in their hands. In this section it becomes evident that exploratory operations and operative fixation of fragments are coming

more and more into vogue. The operative treatment of fracture of the patella is warmly advocated.

The above is a very hasty and imperfect sketch of some of the views set forth in this useful and scientific book.

The *Manual of Surgical Treatment*, although it omits some valuable methods, is, in our opinion, one of the most practically useful of recent publications. We employ it frequently and warmly commend it. The scope of it will be best realized by noting the subjects treated in each volume.

Vol. I.: The treatment of general surgical diseases, including inflammation, suppuration, ulceration, gangrene, wounds and their complications, infective diseases and tumors; also an article on the administration of anesthetics, by Dr. Silk.

Vol. II.: Treatment of the surgical affections of the tissues, including the skin and subcutaneous tissues, the nails, the lymphatic vessels and glands, the fasciæ, buræ, muscles, tendons and tendon sheaths, nerves, veins, arteries, and deformities.

Vol. III.: Treatment of the surgical affections of the bones, and amputations.

Vol. IV.: Treatment of the surgical affections of the joints (including excisions) and the spine.

Vol. V.: Treatment of the surgical affections of the head, face, jaws, lips, larynx and trachea, and the intrinsic diseases of the nose, ear, and larynx.

These five volumes contain over 1800 pages and nearly 600 illustrations.

J. C. DA C.

PRACTICAL SURGERY: A WORK FOR THE GENERAL PRACTITIONER. By NICHOLAS SENN, M.D., Ph.D., LL.D., Professor of Surgery, Rush Medical College, Chicago. Handsome octavo volume of 1133 pages, with 650 illustrations, many in colors. Philadelphia and London: W. B. Saunders & Co., 1901.

This is Dr. Senn's latest and, in some respects, most elaborate and pretentious work. It does not pretend to cover the whole field of surgery, but is supposed to contain much or most of that which is most important and most practical. In the preface the announcement is made that its contents are devoted to those sections of surgery which are of especial interest to the general practitioner. Inasmuch, however, as a large part of the volume is given up to a discussion of topics such as gunshot wounds and others quite within the domain of military surgery, we do not see exactly how this claim has been substantiated. To be sure, injuries and acute surgical diseases do usually come first under the notice of the general practitioner, but this is not true of gunshot wounds.

The volume opens with a chapter on Emergency and Military Surgery. The chapter is, in large measure, a homily in which are dealt with the qualifications and duties of the military surgeon, with remarks upon military spirit, courage, personal habits, etc., all of which make interesting reading, although their insertion here is a matter of questionable taste.

In the chapter on Traumatic Shock the subject is exceedingly well handled, both from its theoretical and practical aspects.

The following chapter is on General Anæsthesia, in which are fully discussed the accidents which may happen during narcosis, the methods of their treatment, the merits of various anæsthetics, etc. Under the head of local anæsthesia, however, there is no mention of intraspinal cocaine injection, and lumbar puncture is but briefly alluded to much later in the volume. Surely the method deserves description in such a work as this, even though the author may be averse to its use. There is nothing in the book, however, which would indicate what his views are in regard to this subject of great recent interest. Neither is there any mention made of nitrous oxide gas, nor of its use in combination with oxygen, or as a preliminary to the administration of ether, nor is the combination of chloroform with oxygen gas mentioned. In this respect there is an important omission which should be remedied in the next edition. The subject of prophylactic hæmostasis is carefully considered, including the disagreeable consequences which may follow the employment of too tight constriction, such as paralysis, etc. Angiotripsy receives very scant mention. The treatment of hemorrhage leads abruptly into a discussion of sterilization of ligature materials, and then to the practical application of ligatures to the principal vessels, to illustrate which a few of Zuckerkandl's colored plates have been introduced.

The sixth chapter is devoted to the subject of Wounds and Wound Infection, which leads the author without break into the consideration of the proper operating-room, and then of hand disinfection, etc. While insisting on the necessity of careful hand disinfection, there is no allusion to perhaps the most admirable means for accomplishing this in the use of Schleich's marble-dust soap, with its most efficient qualities. Again, in the list of various antiseptic materials which may be used we find no allusion to the silver salts, which are now coming into such general use. Neither is intravenous injection of Crêdê's soluble silver mentioned in the treatment of septicæmia.

Abruptly following the chapter on gunshot wounds, and sandwiched in between it and that on fractures, is a chapter on Rupture of the Urethra, with which no fault can be found except that in this particular location it seems very much out of place.

Then follow about 250 pages devoted to the general subject of fractures, which is dealt with in the most thorough manner, and which will constitute most interesting reading for everyone, surgeon or general practitioner. Almost all possible contingencies have been taken up and dealt with, and these pages bristle with practical hints of great value. It must be said that some of the methods of treatment described are now obsolete and of historical interest only, and yet the general practitioner is tempted always to exclude the historical side of these subjects and to think only of what can be done now and not of what was formerly done for the relief of such surgical lesions. Delayed union and false joint are fully considered, and there is displayed throughout a very wide range of knowledge of and familiarity with the subject in all its aspects. We are glad to see justice done to the views of Moore, of Rochester, N. Y., whose researches on fractures and dislocations are not widely enough recognized, and who is a pioneer in many of these investigations. Skiagrams are very well used where they will prove of service, and add great value to the text. Fractures of the skull are not

separated from other fractures, as is usual in discussing regional surgery, but are brought in with the various inseparable considerations concerning intracranial injuries, which, however, are only incidentally discussed. Particularly good is the discussion on bone suture and the union of fragments in compound and comminuted fractures. The writer is not familiar with any other text-book in which so good an idea can be obtained of this important matter.

Chapter XII. deals with Dislocations, and is very complete so far as it goes. The author, however, apologizes for what seems a very great inconsistency in that dislocations of the shoulder-joint are discussed at great length and with great care, and yet no allusion is made to dislocations of the hip-joint. In spite of Dr. Senn's statement that he has seen so many fractures of the neck of the femur mistaken for dislocation that he felt called upon to emphasize description of this particular kind of dislocation, this can hardly be considered a sufficient reason for omitting all allusion to the hip dislocations, which are almost equally important and often more difficult of management. It would seem as if lack of time or haste in preparation were the real cause for this omission rather than other considerations.

Exploratory puncture, subcutaneous medication, paracentesis, drainage of joints, and aseptic catheterization follow the chapter on Dislocation, without any particular logical order, although the pages are full of practical discussion and direction; then come emergency operations on the air-passages and description of empyema and its treatment. The author is not quite correct when he says that empyema is a term used to designate the presence of pus in the pleural cavity. The term properly means the presence of pus in any pre-existing cavity, and he should have added that, by common consent, when no particular cavity is specified the pleural cavity is understood. He defines it as representing a pathological product of either a primary or a secondary suppurative pleurisy. In discussing the causes of suppuration he introduces a number of interesting facts calculated to maintain his views that the inhalation of dust is a very important causative factor, the dust acting either as a mechanical irritant or because of its contained microbes. He gives an admirable description of Schede's plastic operation upon the chest, but says very little about Estlander's less formidable and often equally serviceable method.

Chapter XVII. is a discussion of the general subject of Peritonitis, and constitutes one of the most valuable chapters in the book. He classifies the disease anatomically, then etiologically, then pathologically, then bacteriologically, and, finally, clinically. It probably is the most elaborate treatment which the subject has received in any text-book. He then proceeds to discuss the handling of each of the clinical forms, which naturally leads him into the discussion of eventration, irrigation, incision of distended bowel, abdominal drainage, and after-treatment; incidentally, also, but fortunately, the subjects of perforating ulcer of the stomach and duodenum, as well as of the typhoid lesion of the small intestine, are dealt with, and the importance of their operative treatment not minimized. It is interesting to note that Senn discusses a so-called hæmatogenous peritonitis, which he describes as occurring without injury or discoverable lesion, and consequently as the result of infection through the blood. It has been observed most often in connection with nephritis, pyæmia, rheumatic arthritis, and the acute

exanthems. A careful perusal of these pages on the general topic of peritonitis will amply repay the time spent. Appendicitis is discussed in the same complete and thorough manner as peritonitis. Its various forms are fully described, and, indeed, well illustrated with a colored plate. The author believes that its rational treatment should depend entirely on its anatomico-pathologic form. Its drug treatment is discussed only from the surgical point of view, which is probably the correct position to take. Intestinal obstruction is the subject of an important chapter which is devoted, however, rather to the therapeutic aspect than to the anatomical or pathological aspect of the topic. The various operations are well described.

A general chapter on Abdominal Section is made to include a large number of the intra-abdominal operations, intestinal suture, anastomosis, etc., while a further special chapter is given to a particular consideration of the latter suture alone. All forms of anastomosis and implantation are in this chapter discussed and suitably described. The next chapter takes up the various pathological forms of Intestinal Obstruction, which are here treated at considerable length and suitably illustrated by diagrams and plates. The various impactions by foreign bodies and their suitable treatment are also described. Intraperitoneal tuberculosis, aside from that of the peritoneum, here finds consideration extending over some thirty pages, which are all replete with interesting statements. Intestinal obstruction after abdominal section is also considered. To the subject of strangulated hernia some twenty pages are devoted, while intestinal fistula receives about the same number, the various pathological causes for the same being fully rehearsed. The concluding portion of the volume, some eighty pages, is devoted to excision of joints and amputation of limbs. It cannot be said that these subjects are completely dealt with by any means, but rather treated by general considerations. To what has been said no exception can be taken. One is tempted only to wish, as he reads, that more complete discussion had been given them.

Altogether the book can be regarded as representing the results of many years of most extensive and varied surgical experience. As such it is an extremely valuable contribution. No one can study it without realizing its value, and, at the same time, without wishing that the author might have been more explicit or extended in certain respects. Such a regret must always be voiced when such an author deals but partially with a given subject or but incompletely with the entire list of surgical topics. A regret thus expressed is, however, the highest compliment that can be given to a work. Altogether, the reviewer regards the book as the best product of Dr. Senn's brain and activity, and bespeaks for it wide-spread attention.

R. P.

OPERATIVE SURGERY. By JOSEPH D. BRYANT, M.D., Professor of the Principles and Practice of Surgery in the University and Bellevue Hospital Medical College, etc. Vol. II., containing 827 illustrations, of which 40 are colored. New York: D. Appleton & Co., 1901.

THE second volume of Dr. Bryant's work on *Operative Surgery* seems to us even more full and commendable than the first. He begins with operations on the mouth and respiratory passages. There has been no

omission of any of the operations on the tongue or mouth which can in any way detract from the value of such an encyclopædic work. The various methods of pharyngotomy are succinctly and well given. Operations on the nose are more fully described than usually, save, perhaps, some of those plastic operations for minor deformity and for purely cosmetic results, which might find more extensive mention. Operative surgery of the œsophagus is fully dealt with, including all the newer operations for stricture, most of which are well illustrated. Tubage of the œsophagus also is described more fully than in almost any other work with which we are familiar. Its complete description, however, does not commend it to the practising surgeon, and the statement that of seventeen cases treated by the method nine died, without obstruction, from the effects of the advance of the disease, is scarcely enough to seriously commend it.

The chapter on Operations on Viscera Connected with the Peritoneum is admirable. It is opened by general considerations, including the toilet of the peritoneum, intra-abdominal drainage, after-treatment, etc. The author then proceeds at once to operations on the intestine, in which are described not quite all the various sutures that have ever been devised, but all that have proven of sufficient value to commend them to the surgeon, and more, in fact, than the ordinary operator can well bear in mind. Intestinal anastomosis by various methods is shown, and the various instruments which have been recently invented for this are sufficiently described. A most valuable addition to the ordinary minute directions for operations will be found in certain sections like that devoted, for instance, to the general operative treatment of gunshot and contused wounds of the abdomen. These are cases which demand all the judgment of an experienced operator, and such judgment has been evinced in general treatment of the subject by Dr. Bryant's discussion. Intussusception, along with other forms of intestinal obstruction, is fully dealt with, both in the text and pictorially.

No such work as this could neglect the general subject of appendicitis, to which a number of pages are devoted, with admirable directions for practitioners, under varying conditions, both acute and chronic. A full description of the various modified incisions and discussion of their advantages are given. The surgical treatment of peritonitis and of perforating ulcer is also fully described. The section on operations on the stomach is preceded by a colored diagram, with discussion of certain anatomical points which one must have ever in mind in order to become a successful operator upon this viscus. Numerous methods of gastrostomy are illustrated, and the author then proceeds to the various ways of bringing about anastomosis between the stomach and bowels. These pages are amply illustrated, and the directions are made so plain that no one can misunderstand them. Surgery of the liver, especially of the gall-bladder and biliary passages, is admirably treated, and the illustrations in this regard leave nothing to be desired. The same may be said of operations upon the kidneys. Modern surgery of the ureters is not neglected, and the reader will find here an epitome of practically everything that has been done in this direction. Hernia is fully treated and numerous methods described. Here, as in some other instances, more methods are given than any one surgeon can well familiarize himself with, and certainly more than anyone ought to practise. Nevertheless, enough is said of all in order to allow an intelligent

reader to select that of which he will most often avail himself. We are particularly struck by the wealth of description of methods of treatment for prolapse of the rectum, everything of known value being mentioned, and even fully described. The various methods of attacking cancer of the rectum are given sufficient space, although the author is not quite so full in this regard as he has been in most others. Operations on the breast are fully described, and the proper plea for thoroughness of work is not omitted.

Surgery of the lung is given its due proportion of space, and that nothing is neglected one may see when even the resection of a tubercular deposit in the lung is described. The author's method of attacking the mediastinum from the dorsal region is furnished, and is made to appear in its full value. The chapter upon Operations on the Neck is full of value and sufficiently illustrated. The chapter upon Operations on the Genito-urinary Tract is exceedingly well illustrated, and we scarcely discover the omission of any operative feature which has any merit whatever. Among the operations on the scrotum and penis one might, perhaps, look in vain for mention of Beck's recent work, and yet this has been, perhaps, too recently published to find mention in this volume. The book closes with miscellaneous operations, among which especially are to be mentioned those upon the patella, which are well described, and the various methods of uniting broken bones by operative or mechanical intervention. Excision of the cervical sympathetic is also included here, instead of under operations on the neck, the description being complete and admirable, and the results summarized sufficiently to enable one to judge of its merits.

Altogether, the work is most admirable and indispensable to every operating surgeon of large practice. As the reviewer compares it, in his mind, with other works covering the same ground, he cannot think of one to which he would wish to turn so often as to this monumental work of Dr. Bryant's.

R. P.

ESSENTIALS OF REFRACTION AND OF DISEASES OF THE EYE. By EDWARD JACKSON, A.M., M.D., Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. Third edition, revised and enlarged. 12 mo., 261 pages, 82 illustrations. Philadelphia and London: W. B. Saunders & Co., 1901.

THIS, the third edition of Jackson's well-known work, the best of its kind, has undergone most careful revision and extension; much that is new has been added, while the entire subject-matter has been brought to date.

For both the ophthalmic student and the busy practitioner who wish to obtain a succinct general account of diseases of the eye, the book can be heartily recommended.

C. A. O.

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Case of Family Periodic Paralysis, with a Critical Digest of the Literature.—SINGER and GOODBODY (*Brain*, Summer, 1901, p. 257) report a case of periodic paralysis in a boy, aged sixteen years, who was admitted to the National Hospital, London, on October 12, 1900. There was no history of any similar trouble nor of any neuropathic tendency in his family. The boy's first attack occurred when he was fourteen years of age, and the second four to six weeks later. The attacks increased in frequency until they occurred almost weekly, when the boy first came under observation. They had practically always come on at night. The writers describe one of the attacks in detail. It began at 3 P.M., with a dull, aching sensation and weakness in the legs. One hour later weakness of the upper extremities began. By 7.30 the muscles of the neck, trunk, and extremities were almost completely paralyzed. The respirations were almost entirely diaphragmatic. The facial muscles and cranial nerves were not affected. There was no mental disturbance. The deep reflexes disappeared *pari passu* with the loss of power. The abdominal and cremasteric reflexes were absent, and the plantar diminished. During the night muscular power gradually returned, and by 10.30 A.M. the following morning he was practically well. The order of recovery was the reverse of the order of involvement—that is, the last muscles to lose power were the first to regain it. Electrical examinations in this and subsequent attacks showed that during an attack there is a gradual reduction of the excitability both to faradism and galvanism until, with complete paralysis, there was complete loss of excitability to both currents. The heart in this case was often dilated during the attacks.

The urine and feces were carefully examined. The toxicity of the former, as tested on rabbits by Bouchard's method, was much increased during the attack, but no attempt was made to isolate the toxin. The experiments on the feces were negative.

Sections of the left sartorius muscle excised when it had completely lost its power to contract showed an extraordinary tendency to fissuring of the individual muscle fibres. This was regarded as an artefact, but was held by the writers to indicate that the muscle had undergone changes, owing to the fact that normal muscle did not give the same picture when the same technique was followed.

The first case of the disease was reported by Hartwig in 1874. The affection is comparatively rare. There is usually a marked family history. This is illustrated by the cases recorded by Cousot, Goldflam, Taylor, and Mitchell, who give the number of relatives affected as five, nineteen, eleven, and seven, respectively. Goldflam and Taylor have both traced the disease back through five generations. The age of the patients at the onset of the first attack has ranged between six and twenty-four years. Both sexes are equally affected. The attacks have almost invariably begun at night. The intervals between the attacks gradually diminish until they occur every week or even oftener. The weakness nearly always begins in the legs, extending to the arms, and lastly to the trunk and neck. During the height of the paralysis the intercostals and extraordinary muscles of inspiration are paralyzed. The diaphragm escapes. The heart is often dilated, at times irregular in action, and occasionally a systolic murmur is heard over it. There is loss of excitability of the muscles to faradism and galvanism during an attack, but normal electrical excitability in the intervals.

Regarding the etiology, there is a general consensus of opinion that the affection is due to the action of a toxin. Whether this is a specific toxin or an increase in some of the normal products of metabolism has not been definitely determined. Life does not seem to be shortened by the disease, and death does not appear to have resulted from the attacks. The number of the attacks diminishes toward middle age, and sometimes they cease altogether.

The treatment has hitherto proved unsatisfactory. The writers recommend that the patient should live a quiet, non-exciting life, with plain, simple food and moderate physical exercise. Diuresis should be promoted by large quantities of water and the administration of digitalis and potassium acetate or citrate.

Babinski's Toe Phenomenon.—SCHNEIDER (*Berlin. klin. Wochenschrift*, 1901, xxxviii., 946, as a result of observations in a variety of different conditions in the Clinic of Professor Jolly, comes to the following conclusions :

The normal reflex movements on irritation of the sole of the foot consist of two reflexes which arise from different points in the central nervous system. The first, simple plantar flexion following a weak stimulus, is a cortical reflex. The second, dorsal flexion of the toes with combined movement of the leg on strong stimulus, is a spinal reflex.

Babinski's phenomenon consists in the fact that, upon a feeble stimulus, plantar flexion does not follow, while dorsal flexion of the toes—that is, a spinal reflex—immediately appears. That this is more easily obtained than under normal circumstances depends always upon a general increase of the spinal reflexes.

Babinski's phenomenon may arise in two ways : First, through interrup-

tion of the pyramidal tract, and consequent obliteration of the cortical reflex ; this interruption may also occur at the beginning of the tract—that is, the motor cortex (epilepsy). This reflex, depending upon organic changes, the author describes as “genuine.”

Secondly, if, as a result of general increase of the spinal reflexes (strychnine, diseases resulting in increased reflexes without lesions of the pyramids), or from conditions which diminish the reflex irritability of the cortex (stupor, œdema), the spinal reflex becomes as easily or more easily obtainable than the cortical reflex, then as a result of the dynamic excess of the spinal reflex the cortical reflex may be suppressed and apparently wholly wanting. Thus the typical picture of Babinski's reflex may arise without lesion of the pyramidal tract.

We are not in a position to separate positively this variety of Babinski's phenomenon, with suppression of the cortical reflex, from that with destruction of the cortical reflex ; it is then impossible, in a condition of general increased reflex irritability, to make the diagnosis of a pyramidal lesion from Babinski's phenomenon alone. However, permanent persistence of Babinski's phenomenon is rare, without destruction of the cortical reflex (a single plantar flexion among a large number of dorsal flexions shuts out a genuine Babinski reflex) ; and since the genuine Babinski's phenomenon is present or develops almost always in lesions of the pyramidal tract, this reflex is of practical value in separating pyramidal lesions from similar pictures.

The Alkalinity of the Blood in Physiological and Pathological Conditions.—ORLOVSKY (*Vratch*, 1901, xxii., 1190, 1222), after a review of the literature and control tests of various methods, gives the results of a considerable number of observations on animals and human beings which lead him to the conclusion that :

1. The previous methods of determining the alkalinity of the blood give varying results even in healthy individuals, and should, therefore, be finally abandoned.
2. Constant results are only to be obtained by methods which determine the alkalinity in the laky blood ; these methods alone have a *raison d'être*.
3. Variations in the alkalinity of the blood noted in various diseases and in one and the same disease by various investigators working according to old methods (Landois-Jaksch's, etc.), depend upon the varying isotonicity of the blood and its varying richness in red corpuscles in different cases.
4. The variation of the alkalinity of the blood noted by authors employing titration of laky blood depends upon the variation in number of red blood-corpuscles.
5. The alkalinity of the blood-plasma is markedly diminished in cancerous cachexia, in advanced uræmia, and in severe diabetes mellitus.
6. In other diseases the alkalinity of the blood-plasma remains unchanged, or, if it does differ from the normal, shows but slight and exceptional variations.

The diminution in the alkalinity of the blood in uræmia, diabetes, and cancerous cachexia depends upon the accumulation in the blood of acid products of metabolism. The direct dependence of diabetic coma upon an

acid auto-intoxication is generally recognized, and the use of alkalies internally is, as is well known, often advised. The author has carried on a series of experiments upon the normal subject to determine the best method of administering alkalies. As a result of investigations in six cases, he finds that: (1) a single dose of bicarbonate of soda produces a relatively unimportant and very transient increase in the alkalinity of the blood; (2) warm alkaline enemata increase the alkalinity of the blood appreciably more than the administration of corresponding quantities of alkalies by mouth; (3) this increase in the alkalinity of the blood is slight and of short duration.

In two cases of diabetes the author was able to show clearly that: (1) the administration of alkalies increases the alkalinity of the blood and of its plasma to an appreciably higher degree than in healthy individuals; (2) warm alkaline enemata increase the alkalinity of the blood and of its plasma appreciably more than doses of alkalies of the same quantity by mouth; (3) this increase in the alkalinity of the blood is of short duration, but lasts longer when the alkalies are administered by rectum.

The author believes that these results are important as indicating the advisability of treatment by alkaline enemata at the first evidence of diabetic coma. The administration of alkali should be continued well after the removal of the immediate danger, as otherwise the alkalinity of the blood will rapidly fall again.

On the Influence of Formic Aldehyde upon the Metabolism of Children.—TUNNICLIFFE and ROSENHEIM (*Journal of Hygiene*, July, 1901, p. 321), as a result of very careful experiments on the effect of formic aldehyde on the metabolism in children, as well as from a review of the literature, come to the following conclusions:

1. In healthy children formic aldehyde administered with the food in doses up to 1 : 5000 in milk, or 1 : 9000 in total food and drink, exerted no appreciable effect on the nitrogen or phosphorus metabolism or fat assimilation. The analytical figures suggest, however, that formic aldehyde has a tendency to diminish phosphorus and fat assimilation, and hence it may be inferred that in larger doses, or if continued for a longer period, it would act in this direction. This effect is referable to an influence upon pancreatic digestion.

2. In healthy children formic aldehyde in the above doses produces a retention of water in the body.

3. In a delicate child formic aldehyde in the above maximum dose had a chemically measurable deleterious effect upon the nitrogen, phosphorus, and fat assimilation, again referable to an action upon the pancreatic digestion, combined with a slight intestinal irritant action. There was a slight tendency to stimulate the katabolism of proteid material.

4. In a delicate child formic aldehyde increased the volume of urine and the weight of feces.

5. In all cases the excretion of lecithin in the feces was diminished under the influence of formic aldehyde. This effect is probably referable to a stimulating action of formic aldehyde on the lecithin-splitting ferment of the pancreas.

6. In no instance did formic aldehyde exert any appreciable intestinal antiseptic action.

7. In no instance was there any influence on the general health or well-being of the children.

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The Intrapleural Pressure in Pneumothorax.—BARD (*Revue de Médecine*, 1901, xxi., 441, 576), in an interesting communication which well repays careful study, reaches conclusions which are best stated in his own words :

1. Owing to the anatomical structure of the lung, wounds of its parenchyma of most varied and extensive nature are permeable to gas and liquids only in the direction from the bronchi to the pleura. Only grave and extensive lesions of the organ or absolute obstacles to the retraction of the tissues, such as are found in partial pneumothorax, may permit the passage of gas or liquids in a direction from the pleura to the trachea.

2. In generalized pneumothorax, when the fistula is not obliterated, the pressure of the gases is positive during both periods of the respiration in quiet breathing. This positive pressure results from the spontaneous tendency toward the equilibrium of the intrabronchial and intrapleural gases by the regular play of respiration, without any part being played by the cough. The hypothetical pneumothorax with a valve is the normal form of general pneumothorax open internally.

Cough and efforts act on the intrapleural pressure more energetically than on the intrabronchial pressure. In this manner they contribute toward compressing the lung and rendering it atelectatic, toward pushing over the mediastinum, but in no way toward allowing the penetration of air into the pleura through the pulmonary wound.

The degree of positive intrapleural pressure is but slight and practically constant in a given patient, presenting respiratory oscillations of several centimetres on either side of a mean of about 6–8 cm. of water. After accidental perturbations it tends gradually to re-establish itself at its fixed level by the normal play of the respiration.

3. The degree of this pressure is, if not equal to the pulmonary elasticity, at least in relation with it and determined by it. This elasticity, which, combining with the action of the walls, brings about a negative pressure in the pleural cavity when the lung is intact, provokes, on the other hand, in pneumothorax with persistent pulmonary opening, a positive pressure, owing to its direct transmission along the fistula.

4. The positive intrapleural pressure in pneumothorax is a phenomenon of pathological adaptation and compensation in relation to the persistence of the fistula, in that it assists in obliterating it; it should be respected when present, and one might well hasten its production during the phase of initial suffocation.

5. Mensuration of the pressure of the intrapleural gases is an element essential for the diagnosis of the varieties of pneumothorax, and principally for the appreciation of the existence of a pulmonary fistula, its persistence, or its obliteration. However, the character of the mean pressure, whether positive, nil, or negative, does not furnish exact indications for diagnosis; one should substitute for this the consideration of the extremes of pressure. The pressure is positive at both periods in generalized pneumothorax with

persistent fistula ; it is positive on expiration and negative on inspiration in partial pneumothorax with open fistula ; it is negative in both periods in general pneumothorax when there is no fistula or when this has been obliterated for a greater or less length of time.

SURGERY.

UNDER THE CHARGE OF

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Pancreatitis.—MAYO ROBSON (*British Medical Journal*, May 11, 1901) states that the essential and immediate cause of the various forms of pancreatitis is bacterial infection, this having been positively proved both clinically in the human subject and experimentally in the lower animals. External determining causes are biliary and pancreatic lithiasis, injury, gastro-duodenal catarrh, ulcer and cancer of the stomach, pylorus or duodenum, and zymotic diseases, such as typhoid fever and influenza, and, in some cases, pancreatitis has come on suddenly in persons of robust health, and the determining cause has been beyond recognition. Fat necrosis is commonly found in association with pancreatitis and other diseases of the pancreas, but it is not pathognomonic of disease of the pancreas. Hemorrhage into the pancreas not due to injury may occur, and there is an ill-understood relation between pancreatic disease and serious hemorrhage. Careful investigation has shown (1) that in certain diseases of the pancreas there is a general hemorrhagic tendency which is much intensified by the presence of jaundice ; (2) that hemorrhage may apparently occur in the pancreas unassociated with inflammation or with jaundice, or with a general hemorrhagic tendency ; (3) that both acute and chronic pancreatitis can and do frequently occur without hemorrhage ; (4) that some cases of pancreatitis are associated with local hemorrhage. The treatment of acute infective pancreatitis is practically that of peritonitis, commencing in the superior abdominal region. The pain at the onset is so acute as to necessitate the administration of morphine for its relief, and collapse will demand stimulants, which, on account of the associated vomiting, may have to be given by enema. In the early stages the symptoms are usually so indefinite as not to warrant operation, and until the collapse has passed off no surgical procedure would generally be justifiable. The simulation of intestinal obstruction will probably lead to efforts to secure a bowel movement, and so relieve the distention. An early evacuation of the septic matter is necessary to recovery, so an exploratory operation is demanded so as to evacuate the septic material and give free drainage. The after-treatment should be

directed to combating shock and keeping up the patient's strength. Even if pus is not found, the exploratory operation will do no harm. The incision is best made in the median line above the umbilicus, and should enable one to establish the diagnosis. The best incision for drainage is a free vertical incision in the left costo-vertebral angle. There is then no risk to the general peritoneal cavity, and drainage will be good, because it will be dependent. Subacute pancreatitis is more amenable to treatment—morphine for the pain, calomel as an intestinal antiseptic, and for the relief of the distention. As soon as the constipation is relieved, diarrhoea is apt to supervene, and this should have its appropriate treatment. If surgical treatment be decided upon either the median incision above the umbilicus or the posterior incision in the left or right costo-vertebral angle should be employed. If a definite abscess forms and approaches the surface in front of or in either loin, the treatment will be incision and drainage, as in the case of any other abdominal abscess. The author reports five cases of subacute pancreatitis treated by operation, with the result of three recoveries and two deaths. Chronic pancreatitis must be treated by abdominal section and drainage, but in these cases the drainage should be indirect, and obtained by draining the gall-bladder by cholecystotomy, cholecystenterostomy, or duodeno-choledochotomy. The exact line of treatment cannot be determined until the abdomen is opened. When there are gallstones present they should be removed, unless the patient is too ill to permit of the complete operation; but in every case drainage must be secured, if possible, by cholecystotomy; and, moreover, the drainage must not be stopped before the bile has become healthy, and not before the greater amount of bile is being passed by the bowel, which will be certain to occur as soon as the swollen pancreas has subsided, if the duct be otherwise clear of obstruction. The simulation of malignant disease of the head of the pancreas by chronic interstitial pancreatitis would make the author hesitate to decline operation in any case of distended gall-bladder where the patient is in a condition to bear it, or even in any case of chronic jaundice without distention of the gall-bladder where the general health is deteriorating, as though it should be recognized that if the disease be really malignant very little good will be done, and life may even be shortened or only prolonged for a short time, yet if the disease prove to be chronic pancreatitis a real and permanent cure may be brought about. The results of treatment in this class of cases have been most encouraging, as out of twenty-two cases operated on only one died directly from the operation, and in that case the patient's life was only very slightly shortened, since he was reduced to the last stage of exhaustion before the operation was performed. Of those recovering from the operation, with the exception of two that died a few months later, complete and permanent recovery ensued. These results contrast very markedly with the surgical treatment of cancer of the pancreas, where nearly half the cases operated on have died directly as the result of operation, and in those who have survived life has only been prolonged for a comparatively short time.

The Operative Treatment of Cirrhosis of the Liver.—FRAZIER (*Annals of Surgery*, June, 1901) reports the case of a man with marked cirrhosis of the liver who had been repeatedly tapped and whose condition seemed very

hopeless. Operation being decided upon as a last resort, the peritoneal cavity was opened. The parietal peritoneum of the abdominal wall on either side of the incision was rubbed quite vigorously with a gauze pad, and the omentum, which was very much thickened and contracted, sutured to the parietal peritoneum and to the margins of the wound. The fluid contents of the abdominal cavity were evacuated and the incision closed without drainage. Convalescence was uninterrupted; the patient suffered no ill effects from the operation. The wound healed throughout *per primam*. The history subsequent to the operation—since which three months have elapsed—is briefly as follows: The patient has been tapped twice, once on the thirteenth day, 328 fluidounces having been withdrawn, and again on the thirty-sixth day, on which occasion only ninety-six ounces were withdrawn. From that time to the present writing there has been absolutely no reaccumulation of fluid. The patient has gained rapidly in strength; he is no longer bedridden, goes out daily, and receives no medication other than enough citrate of magnesia to insure a daily evacuation of the bowel. This operation purports to open another channel for the relief of the obstructed portal circulation. The author says another channel, because there already exists a more or less free collateral circulation between the systems of the portal vein and the inferior vena cava. Thus, the coronary anastomose, through the cesophageal plexus, with the azygos veins; the veins of the cæcum and colon with the internal mammary; the hypogastric with the hemorrhoidal; the veins of the hepatic ligament with those of Glisson's capsule; the veins of the round ligament with the epigastric. By inciting the formation of adhesions between the omentum and the abdominal wall and between the surfaces of the liver and spleen and that of the diaphragm, this operation furnishes an additional outlet for the blood of the obstructed portal system. The examination of specimens obtained at the autopsy table has proved beyond a doubt that the operation as conducted accomplishes this purpose. Thus, in the case operated upon by Lens, venous channels were easily demonstrable in the adhesions that had formed between omentum and peritoneum.

The chief indication for the operation is the presence of ascites due to obstruction of the veins of the portal system, when the obstruction itself is due to cirrhosis of the liver. The operation is not indicated in every case of hepatic cirrhosis with ascites; the operation is absolutely dependent for its success upon the retained function of the liver cells. In other words, the absence of functional activity is an absolute contraindication. At the time of this writing the records of but fourteen cases (including the author's) have appeared in literature. The number of operations, therefore, is so limited that no very definite conclusions can as yet be drawn. When we exclude those in which there was some error of technique, those in which there was an error of diagnosis, or those in which the operation was contraindicated, but eight cases remain. Of these none died (mortality, 0 per cent.); one was living and unimproved (12.5 per cent.); one living and improved (12.5 per cent.), and six were living and free from ascites at periods of three, four, six, twenty-four, twenty-four, and twenty-six months respectively (75 per cent.).

This operation is indicated in those cases (1) in which the liver is cirrhotic; (2) those in which there is reason to believe the liver cells are not devoid of function; (3) those in which internal medication (particularly iodide of

potassium) and paracentesis fail to afford relief, or, in other words, in utterly hopeless cases, and (4) those in which there is no reasonable contraindication—the operation has a future. The cases are so hopeless, the technique so simple, the dangers so trivial, and the outlook so promising, that the prospects of this mode of treatment becoming an established one seem bright. That surgeons now have at their command a method of affording some relief in cases of intractable ascites, is evident.

The Application and Merits of Short-circuiting in Certain Diseases of the Intestines.—MAYLARD (*The Medical Chronicle*, April, 1901) states that the term "short-circuiting" is to be understood as the formation of a passage between one segment of the bowel above with another segment below, and thus the intervening portion of intestine is partially thrown out of action. There are two primary uses to which this method may be put—the one where it is desirable to divert the course of the intestinal contents past some obstructing influence, whether within or without the bowel; and the other where such diversion of fecal matter will admit of the closing of an artificial anus or fecal fistula. The author reports two cases of intestinal obstruction, both tubercular in origin, which made uninterrupted recoveries after this operation. This operation is indicated in cases of extensive malignant disease of the colon, and should prove a very valuable method of treatment. There are two methods by which short-circuiting may be executed—one by the lateral approximation and union of two loops of intestine and the establishment of a fistula bimucosa (entero-enterostomy, entero-colostomy, colo-colostomy), and the other by the division of a free segment of the gut, the closure of the distal end, and the lateral implantation of the proximal end into another free segment. The former method incompletely cuts off the passage of the intestinal contents through the partially occluded segment of gut, while the latter completely does so. Both methods have their own peculiar advantages, which are not immaterial in considering the particular method best suited for each individual case. Stitching should alone be employed in either method. In implanting the free end of the ileum into the colon it is as well to invaginate it for about half an inch. This is easily done by passing a traction stitch through the patent end of the ileum, and, after transfixing the colon at a couple of inches or so with the needle carrying the traction thread, pulling on the latter. While thus secured it is carefully stitched all around with a double circle of Lemberts. The traction thread is last withdrawn. The point requiring most attention is at the attachment of the mesentery to the invaginated end of the ileum.

In producing a fistula bimucosa the two portions of the gut involved in the union should be laterally approximated in such a way that the natural downward current of the feces is maintained. From two to three inches of the applied surfaces of the two segments of gut should be stitched first, this line of union forming the posterior boundary of the opening. Then the bowel wall of each segment is incised and the edges of the projecting mucous membrane of each orifice carefully united together, after which the posterior layer of stitches is continued on around the front until an external circle of Lemberts has been completed.

PEDIATRICS.

UNDER THE CHARGE OF

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Diphtheria Bacilli in Noma.—JOSEPH WALSH (*Proceedings of the Philadelphia Pathological Society*, June, 1901, p. 179) reports that he has found diphtheria bacilli in all of eight cases of noma examined bacteriologically. Only one of these cases gave a pure culture of the diphtheria bacillus, and this occurred in a case with associated pharyngeal diphtheria, the child subsequently recovering. Another developed noma during measles, having suffered from diphtheria four months previously, and at the time of development of measles being isolated with another child who also presented clinical symptoms of diphtheria, but with negative cultures as regards this organism. Three of the other cases began just after measles. Four of the eight cases began with an ulcerative stomatitis. Fifteen other cases of ulcerative stomatitis were examined in hope of finding diphtheria bacilli, but with negative results.

He concludes that since noma is a species of moist gangrene, requiring probably, from analogy, two different organisms—one a saprophyte, to produce the putrefaction; another a parasite, to produce the primary necrosis—it is possible that in the cases in which diphtheria bacilli are found they may be the primary causative agent; and, secondly, that when other pathogenic micro-organisms capable of producing necroses are found it is possible that they may be the primary excitants.

Confirmatory Evidence in Favor of the Fourth Disease of Dukes.—FREDERICK T. SIMPSON (*Archives of Pediatrics*, September, 1901, p. 698) adds some confirmatory evidence as to the existence of a fourth disease resembling in some features both rubella and scarlatina, a question which was raised about a year ago by CLEMENT DUKES (see abstract of Dukes' paper in this department of *THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, February, 1901, p. 233). An epidemic of twenty-seven cases of eruptive disease was observed by the author at the American School for the Deaf, in Hartford, in the spring of the present year. The symptoms generally resembled those of rubella, the rash in most of the cases being of the morbillous type. A slight branny desquamation followed the rash in the majority of the cases, but in eight desquamation occurred in strips or lamellæ—in three copiously and universally—in the others confined to the hands and feet. In one case the epidermis of the entire heel was separated like a cast. The cases to desquamate most copiously were of the scarlatinal type, except one of the morbilliform type. The throat was slightly injected in most of the cases, but in only three or four was there a subjective feeling o

soreness so far as could be ascertained. Enlargement of the post-cervical lymph nodes was a marked feature in every case. If looked for, the axillary and inguinal nodes were also found enlarged. True strawberry tongue was never seen, and vomiting occurred in only three cases. Six cases developed a second rash at from ten to fifteen days after the appearance of the first rash. One of these looked like a heat rash, but the others were coarse, discrete, and papular.

The points which justified the diagnosis of German measles as from scarlatina were: 1, absence of albumin in the urine in all cases; 2, known period of incubation of two weeks or more in five cases; 3, previous occurrence of scarlet fever in five cases, or 18 per cent.; 4, profuse development of rash in all cases, with slight disturbance of temperature and pulse; 5, absence of vomiting, strawberry tongue, angina, and of complications; 6, morbilliform type in two-thirds of the cases.

In the epidemic of nineteen cases at Rugby, described by DUKES (*Lancet*, July 14, 1900, p. 89), over 40 per cent. of the patients had already suffered from rubella, which, therefore, proved that the disease in question was not German measles. In the differentiation from scarlet fever, however, as was editorially pointed out by the *Lancet*, two important points were lacking: First, none of the patients had had scarlet fever, and, second, the period of incubation could not be established. In Simpson's cases, however, both these points were determined, so that it may be regarded as demonstrated that some cases of the affection hitherto known as German measles may desquamate like scarlet fever.

Apart from this interesting observation as to desquamation, of which one of the editors of this department can offer personal confirmation, Dr. Simpson advances evidence in support of Dukes' theory. He believes that in the epidemic he here reports two diseases were present instead of one, and that these two were rubella and the "Fourth Disease."

His evidence for this is as follows:

First, a number of patients, at least five, had a second rash. This occurred between two and three weeks after the first, lasted several days, was accompanied by a slight rise of temperature, but by no feeling of malaise. The rash was always coarse and discrete. It is difficult to account for these second rashes, except by assuming another disease, since relapses in German measles do not occur.

Second, four of the patients were stated to have had rubella previously. This has not the same probability as the statement that five of the pupils had had scarlet fever, for it will be recalled that quite a proportion of deaf mutes become so in consequence of scarlet fever. Nevertheless, the statement cannot be wholly discredited.

Third, Dukes has made the statement that a form of pink-eye may be the only symptom of rubella; that it may produce the ordinary form in another patient, and that it is protective against a second attack of rubella. An epidemic of pink-eye, affecting ten pupils, had occurred in the school shortly before the first case of the exanthematous epidemic. Three of these pupils had been subsequently affected.

Fourth, the fact of lamellar desquamation in a large proportion of cases of a disease which was certainly not scarlatina, does more than anything else

to make credible the existence of another eruptive disease. This form of desquamation is the chief diagnostic distinction between the fourth disease and rubella. If it belonged to rubella it would certainly have been noted long before Dukes' observations.

To restate the case: here is a series of cases of an eruptive fever having an incubation of at least two weeks, eight of which peeled in strips, five of which previously had had scarlet fever, and four, and possibly seven, had had German measles.

It is quite possible, therefore, that some of the mild epidemics of so-called scarlet fever occurring in the spring and summer are in reality the harmless affections which now pass under the name of German measles.

The Treatment of Intussusception in Children.—An interesting discussion on this subject before the British Medical Association, at its recent meeting, was opened by BERNARD PITTS (*British Medical Journal*, September 7, 1901, p. 574). The conclusions of his paper were formulated as follows:

1. Try inflation only when the case is seen within a few hours of onset, and is not of a very acute character. In the great majority of hospital cases it is better to open the abdomen at once.

2. Inflation may be tried in certain other cases for the purpose of reducing the main portion of the intussusception and enabling the incision to be made directly over the cæcum.

3. When reduction is found impossible in chronic cases a resection may be generally done through an incision in the ensheathing bowel.

4. In acute cases, and especially if gangrene is present or the condition of the bowel requires its removal, a wide resection should be undertaken as rapidly as possible, and the ends brought outside the abdomen; continuity should be restored at a subsequent operation.

5. In exceptional cases of enteric intussusception resection and immediate restoration of continuity gives the only chance.

He called attention to the fact that he had noticed after abdominal section in young infants, especially when there had been much manipulation of the intestines, that death is often preceded by high temperature and delirium, and takes place within twenty-four hours after the operation. Nothing is found post-mortem to explain the temperature or the death.

The general consensus of opinion of other hospital surgeons who discussed Mr. Pitts' paper was against the employment of inflation or injection, not even excepting the cases in which early recognition of the condition had been possible.

The Pathogenesis of Rhachitis.—RUDOLF FISCHL (*Archiv für Kinderheilkunde*, 1901, Hefte 5 u. 6) reviews the various theories that have been advanced to explain the etiology of this disease, but regretfully concludes that careful inquiry into the pathogenesis of rhachitis has failed to throw light upon the subject. On the contrary, he states, theories that hitherto seemed well-founded upon clinical observation and statistics have been found fallacious. He considers that Stölzner is right in holding that the future theory of rhachitis must be founded upon cellular pathology and biology.

THERAPEUTICS.

 UNDER THE CHARGE OF

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Medication by Latent Arsenic.—DR. ARMAND GAUTIER under this title gives a résumé of his own and other researches on the therapeutic value of the cacodylates. He says that there are a large number of observations now on hand which justify for this form of medicament a settled place in therapy. They have been employed with benefit in a vast variety of diseases; grave anemia, cachexia of various origins, tuberculosis in its numerous forms, pulmonary, osseous, and visceral, scrofulosis, herpetism, asthma, cutaneous diseases of many types, malaria, disturbances of menstruation, obesity, chorea, paralysis agitans, neurasthenia; myxœdema, scleroderma, exophthalmic goitre, syphilis, and sarcoma—truly a formidable array. In the matter of administration it is best given by the hypodermic method. Patients can bear five-sixths to one and two-thirds grains a day and continue such doses for at least two or three years without symptoms of arsenical poisoning or the occurrence of anatomical lesions in the viscera. The drug may also be administered by mouth, but it seems to be less well borne. This is interpreted as due to processes of oxidation and transformation of the cacodylates into more active forms of arsenic. He concluded his paper by giving the histories of a number of patients suffering from tuberculosis. These were much benefited, and the success of his treatment lead him to conclude that the cacodylates are capable of curing many patients with pulmonary and visceral tuberculosis, even when in seemingly advanced stages. —*Bulletin de l'Académie de Médecine*, 1901, vol. lxx., p. 20.

Action of Arsenic on Healthy Tissues of the Skin.—DR. LESLIE ROBERTS treats this subject from a broad biological point of view. He says that arsenic is universal in its distribution, being like nitrates and nitrites, a soil ingredient, and capable of entering into the tissues of plants and animals. It is closely associated with iron, nickel, and cobalt. In the human body arsenic combines with albumin, and excites the metabolism of cells to greater activity. Albumin and fibrin, according to the observations of Binz and Schultz, are capable of reducing arsenic acid to arsenous acid; blood exerts a powerful reducing action on arsenic acid, but has no action on arsenous acid. Fat exercises no action on the oxidation of this metal. The brain, liver, pancreas, and kidney all exercise a great activity. This arsenical oxidation is held by the author to be in all probability a normal function of

the skin, and he draws an analogy between the nutritive value of this metal for the skin and the thyroid for other types of metabolism. These metabolic activities are brought about by the formation in the tissues of nascent oxygen. He further draws other analogies, saying that perhaps Gautier's inference, that there exist in the body certain functional elements, of which very few have as yet been discovered, but which are essential to the performance of certain functions, is correct. Thus manganese is said to be essential to the oxidizing ferment; iodine to the thyroid; phosphorus to the nucleinic bodies; fluorine to the bone cells. Of the functional elements, some appear to play the rôle of "principal actors," while others may be termed substitutional elements or "understudies." Thus arsenic may, to some extent, take the place of phosphorus in relation to the nucleins; selenium is thought to be able to act as sulphur; copper, zinc, and manganese may replace iron; while phosphorus, arsenic, vanadium, or bismuth may play the rôle of nitrogen. Beyond these as yet theoretical assumptions, it is certain that arsenic is not merely an irritant poison, but its action is determined by the tissues themselves; that its effects are essentially of a nutritive order brought about by the agency of active oxygen; that these effects are of benefit to the organism when the oxidation is slow and detrimental when rapid; that the more highly organized the cell the more unstable its protoplasm, and the more rapid its metabolic processes, the more readily does it feel and manifest the action of arsenic.—*British Medical Journal*, 1901, No. 2126, p. 861.

Action of Arsenic on the Skin.—DR. H. G. BROOKE concludes from a series of microscopical studies that the main action of arsenic is obviously a stimulation of the growth and functions of the epithelium. The inter-papillary pegs are very irregularly enlarged and prolonged downward, the prickle cell layer increased, the granular layer always and often very considerably thickened, the stratum lucidum is abnormally well marked, and the horny layer altered in character and unusually pronounced. As the poisonous action of the arsenic continues, either by the further absorption or the process of elimination of the arsenic through the skin, the hyperidrosis ceases and the horny layer becomes dry and lamellar, and scales off rapidly. Usually this is preliminary to restitution of tissue, but in pronounced cases the stimulation may have been so great that the cells are unable to recover, and atrophy of a more or less complete character sets in. The papillæ flatten, the cells lose their position and special characters until the whole epithelial layer is represented by three or four rows of degenerate cells, with a last trace of a stratum lucidum and a covering of very thin flat paper-like lamellæ. The sebaceous glands are not affected except by general atrophy, their fatty contents apparently rendering them immune to the action of the drug.—*British Medical Journal*, 1901, No. 2126, p. 860.

Quinine as a Prophylactic in Malaria.—SIR WILLIAM MACGREGOR contributes an affirmative note to the discussion as to the value of quinine as a prophylactic. He thinks it highly probable that at least as much can be done by the use of quinine as by the employment of mosquito netting. The greater number of government officers take quinine regularly; but, he adds, so long as the taking of quinine is not compulsory there will always

be a residuum of men who, either because they cannot tolerate quinine or from some other reason, will not use it as a preventive of fever. The more common method in vogue in Lagos is to take daily doses of from two and one-half to five grains, but several take a large dose weekly and others irregularly. As a general prophylactic for the native he gives some interesting figures, showing that it would take seventy tons of quinine a year to efficiently protect the entire population of three millions at a grain a day regular dosing. This is an economic impossibility, and their efforts are being concentrated on the foreign population.—*British Medical Journal*, 1901, No. 2124, p. 680.

Prophylaxis in Malaria.—DR. R. BLANCHARD, in an interesting and complete paper on the mosquitoes of Paris, shows that that city is more than well supplied with various species of Anopheles, and he formulates the following general rules for the prophylaxis of malaria, which follow closely those laid down by Laveran in 1900: 1. In quarters where Anopheles are found it is indispensable to make use of mosquito netting during the sleeping hours. Such mosquito netting arrangements should be so constructed as to permit of plenty of air and yet not admit the insects. 2. To protect the body from the bites of mosquitoes the author has found that the most efficient lotion is one from quassia—prepared by simple maceration in water. The various pomades and tinctures advised have been found less efficient. 3. To drive mosquitoes from the rooms he recommends the use of formaldehyde vapor, freshly generated from a lamp. This should be done about an hour before retiring. For those who are particularly susceptible to this vapor, pyrethrum pastilles may be burned. 4. He further advises the use of wire screens. 5. To treat the actual sting, tincture of iodine is an efficient remedy. 6. The breeding places should be carefully drained.—*Bulletin de l'Académie de Médecine*, 1901, vol. lxx., p. 223.

Treatment of Malarial Fever by Iodine and Potassium Iodide.—DR. A. REGNAULT, while recognizing the great value of quinine in the treatment of malaria, says that it is now becoming a matter of general recognition that the quinine-series of drugs is of service only during certain developmental periods of the disease. It is only while the parasite is undergoing its definite evolutionary history that these agents are poisonous enough to kill, and it is the practice at the present time, in France, at least, to administer quinine from six to eight hours preceding the onset of the fever, in order to cut short the stage of the division of the parasite. He also points out, by analogy, with certain septicæmic and pyæmic organisms, that the chill of malarial fever is to be interpreted as a chemical reaction to a toxin; and certain nervous manifestations of the disease, notably malarial polyneuritis, seem to corroborate this view. It is held that the toxins are developed with great rapidity just at the time of the division of the parasites, and the fever is therefore but a sign of the reaction to these protozoal poisons. It is with the idea of bringing about a chemical destruction of such poisonous bodies that the author has suggested the use of iodine and potassium iodide, iodine being a body which has a special affinity for bodies of the alkaloid class, and presumably for the supposed analogies of these

toxins. The results obtained are reported as being very striking, in that not only were the attacks aborted, but the action on the fever itself was striking and immediate, chills, vomiting, and malaise disappearing rapidly. The remedies were employed in the following strength: Tincture of iodine and potassium iodide, of each, 1; distilled water, 25. A teaspoonful in a little water at the beginning of an attack; a second teaspoonful fifteen or twenty minutes later, if required. In those patients in whom vomiting is a prominent symptom fifteen minims of ether may be added to the first dose. The use of Lugol's solution in chronic malaria is old, but this remedy has rarely been used in the attacks. It should be borne in mind that there is a chemical antagonism between iodine and quinine, and that when both are used enough time should elapse to permit of the elimination of either.—*Revue de Médecine*, 1901, vol. xxi., p. 804.

Antidotes for Cyanide Poisoning.—DRS. C. J. MARTIN and R. A. O'BRIEN, of Melbourne, commenting on the fact that cyanide poisoning had become not infrequent among the Australian miners since the introduction of the use of the cyanides in the extraction of gold, present a careful series of studies to determine, if possible, a practical course to pursue in the treatment of this condition. Hydrogen dioxide and the salts of cobalt have of late years been extolled, Kobert writing at great length of the value of the former antidote. Shouli first pointed out the desirability of forming insoluble cobalt cyanide. Ferrous hydrate has had much vogue in hereditary text-book writings. These investigators have studied these three agents in particular. Pure chemicals were employed, and rabbits were used in the animal experiments. In these animals 1 to 100,000 of potassium cyanide produced, within seven minutes, staggering gait, very rapid respirations, followed shortly by labored breathing; convulsions of varying intensity always occurred, and at times appeared within three minutes after the introduction of the drug. Exhaustion followed the spasms. Respiration then consisted of heaving movements of the chest and abdomen. The breathing became shallower and shallower, and the animals died. If the animal did not die within thirty minutes it usually survived. Hydrogen dioxide as recommended, subcutaneously in 3 per cent. solution and by the stomach in 2 per cent. solutions, was employed. When introduced subcutaneously it was immediately broken up, and its effects were *nil*—the animals all died; a similar result was obtained by its use by the stomach, even when the poison and its supposed antidote were introduced mixed. The authors say that hydrogen dioxide does oxidize cyanides, but such action is too slow to be of therapeutic value, and they detail a series of experiments to determine the velocity of this reaction ($2\text{HCN} + \text{H}_2\text{O}_2 = \text{C}_2\text{O}_2\text{N}_2\text{H}_2$). This interaction requires from twenty to seventy minutes, and any antidote for potassium cyanide must act instantaneously. Cobalt chloride was then studied in the same careful manner. Twenty-six experiments pointed to the fact that the cobalt chloride was capable of forming an insoluble cyanide, but that it must be given in excess; the acid of the gastric juice does not interfere with the reaction. Cobalt chloride itself, however, as pointed out by Stuart, is not free from poisonous action, producing severe gastro-enteric symptoms. This should be carefully guarded. Ferrous hydrate when added to a cyanide

salt forms a ferrocyanide almost instantaneously. Such ferrocyanides are but slightly poisonous, and its administration would seem to be even better than the cobalt salts, in view of their toxic action; but, unfortunately, the strongly acid character of the stomach contents greatly hinders this action, hence alkalis must be added to neutralize the free hydrochloric acid. The author's summary of results shows that there is no known physiological antidote for cyanide poisoning. Hydrogen dioxide is practically worthless, given in any form; cobalt salts are valuable, but are poisonous; ferrous salts administered with sufficient alkali are as efficacious as cobalt salts. At the body temperature the formation of ferrocyanides is instantaneous, but they possess two disadvantages: (1) They are kept in solution with difficulty, and (2) there is an absolute necessity of using enough alkali. This is best effected by the use of doses of magnesium oxide. In all mines where such processes are employed there should be kept solutions of ferrous sulphate, weak potash, and a small packet of magnesium oxide, together with a stomach-tube and a suitable receptacle for mixing, so that the remedies could be administered without delay, as time is the most important factor. They recommend an ounce of a 23 per cent. solution of ferrous sulphate; one ounce of a 5 per cent. solution of caustic potash; thirty grains of powdered magnesium oxide; a metal receptacle of one pint capacity; stomach tube. The first two solutions should be kept in air-tight tubes which can be broken into the receptacle, and powdered magnesia and half a pint of water added, shaken up, and administered. This amount of antidote will take care of seventy-five grains of potassium cyanide.—*Intercolonial Medical Journal of Australasia*, 1901, vol. vi., p. 245.

Malarial Parasites and Methylene Blue.—DR. A. IWANOFF administers methylene blue in three daily doses of 5 grains each to patients in whose blood tertian parasites have been found. The first changes observed began at the end of the second day, and affected only the full-grown forms; the smaller, younger forms remain unchanged. The adult organism showed diminished movements and a breaking up of the protoplasm into lumpy-like bodies, which are rounded and lie on the blood cell apart from one another, or are connected with each other by minute bands of protoplasm and pigment granules. The protoplasm of these lumps is shrunken. The nucleus of the parasite is found generally near the peripheral portion of the blood-cell, and shows in its carmine-violet stained chromatin that it consists of numerous granules. Among the sporulation forms he found, beside normal sporozoites, others which were not fully formed. Some show their protoplasm very distinctly, while others appear to have no plasma at all. The pigment is gathered in patches, and these are irregularly distributed. The chief points he noted in the action of methylene blue on the tertian forms are destruction of the protoplasm and absence of alteration of the chromatin. In the parasite of æstivo-autumnal fever no changes were observed. In the crescent forms there was shrinking and granulation of the protoplasm. He noticed a more equal staining of the whole parasite than in the normal. The pigment granules appear coarser and are grouped in an irregular mass, which projects from the surface of the shrunken protoplasm. The destructive process of the protoplasm of malarial crescents goes on until there is noth-

ing of the parasite left except the pigment. Comparing the effects of methylene blue with those of quinine on the various forms of malarial parasites, it is said that the former affects the protoplasm, and the latter the pigment (chromatin). In the early stages the proportion of the pigment to the protoplasm is quite different to that in the adult, and thus the early forms are affected but slightly by methylene blue, while they are affected markedly by quinine, while the adult forms show the reverse.—*Deutsche med. Wochenschrift*, 1901, No. 18, p. 276.

GYNECOLOGY.

UNDER THE CHARGE OF

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Cancerous Infiltration of Fibromyoma.—GLOCKNER (*Centralblatt für Gynäkologie*, No. 37, 1901) reports an interesting case of adenocarcinoma of the body of the uterus complicated with intraligamentary fibroid. The entire mass was removed by abdominal section, the patient succumbing to septic peritonitis following perforation of the intestine at the site of a secondary cancerous nodule. Metastases were found at autopsy in the retroperitoneal glands, omentum, and intestines.

An examination of the specimen showed that the disease had penetrated the uterine wall at a point adjacent to the attachment of the fibroid, and had extended to the centre of the latter growth, a large cavity having been formed, which was lined with cancerous masses. The interior of the uterus was studded with nodules separated by islands of normal endometrium. The latter condition confirmed the reporter in his view that carcinoma of the uterine body often develops from a number of isolated nodules in the endometrium.

Origin of Uterine Fibroids.—BLAND SUTTON (*British Medical Journal*, April 6, 1901) reports a case in which a fibroid uterus the size of the fist was found to contain by actual count 120 fibromyomata, varying in size from a coriander seed to a cherrystone. He notes the fact that in every instance the nodule surrounded an artery, hence his inference that they originated in the muscular coat of the vessels. He formerly believed that they bore a close relation to the nerves.

Observations on Cancer of the Uterus.—BECKMANN (*Centralblatt für Gynäkologie*, No. 38, 1901) presents the results of his clinical studies in 437 cases of inoperable carcinoma of the uterus, which were under observation in the hospital in the course of two and a half years. These included forty-

three cases of recurrence after radical operations. The majority of the patients had borne several children. Dating from the appearance of the first symptoms of disease, the average duration of life was longer in women with recurrence following hysterectomy.

Primary Carcinoma of the Fallopian Tube.—BOURSIER and VENOT (*Revue de Gyn. et de Chir. Abdom.*, 1901, No. 2), in reporting a case of this rare condition, state that thirty-one have already been recorded. The ages of the patients varied from forty to sixty years. The menopause does not seem to be an important factor in the development of the disease. Salpingitis has a direct bearing upon it.

Retro-uterine Hæmatocoele.—LEWINSOHN (Inaugural Diss.; abstract in *Centralblatt für Gynäkologie*, 1901, No. 38) gives the result of a study of eleven cases, in only four of which was the hemorrhage clearly due to tubal abortion. In one the blood came from a ruptured varicose vein on the surface of an ovarian cyst, but in the remaining six no cause could be assigned. Eight patients recovered without operation, the blood becoming entirely absorbed.

The writer states that Winckel never incises a retro-uterine hæmatocoele, and has never lost a patient in consequence of this conservative treatment.

KOBER (*Centralblatt für Gynäkologie*, 1901, No. 39) reports two cases of celiotomy for supposed ruptured ectopic gestation, in both of which extensive retro-uterine hæmatocoeles were found. In both instances the tubes showed absolutely no evidence of gestation, and were clearly not the seat of the hemorrhage. In the first case it was probably due to excessive coitus; in the second to a violent muscular effort.

The writer does not approve of operation in the case of old hæmatocoeles which tend to become absorbed.

Alcohol in the Treatment of Inflammation of the Pelvic Organs.—SCHMID (Inaugural Diss.; abstract in *Centralblatt für Gynäkologie*, 1901, No. 39) reports seventy cases of endometritis and diseased adnexa, which were treated as follows: The abdomen was covered with a compress saturated with 60 per cent. (later, 95 per cent.) alcohol, over which was placed rubber tissue, these being renewed thrice daily. Tampons saturated with 30 per cent. alcohol were inserted in the vagina every other day. The rest of the treatment consisted in lysol douches, rest in bed, massage and simple diet. At first there were some pain and elevation of temperature. 20 patients were cured; 11 were improved; 31 had a symptomatic cure, while the local condition was improved. 26 patients were treated with ichthyol tampons for the sake of comparison. Of these only 1 was cured; in 5 the symptoms were entirely relieved; in 9 there was improvement both in the symptoms and in the local condition; in 9 in the symptoms only.

Pseudoligamentary Ovarian Tumors.—SCHENK (*Centralblatt für Gynäkologie*, 1901, No. 13) calls attention to the fact that tumors of the ovary rarely develop between the folds of the broad ligaments, but more often behind them. To these he applies the term "pseudo-intraligamentary."

This condition is usually due to peri-oöphoritis, with resulting exudates which surround the growth. He reports a case which seemed to justify the inference that the inflammatory capsule is formed around the ovary before it undergoes cystic degeneration. It is sometimes exceedingly difficult to decide, even on careful examination of the specimen after removal, whether the neoplasm is intraligamentary or pseudo-intraligamentary.

Extension of Uterine Cancer through the Lymphatics.—PUPPEL (*Centralblatt für Gynäkologie*, 1901, No. 13), after careful studies of cancerous uteri, arrives at the conclusion that extirpation of the entire organ for cancer of the portio vaginalis is not justifiable. He found that the first lymphatics to be infected are those in the middle muscular layer. From this point extension of the disease takes place either to the vagina, or to the lymphatics of the broad ligaments adjacent to the original focus.

Metastases in the body of the uterus occur subsequently, the lymphatics of the inner muscular layer being first affected. Hence the deduction that high amputation, with resection of the broad ligaments, is the operation to be performed in cases of operable cancer of the portio, especially in elderly women.

Etiology of Hydrosalpinx.—POMPE VAN MEERDERVOORT (*Nederl. Tijdschr. v. Verlosk en Gyn.*; *Centralblatt für Gynäkologie*, 1901, No. 13), reporting a case in which a large hydrosalpinx developed in a patient, aged fifty-four years, five weeks after curettement, explains its occurrence by the theory that the hyperæmia of the pelvic organs preceding the climacteric leads to an increase of the normal secretion of the tubal mucosa. In consequence of the subsequent senile involution of the tube its epithelial lining is lost, the opposite surfaces adhere, and the abdominal ostium is closed so that the contained fluid cannot escape and a hydrosalpinx results.

Errors in Diagnosis of Ectopic Gestation.—EDGAR (*Glasgow Medical Journal*, 1900, No. 6) reports a case of pelvic hæmatocele resulting from the rupture of hæmatoma of the ovary, two cases of pyosalpinx, and one of suppurating intraligamentary cystoma—all of which presented subjective and objective symptoms of extra-uterine gestation.

Salpingotomy.—GOVILLIOVD (*Lyon Méd.*; *Centralblatt für Gynäkologie*, 1901, No. 13) recognizes only two indications for this operation, viz.: (1) Cases in which the tube is thickened and its abdominal end is adherent and closed, the patient suffering so much pain that she is incapacitated from going about and attending to her work. (2) Chronic catarrhal salpingitis and hydrosalpinx. He does not approve of the conservative treatment of pyosalpinx except in those cases in which the pus is known to be absolutely sterile. Salpingotomy is unattended with risk, and recurrence is rare, while the possibility of future conception is a decided advantage.

Post-operative Peritonitis.—HINTZE (*Centralblatt für Gynäkologie*, 1901, No. 28) reports two successful operations for this condition. In a case of severe infection after Cæsarean section the abdomen was twice opened within twenty hours after the primary operation. The writer believes that the earlier interference is resorted to the better is the prognosis. The operator should confine himself to the removal of the masses of plastic lymph.

DERMATOLOGY.

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INSTRUCTOR IN DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

Ringworm from Canaries.—MACLEOD (*British Journal of Dermatology*, May, 1901), at a séance of the Dermatological Society of London, presented a photograph of a case of ringworm in a boy, aged nine years, of more than ordinary interest, owing to the unusual source of the infection. There was a well-defined plaque upon the back of the left wrist about two inches in diameter, reddish in color, uniformly elevated above the surrounding skin, with a slightly raised vesicular border. The patch, which had lasted about ten days, was peculiar in that it had not cleared up in the centre. The disease was traced to a pair of pet canaries which the patient was accustomed to feed upon his hand. The skin of the head and neck of both birds was bald, presenting here and there stumps of feathers, but was not inflamed. Microscopical examination of the stumps of feathers showed spores and fragments of mycelium. The spores were irregular in shape and size, and seemed to be less resistant than those of ordinary ringworm, breaking up readily in liquor potassæ, so that it was difficult to obtain satisfactory specimens. Attempts to cultivate the fungus were unsuccessful.

Disseminated Gangrene of the Skin in Children.—VEILLON and HALLÉ (*Annales de Dermatologie et de Syphiligraphie*, 1901, No. 5) report a case of multiple gangrene of the skin occurring in a child, aged eighteen months, following an attack of measles. The measles presented nothing peculiar, the attack being of ordinary severity; but four days after the appearance of the eruption lesions filled with a bloody fluid appeared upon the back of the neck, which were soon transformed into variously-sized, round and oval ulcers involving the entire thickness of the skin. These gangrenous ulcers rapidly extended, in some instances destroying large areas of skin. Besides these ulcers impetiginous, ecthymatous, and phlegmonous lesions occurred upon the forehead, scalp, and hand. Marked elevation of temperature and dyspnoea accompanied the disease, which terminated fatally at the end of two weeks. Bacteriological examination of the pus from a number of the lesions showed the presence of the staphylococcus aureus and an anaërobic bacillus previously described by one of the authors as the bacillus ramosus; microscopical examination of sections of the gangrenous tissue showed the same bacillus. The authors conclude that their case shows that disseminated gangrene of the skin in children is not an isolated affec-

tion, but that, clinically and pathogenically, it is a disease which should be placed in the class of true gangrenes, which bacteriology shows are due to the growth in the tissues of strictly anaërobic micro-organisms.

Multiple Ulcers of the Hands Due to Occupation.—HALL (*British Journal of Dermatology* June, 1901) reports the following case: A man, aged sixty-two years, suffered from an eruption of the hands of ten years' duration, characterized by hyperæmia, roughness, and branny desquamation; cracks and fissures also occurred about the knuckles and on the backs of the hands. The chief feature, however, was the presence of isolated ulcers on various parts of the fingers, which appeared from time to time, healing up after some weeks' duration or enlarging without any attempt at healing. These ulcers varied in size from a split pea to a sixpence, and were painful. The patient was employed in polishing electro-plate by means of finely powdered lime mixed with a small quantity of olive oil, which, getting into the fissures in the skin, produced ulceration. When measures were taken to prevent this the ulcers speedily healed.

BROCQ and LAUBRY (*Annales de Dermatologie et de Syphiligraphie*, 1901, No. 4) describe a form of ulceration occurring upon the hands of those employed in dyeing skins, vulgarly called "*le pigeonneau*." The affection is characterized by the presence of variously sized, round or oval, punched-out, quite deep ulcers situated upon the ends of the fingers and the dorsal surface of the left thumb. The pain accompanying the affection is usually quite severe, often causing the patient to cry out. The ulcerations take their origin in slight excoriations or fissures arising from any cause, and continue so long as the patient continues at his work, slowly extending in circumference and depth. Withdrawal from work is followed by healing of the ulcerations. The authors attribute the malady to the action of the irritating and mildly caustic substances contained in the various fluids employed in preparing the skins for dyeing.

Phlebitis Nodularis Necrotisans.—PHILIPPSON (*Archiv für Dermatologie und Syphilis*, Band lv., Heft 2) under the above title reports a case which exhibited lesions corresponding clinically with the so-called tuberculides of Darier. The patient, a girl, aged eighteen years, two years before coming under Philippson's observation, had had an eruption of painful pustules upon the legs, which after a time became ulcers and healed very slowly. When first seen there were a number of rounded ulcers surrounded by a marked halo, irregularly distributed over the legs, the bottoms covered with necrotic tissue. These began as inflammatory spots, which after a week or two became necrotic in the centre, forming ulcers which slowly spread peripherally. A few firm, bluish nodules were also observed on the heel and outer side of the foot. Microscopical examination of excised lesions showed that the process was an inflammatory one arising in the bloodvessels, the most marked changes being in the branches of the venous network which lies in the fat-tissue beneath the cutis. The author concludes from the study of this case that the tuberculides begin as a phlebitis.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

CHARLES HARRINGTON, M.D.,

ASSISTANT PROFESSOR OF HYGIENE, HARVARD MEDICAL SCHOOL,

AND

EDWARD F. WILLOUGHBY, M.D.,

OF LONDON.

Influence of Ozone on Bacteria.—In view of the inconsistent results arrived at by a number of experimenters on the bactericidal influence of ozone, DR. ARTHUR RANSOME and MR. A. G. R. FOULERTON (*Public Health*, July, 1901, p. 684) undertook to investigate the subject anew, in the hope of coming to a definite conclusion with especial reference to the availability of this agent as a room disinfectant. The experiments were planned to ascertain whether ozone applied in large quantities, mixed with atmospheric air or pure oxygen, has a true bactericidal influence, and included tests of its action on the vitality of certain pathogenic and saprophytic bacteria and on the virulence of one pathogenic species (*B. tuberculosis*). The organisms employed in the several experiments included the following: *B. tuberculosis*, *B. mallei*, *B. diphtheriæ*, *B. anthracis* (sporing), *B. typhosus*, *micr. melitensis*, *micr. candidans*, *B. coli communis*, *B. pyocyaneus*, *B. pneumoniæ* (Friedländer), *B. prodigiosus*, *staph. pyogenes aureus*, *str. pyogenes*, *saccharomyces albicans*, *sarcina ventriculi*, and an anaërobic sporing butyric-acid-forming bacillus. The results of the experiments demonstrate that dry ozone has no appreciable action on the vitality of these organisms; that prolonged exposure does not diminish the pathogenic virulence of *B. tuberculosis* (in sputum), *B. mallei*, or *B. anthracis*; that ozone passed through a fluid medium containing bacteria has bactericidal power; "that any purifying action which ozone may have in the economy of nature is due to the direct chemical oxidation of putrescible matter, and that it does not in any way hinder the action of bacteria, which latter are, indeed, in their own way, working toward the same end as the ozone itself in resolving dead organic matter to simple non-putrescible substances."

Frequency of Trichinosis in the United States.—It is commonly stated that 1 to 2 per cent. of dissecting-room subjects contain trichinæ, but most of the observations upon which this is based fail to state that systematic microscopical examinations of all cadavers were made. Believing that trichinosis occurs more commonly than is generally thought to be the case, DR. HERBERT U. WILLIAMS (*Journal of Medical Research*, July, 1901, p. 64) made an extended investigation, in the course of which 505 cadavers, not selected as being more likely to be infested with trichinæ than any others, but taken at random, were examined. The proportion of cases which yielded positive results was much larger than was anticipated, being

no less than 5.34 per cent. In none of the cases was the infection very recent, as shown by encapsulation and calcification, and not one of the subjects died of trichinosis. The infections were of varying degrees of severity: sometimes very extensive and sometimes very slight. The birthplaces of the subjects included the most important countries of North America and Europe, but the number of cases was not enough to admit of accurate conclusions as to the influence of nationality upon the frequency of the disease. It is concluded that many cases of old trichinosis escape detection at autopsies, and that a large part of the published statistics are probably based on the naked-eye diagnosis, which will detect only the very severe infections.

Influence of Formaldehyde upon the Metabolism of Children.—Formaldehyde has come into more or less extensive use as a milk preservative, though it does not lend itself for the purposes of a general food preservative on account of its hardening influence upon certain tissues. As is the case with boric acid and borax, there is a decided conflict of testimony as to its effect upon the consumer; and since, in the absence of direct observations upon man, no conclusions can be drawn from experiments with young animals concerning the possible effects of small doses upon him, TUNNICLIFFE and ROSENHEIM (*Journal of Hygiene*, July, 1901, p. 321) concluded that the only way to gain information on this point was to make a series of metabolic experiments similar to those made in their investigations concerning the influence of boric acid and borax. These led to the following general conclusions: In doses up to 1:5000 in milk or 1:9000 in the total food and drink, formaldehyde exerts in healthy children no appreciable effects on the nitrogen or phosphorus metabolism or fat assimilation, but in larger doses or long continued it might tend to diminish phosphorus and fat assimilation, this effect being referable to an influence upon pancreatic digestion. In the above doses in healthy children it produces a retention of water in the body. In a delicate child in the above maximum doses it has a chemically measurable deleterious effect upon the nitrogen, phosphorus, and fat assimilation, again referable to an action upon the pancreatic digestion, combined with a slight intestinal irritant action, and increases the volume of urine and weight of feces. The excretion of lecithin in the feces is diminished, this effect being probably due to a stimulating action on the lecithin-splitting ferment of the pancreas. It exerts no appreciable intestinal antiseptic action, and has no influence upon the general health or well-being of children.

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THE
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FEBRUARY, 1902.

AFFECTIONS OF THE MOUTH AND THROAT ASSOCIATED
WITH THE FUSIFORM BACILLUS AND
SPIRILLUM OF VINCENT.¹

BY EMIL MAYER, M.D.,
OF NEW YORK,

SURGEON TO THE NEW YORK EYE AND EAR INFIRMARY, THROAT DEPARTMENT; FELLOW OF THE
AMERICAN LARYNGOLOGICAL ASSOCIATION, OF THE ACADEMY OF MEDICINE,
OF THE AMERICAN MEDICAL ASSOCIATION, ETC.

In 1896, in an article on hospital gangrene,² Vincent, having found a fusiform bacillus and spirochetæ in his cases, stated that the same bacillus and spirillum could be found in certain anginas of an ulcerative type. In 1897, a year later, Bernheim³ published his researches in thirty cases of angina and stomatitis, in all of which a fusiform bacillus and spirillum were found. In 1898 Vincent reported fourteen further cases.⁴ Bernheim's researches were evidently independently made, and some discussion has arisen as to the priority of their discovery as between these two. Tarassiewicz⁵ states that he has seen the report of the Children's Hospital of the Duke of Oldenbourg, in St. Petersburg, 1893, by Dr. Rauchfus. The latter presented cases of ulcero-membranous angina with sharpened bacilli and spirilla, and the photographs prove their identity to those of Vincent. According to Tarassiewicz the credit of the priority belongs to Rauchfus.

Be the credit of priority wherever it may, these anginas are referred to by all writers with the name of the bacillus of Vincent attached. Recently, Niclot and Marotte⁶ have presented an exhaustive article on

¹ Read before the Section on Laryngology of the New York Academy of Medicine, November 27, 1901.

² *Annales de l'Institut Pasteur*, 1896, p. 488.

³ *Bull. de la Soc. des Hôpitaux*, March 11, 1898.

⁴ *Ann. Arch. f. Bak. and Path.*, 1899, p. 412.

⁵ *Deutsche med. Wochenschr.*, 1897.

⁶ *Revue de Médecine*, April 10, 1901.

this subject, with a reference list of sixty different papers. It is rather remarkable, in view of all this, that no cases of the kind have been thus far recorded in this country.

Were it simply the announcement of the existence of another bacillus, this might well be relegated to the laboratory or receive but casual mention as an existing fact; one of the interesting novelties found.

The difficulties in the way of diagnosis in membranous and ulcerative lesions of the mouth, however, and the grave importance of the need or not of subsequent treatment, bring the matter urgently before us and make it of vital interest.

My own experience relates to but a single case, but it presents the subject for careful study.

Male, aged twenty-three years, called to see me on September 28, 1901, with the statement that he had had a sore-throat for the past two weeks, which, while not interfering with his business, was daily getting

FIG. 1.



P.H.E..

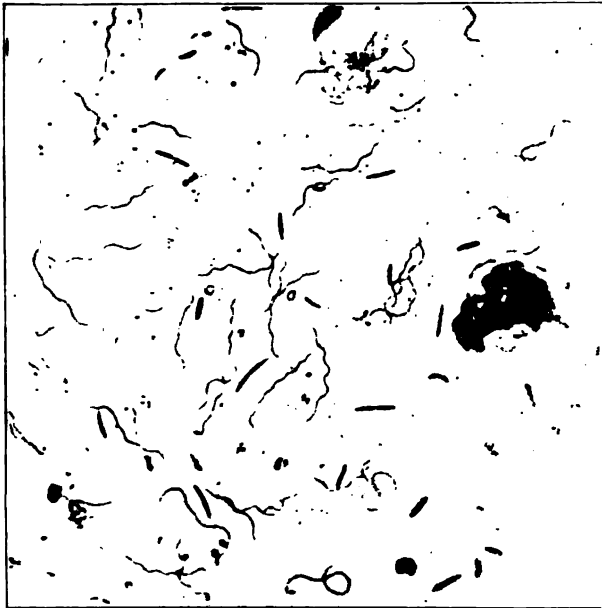
Dr. Mayer's case of ulcero-membranous angina, with the fusiform bacillus and spirillum of Vincent, showing the tonsillar and palatal appearance.

worse. He had seen his family physician, who referred him to me. He was more or less subject to sore-throat, which always yielded to gargles of chlorate of potash. His health had always been good, except for scarlet fever, measles, and pertussis in childhood. His father had died at the age of sixty-eight of pneumonia, a sister at the age of seventeen of the same illness, and two young children, a brother and a sister, in infancy. His mother and a brother are living, and the family in all the collateral branches are healthy. His habits are excellent; he belongs to the higher walks of life; he is a moderate smoker; is tall, athletic, and of fine physique, and has never had any venereal disease, nor has he been exposed to any infected person so far as he knows.

The pain is of a rather mild type, salivation is present, and glands at the angle of the jaw are slightly swollen. He prefers to swallow soft foods; the effort to swallow solids is too painful to persist in so doing.

Examination. Right tonsil is the seat of three separate spots of white deposits, irregular in shape, reddened about the edges, easily detached, having an ulcerated and bleeding surface underneath. Between the membranous deposits is normal tonsillar tissue. Two similar deposits exist on the left tonsil. Although the membrane was readily detached, crumbling even when touched, it re-formed very promptly. A few days later a few spots showed to the left of the uvula, and subsequently

FIG. 2.



Showing the fusiform bacillus and spirillum of Vincent from Dr. Mayer's case.

all the spots joined into a single mass of a pearly white color, and there was a line of deposit over the gums of both upper and lower teeth.

A specimen had been removed for microscopical examination and the bacillus and spirillum of Vincent found. In order that the bacteriological examination should be free from possible extraneous additions, the patient was sent to the laboratory, where spreads were made by the bacteriologist, whose report is here appended.

THE MOUNT SINAI HOSPITAL, PATHOLOGICAL DEPARTMENT,
NEW YORK, November 7, 1901.

DEAR DR. MAYER: The results of the examinations in your case are as follows:

1. The small piece cut from the tonsil on September 30th showed areas of necrosis and areas of inflammation, with suppuration. In some

parts there were masses of fusiform bacilli, apparently the same later found in spreads from the affected areas. The areas of necrosis were quite granular in appearance, there being an occasional small, round nucleus present.

2. The spreads made by myself from scrapings of the surface of the ulcerations showed the same appearance on both days on which they were made (October 4th and 5th).

Of cellular elements there were present mainly polymorphonuclear neutrophilic leucocytes and a few small mononuclear cells, with a round, comparatively large nucleus and no granulations.

In the spreads were found numerous bacilli and spirochetæ, and very few short chains of streptococci. The bacilli varied in length from 4 to 12 μ . They were non-motile. Some were completely decolorized by the Gram procedure, some partly, and some not at all. The bacilli were fairly thick; some were thicker at one pole than at the other; many were curved, some quite irregularly. Most were pointed at both ends; a fair number were swollen in the centre. They occurred as diplobacilli, in small masses or lying at angles to each other in groups, somewhat similar to the arrangement of diphtheria bacilli.

They stained well with the usual basic stains, but were best seen when diluted carbol-fuchsin was used. Often clear places were seen in the bacilli, but no spores could be found.

The spirochetæ were rather long and were distributed irregularly. They were non-motile. They were decolorized completely by the Gram procedure. Staining reactions same as for the bacillus.

Cultures from the secretions of the ulcers in ordinary media, aërobically and anaërobically, showed streptococci only. Inoculations were also made on glucose-serum media, both fluid and solid,¹ and on the ordinary serum-bouillon and serum-agar. They were grown aërobically and anaërobically. Only streptococci were found. There was no increase in bacilli or spirochetæ in the condensation water in the serum-agar tubes.

The findings in this case are the same as those made in a number of cases of ulceration in various parts of the mouth by Vincent and later by Bernheim. The only difference present is that the bacteriæ in your case were not motile, whereas the spirochetæ have generally been found quite motile. Reports as to the motility of the bacilli vary. The picture, however, is so characteristic that the difference is of no moment.

As to the culture experiments: Niclot and Marotte² used serum-media of the same kinds that I did (none, however, with glucose). They succeeded in some of their cases only in obtaining results either in fluid media or in the condensation water of the solid media. They never obtained pure cultures; streptococci were always present.

I made a few tests with the 1 per cent. acetic-acid bouillon recommended by Silberschmidt,³ but all the tubes remained sterile.

The time of observation of all culture tubes was four days.

Very truly yours,

E. LIBMAN.

The treatment instituted was frequent gargles of boric-acid solution and daily application of tr. iodin. comp. For nearly two weeks the

¹ See Libman, *Journal of Medical Research*, vol. i. p. 84.

² *Revue de Médecine*, 1901, No. 4.

³ *Centralblatt für Bakteriologie*, Band xxx., No. 4.

deposit seemed to remain, when it gradually began to disappear, and on October 25th, four weeks after I first saw him, there was no sign of any deposit, the patient felt well and has remained so.

There is a varied nomenclature regarding this affection. Vincent himself and subsequently Freyche¹ use the term diphtheroid angina in mentioning the condition. Panoff² and Marian³ speak of it as chancriform angina. Bonnus and Deguy⁴ call it ulcero-membranous angina; Athanasiu,⁵ merely ulcerous angina; while Salomon,⁶ in a recent article, defies the translator and, in the happy manner of word-building so potent and so beautiful in the German language, calls it "spirochäten-bazillenangina."

This affection seems to have a predilection for the adult male, and it is said to be due to the use of tobacco, eruption of a wisdom tooth, defective teeth or those covered with tartar, lymphoid tendency, syphilis, and mercurial stomatitis. It is mainly an affection of the tonsils, but also occurs as a stomatitis. It is generally conceded that the presence of the bacillus of Vincent precludes the existence of diphtheria bacillus, de Stoecklin⁷ having pretty well established that fact. Because of our inability to inoculate with or to produce pure cultures of these bacilli and spirochetæ, we are not prepared to say that these are the sole controlling factors in the etiology of this affection, hence the title of this article reads "associated with" rather than "caused by" them.

These bacilli may readily be associated with or engrafted upon a syphilitic soil, Salomon⁸ having recorded two cases in which the bacillus and spirillum were found. After a time the characteristic deposits ceased, and with it the microscopical findings and undoubted mucous patches appeared.

The symptoms present are of the slightest kind, as a rule, the deposit existing for quite a time without being noticed. The lesion has a distinctly chancriform appearance at first, and, while usually situated on the centre of the tonsil, it is often on its most dependent portion. The deposit is white, soft on top, readily detachable, and leaving an excavated and bloody surface. There is a certain amount of induration, the breath has a peculiar fetid odor, salivation is present, submaxillary glands are infiltrated. There is slight pain in deglutition. In a case recorded by C. Nicolle⁹ there was an ulceration on the lip similar to those on the tonsils, and on the upper part of the face there were several cedematous spots, painful to the touch, with erythema and

¹ Thèse de Toulouse, 1899.

² Nord. Medical, March 1, 1900.

³ Soc. de Ped., June 12, 1900.

⁴ Centralblatt f. Bakteriologie, 1898, No. 17.

⁵ Loc. cit.

⁶ Thèse de Nancy, 1899.

⁷ Journal des Praticiens, May 19, 1900.

⁸ Deutsche med. Wochenschr., No. 34, August, 1901.

⁹ Normandie Med., June 1, 1899.

discoloration of the skin. Nielot and Marotte¹ mention a complication of appendicitis in one and pseudo-rheumatism in another.

The borders of the gums, the lips, and tongue are the principal seats of this affection in the mouth. The duration of the affection seems to be from eight days to four or six weeks. The prognosis is, as a rule, very good, but there is a tendency to recurrence. Like all other infectious maladies, it is susceptible of complications of a various and sometimes serious nature. One fatal case is recorded where there was an ulcero-membranous stomatitis complicated by infectious purpura. Endocarditis may follow the condition.

The treatment consists in the use of boric-acid solution as a gargle, the local application of iodine and of peroxide of hydrogen.

Of utmost importance is the question of diagnosis, the two most important conditions differentiated being diphtheria and syphilis. In the former the exposure to contagion, the presence of temperature, cardiac depression and albuminuria will serve to differentiate, but in both the bacteriological diagnosis must be resorted to as definitely settling the matter.

Clinically, this affection is exceedingly difficult to differentiate from any one of the forms of specific disease, and hence the importance of its careful study. This becomes necessary not only for the patient's future welfare, but also for his present needs, for it is a well-known fact that mercury in any form interferes with the healing of an ordinary stomatitis.

It must also be borne in mind that syphilis may be present, and we may have a membranous deposit with the fusiform bacillus and spirillum associated with an underlying syphilis, and here the question arises as to what treatment should be instituted. To this I would answer, treat the angina first, and eliminate that, incidentally, if necessary, giving iodide of potassium internally until the angina is entirely healed.

In differentiating from a primary lesion or tonsillar chancre, the question of induration can hardly help us, for the reason that palpation in this region is almost a physical impossibility. The presence of cervical glands in syphilis may be of some help, as will also the history of exposure.

The duration of both may be long. In the secondary conditions the ulceration is much more apt to be deep and continuous. In the tertiary form gummatous lesions are more apt to be prevalent, and the history by this time has probably been sufficiently clear; but, under all circumstances, the final decision as to the presence of these bacilli and spirilla must depend upon the results of the bacteriological examination. Without the laboratory many an unfortunate would be

¹ Loc. cit.

promptly placed upon anti-syphilitic treatment, doomed to take quantities of drugs for years and years, fearful lest at any time there might be an outbreak of a disease he never had; if unmarried, always doubting his right to take such a step, and if married, shunning those near and dear to him.

It is surely worth our while to make haste slowly, and to examine every single case of membranous sore-throat with all the means at our command, or, if not every case, surely those whose history is not clear and where there may be the least doubt.

[The gross illustration is made from a sketch kindly drawn by Dr. Percy Fridenberg. The photomicrograph (Fig. 2) shows the bacilli and spirilla very clearly.]

A CASE OF ACUTE CHOLECYSTITIS WITH GANGRENE; CHOLECYSTECTOMY; RECOVERY.¹

BY FRANCIS D. DONOGHUE, M.D.,
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THE report of the following case of cholecystitis with gangrene is of interest as showing the rapidity with which a case of quiescent cholelithiasis may be converted into an acute fulminating condition, requiring the prompt operative treatment which should be given to all cases of severe perforative peritonitis, and goes to prove the statement "that cholelithiasis is a disease which becomes dangerous through cholecystitis or its consequences."

Julia E., residing in Salisbury, Mass., aged fifty years, married, and the mother of six children, was taken with a severe pain in the region of the gall-bladder on Sunday, May 5, 1901. It began about 11.30 P. M., waking her from a sound sleep. Pain lasted for twelve hours. She vomited during the night, and the vomiting continued until 3 P. M. Monday. Monday night she felt so much easier that she went home on the electric car, a distance of three miles. Tuesday she felt sick, but there was no vomiting or severe pain. There was a good deal of tenderness over the upper abdomen, and a bunch was discovered under the ribs. Tuesday evening she called Dr. Spaulding, who found her with a temperature of 102.5° F., a pulse of 100, and on palpation discovered an elongated tumor extending below the edge of the liver, very sensitive on pressure. The next morning she was taken to the Anna Jacques Hospital in Newburyport.

On Wednesday P. M., May 8th, I saw her in consultation with Dr. Spaulding and Dr. Abby Noyes Little. Found a fairly stout, strong, well-nourished woman, giving the following history: Had usual dis-

¹ Read before the Suffolk District Medical Society, Surgical Section, December 4, 1901.

eases of childhood, including scarlet fever and diphtheria. As a child was subject to sick headaches. After the age of thirteen years was never seriously ill until last fall, when she had an attack of "malarial fever," as she called it. Had chills and fever for six weeks, but there was no pain or stomach or bowel disturbance. "Years ago" would occasionally have sudden severe attacks of pain across the upper abdomen. They lasted from three hours to half a day, and were relieved by vomiting. Never was jaundiced and never called a physician. She has been free from such attacks for the past ten years. Six or seven years ago had a "nervous attack" lasting eight weeks. For three or four years has had by spells a sense of pressure and some distress in region of gall-bladder, especially after exertion. Menopause a year ago, accompanied by indefinite nervous symptoms. Examination of abdomen showed in right hypochondrium an elongated tumor extending about three fingers' breadth below outer border of ribs and lying under the right rectus muscle. It was dull on percussion, sensitive on pressure, and moved slightly with respiration. The remainder of the abdomen, as well as the heart, lungs, and urine, presented nothing abnormal. Stool following enema was of fairly normal color and formed. Temperature was 103.5° F.; pulse, 130. There was no icterus of the skin or of the sclera, and the liver did not appear to be enlarged.

Operation at 5 P. M., under ether, Dr. Little assisting. The incision through the outer border of the right rectus muscle opened directly onto the gall-bladder, which was found tense, distended, and apparently full of calculi. Its surface was smooth except for some fibrin upon the inner end, and there were no adhesions. At the outer end was a very thin and apparently necrotic spot. The bile-ducts were palpated, and no obstruction other than in the cystic duct could be made out. The gall-bladder was freed from its "liver bed," the duct below the obstruction was clamped, a strong silk ligature was tied, and the gall-bladder cut between the clamp and tie and removed. Two gauze drains were passed down to the stump and the incision closed by interrupted catgut sutures of rectus fascia, muscle and peritoneum, except at upper angle, out of which the gauze drain and end of silk ligature were brought. Skin closed by interrupted silkworm-gut sutures.

For the subsequent history I am indebted to Dr. Little. Bowels moved on the second day. First dressing on fourth day; wound clean; no discharge. Sat up on the fourteenth day. Ligature came away on the sixteenth, and she was discharged on the twenty-third day, with the wound entirely closed. On the tenth and eleventh days the temperature ran up to 101° F., accompanying an attack of facial neuralgia. There was no trouble in the wound.

Dr. Spaulding writes me under date of December 2d as follows: Mrs. E. is in perfect health, and looks it. She is at work nursing; eats anything and everything, and suffers no distress.

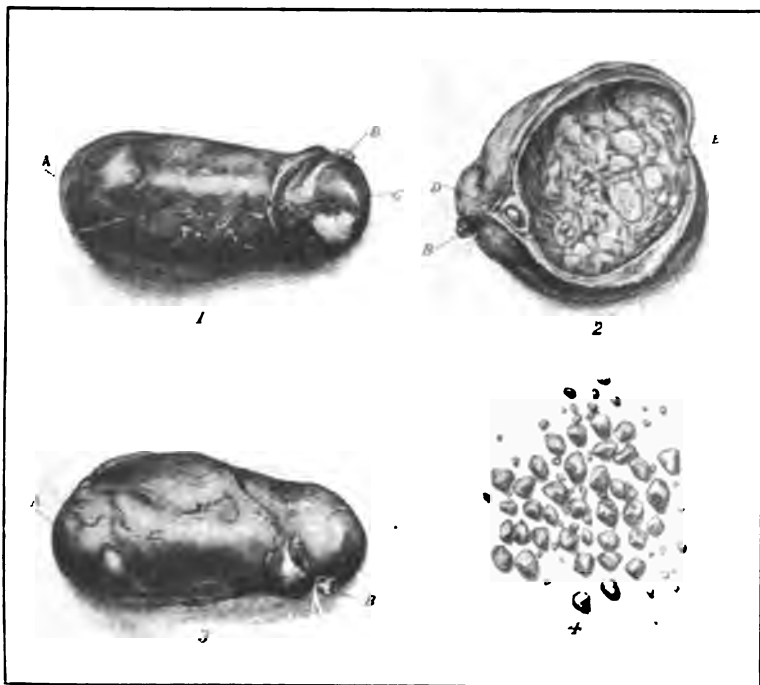
Report of Dr. W. F. Whitney on Specimens.

Gall-bladder distended to twice natural size. Walls hemorrhagic and dark red in color externally. Internally, mucous membrane deeply ulcerated in places, as if from pressure. Where this is not the case it is intensely injected. Summit (or apex) of gall-bladder shows a dark green discoloration, as if gangrenous. Cystic duct blocked by

small calculus. Contents of gall-bladder: mucopurulent secretion and large number of calculi.

Diagnosis. Acute cholecystitis, with beginning gangrene.

Cholecystectomy in this case seemed to be the operation of choice, as it should be in most cases where there is gangrene or necrosis of the mucous membrane or of the gall-bladder wall. It removes immediately the major part of the infection and does away with the possibility of a recurrence, and obviates the possibility that even after a successful cholecystotomy distress may again occur because of extensive adhesive processes.



The absence of icterus, clay-colored stools, or stones in the feces was noted in this case, and in this connection the conclusions of Professor Kehr, of Halberstadt, from an analysis of 535 laparotomies for gallstone disease, are interesting. He believes "that icterus is wanting almost always in an inflammatory process of the gall-bladder and in the beginning of gallstone disease. The passage of the stone in the feces is not so common as it has hitherto been considered, for most colics are unsuccessful; that is, the stone remains in the gall-bladder. Finally, it is time that the physician should give up the view that icterus belongs to cholelithiasis, as in 80 per cent. of all gallstone cases it is absent."

PHRENIC NERVE INJURIES. REPORT OF A CASE. ANATOMICAL AND EXPERIMENTAL RESEARCHES, AND CRITICAL REVIEW OF THE LITERATURE.*

By W. E. SCHROEDER, M.D.,

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WITH

ANATOMICAL OBSERVATIONS ON THE INNERVATION OF THE DIAPHRAGM.

By F. R. GREEN, A.M., M.D.,

ASSISTANT DEMONSTRATOR OF ANATOMY, NORTHWESTERN UNIVERSITY.

IN a careful examination of the literature on this subject, one finds that most authorities agree that a division of the phrenic nerve or any injury to it is fatal, or certainly very dangerous. I was led to doubt the accuracy of this conclusion, first, by a case of paralysis of the right half of the diaphragm, complicated by a diaphragmatic hernia following an anterior poliomyelitis which I saw in 1892; secondly, by Dr. Murphy's nitrogen gas method of collapsing one lung for tuberculosis; and thirdly, by cases of recovery from surgical pneumothorax, which made me think that the worst that could happen in injury to the phrenic nerve would be a paralysis of one-half of the diaphragm leading to a partial collapse of one lung only.

My skepticism led me to take liberties with the nerve, so that recently I have pinched it systematically every time it has been exposed during a tubercular gland operation, after Fenger's² method, and when, during an operation for a tumor of the neck, it was found impossible to remove it without dividing the nerve, I did so unhesitatingly.

I am indebted to Dr. J. Eskridge for this interesting case. It was operated upon at the Provident Hospital, August 30, 1900.

The patient was a widower, white, aged sixty-two years; American, an upholsterer by occupation.

The family history was good, with the exception that two cousins on the mother's side died of cancer of the uterus, and two sisters of heart disease. Father died of pneumonia at the age of eighty-seven.

Patient had been a heavy whiskey drinker until nine years ago, and he still smokes and chews a great deal. He had the usual diseases of childhood, but no others. Twelve years ago his lower jaw was fractured in a railway accident, and eight years ago he was cut in the right supraorbital region by an ax, which resulted in a paresis of the orbicularis palpebrarum muscle. There were no congenital deformities.

The present trouble began nine years ago, when the patient noticed a tumor on the left side of the neck about as large as a hazelnut, but painless. This increased in size very slowly during the first four years,

* Read before the Chicago Medical Society, March 18, 1901.

at the end of which time it was about as large as a walnut and painful upon pressure. During the last five years the tumor has grown somewhat more rapidly and has become more painful, and the pain has been constant during the last year.

The tumor is situated on the left side of the neck immediately behind the sternocleidomastoid muscle and partly covered by it. (See Fig. 1.) It begins three-fourths of an inch above the clavicle and extends upward two and one-half inches. It is two and one-half inches long in the long diameter, which is parallel with the long axis of the body; its inner margin lies one inch from the left border of the trachea. The skin covering the tumor is normal and freely movable. There are no large vessels to be seen. The tumor feels hard, and is so

FIG. 1.



Before operation.

firmly attached deep down that it cannot be moved. No fluctuation, pulsation, or bruit. No enlarged glands. Aspiration shows, macroscopically, bright red blood in considerable quantity; microscopically, nothing but blood-corpuscles, neither epithelial nor other cells being found. No pressure symptoms.

The physical examination of the patient resulted as follows:

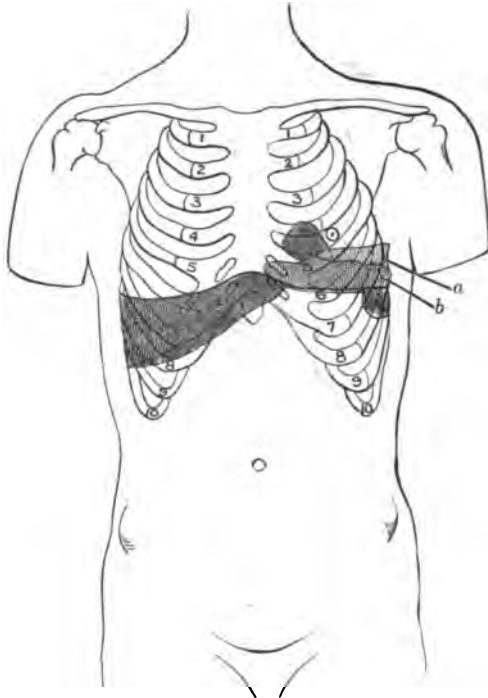
Body well nourished; weight, 160 pounds; no recent loss in weight; no abnormality of skin or glandular system. Pulse, temperature, and respiration normal; appetite good; walls of arteries soft; no symptoms of pressure on sympathetic nerves. Paresis of orbicularis palpebrarum due to old injury. Heart and lungs normal. Special attention was paid to the lungs and the position of the diaphragm. Liver,

spleen, etc., normal. Urine and blood count normal. Patient was prepared for operation by usual method.

An incision was made from the posterior edge of sternocleidomastoid muscle one inch below the mastoid process to the clavicle. The external jugular vein was ligated and divided between the ligatures. Continuing the dissection, the sternocleidomastoid muscle was liberated and the spinal accessory nerve found and dissected out to the trapezius muscle.

The tumor came into view and showed a capsule which was not adherent to any great extent to the surrounding tissues, but was firmly

FIG. 2.*



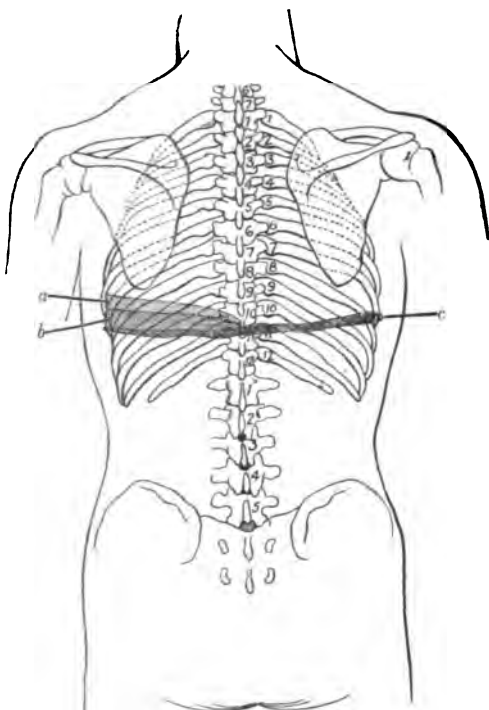
Anterior view. a. Relative dullness. b. Absolute dullness.

attached deep down. It was at once noticed that the phrenic nerve lay over the upper outer part of it and that its lower branch was but slightly visible. The nerve was quite tense, but responded twice when it was pinched. As I believed the tumor to be of a malignant nature, I did not wish to divide it for fear of implanting cells in the wound. Therefore, I tried to dissect the nerve from the tumor, but in doing so the small branch from the fifth cervical ruptured, and soon after the main branch from the fourth cervical did likewise.

* Figs. 2, 3, and 4: Findings upon percussion after division of nerve. a. Relative dullness. b. Absolute dullness.

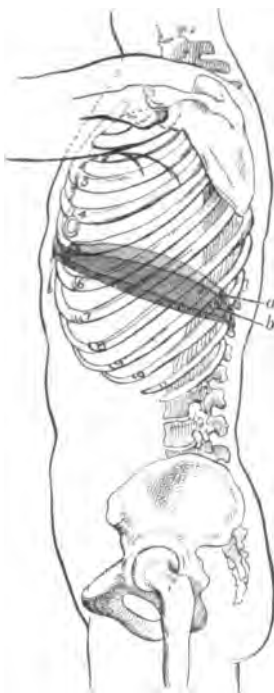
I noticed no material change in the patient excepting an increase in the number of respirations to thirty-two. The tumor was so situated that the upper and main root of the phrenic traversed the upper outer part of it, while the lower root came from underneath. It was firmly attached to the foramen formed by the third and fourth cervical vertebrae, and its removal caused a venous hemorrhage from this point. The fat and various glands were removed from the side of the neck. I followed the phrenic down to the clavicle, injuring the thyroid axis, which produced some hemorrhage. No other branches of the phrenic were found. In trying to unite the divided parts of the nerve, it was

FIG. 3.



Posterior view. a. Relative dulness. b. Absolute dulness. c. Normal side.

FIG. 4.



Lateral view. a. Relative dulness. b. Absolute dulness.

impossible to find the little branch from the fifth cervical, but the ragged end of the trunk and that of the fourth root were cut off in order to give uninjured ends, and reunited by means of silk sutures. The nerve was now pinched below the suture, and responded. There was no cough, no singultus, no sneezing, none of the symptoms reported in other cases. The wound was closed in the usual manner. After the patient recovered consciousness, I asked him whether he had any difficulty in breathing or any pain in the region of the diaphragm, but he said he had not. However, the respirations remained more frequent (24 to 30) for four or five days, after which they came down to

20, and remained so. About eight hours after the operation, percussion showed that the left half of the diaphragm was two and one-half inches above its normal position. An area of the lower portion of the lung, an inch and a half in width, showed relative dullness. (See Figs. 2, 3, and 4.) Litten's sign was absent.

Auscultation over dull area showed vesicular breathing rather distant. Slight abdominal retraction on inspiration. During his three weeks' stay in the hospital the position of the diaphragm remained the same, but the area of relative dullness gradually disappeared and the lung returned to its normal condition. Measurement of the thorax showed right half 44 cm. Deep expiration, 41½ cm. Inspiration, 44½. Left half, 43 cm. Deep expiration, 41½ cm. Inspiration, 43½.

At present the patient is employed as usual, and is suffering no inconvenience from his paralysis.

Macroscopical Examination. The tumor is solid but very vascular. It is two inches in its long diameter and an inch and three-fourths in its transverse diameter. Upon cutting the tumor in half the interior presented a solid consistency, but many large vessels were to be seen. A few small areas, 1 cm. square, were somewhat softer than the main body of the tumor.

Microscopical Examination. One sees numerous connective tissue fibres around the periphery, forming a well-defined capsule, and many areas of leucocytes around which there seems to be young connective tissue. In the centre of the tumor there is a hemorrhage into the tissue. This is very recent, and is probably due to the puncture made by the aspiratory syringe. There are many large and small bloodvessels, but no new ones to be seen. Leucocytes are grouped at various places, resembling an inflammatory process. I believe the tumor to be a fibroma and not, as was thought from the clinical evidence, a fibrosarcoma.

Experimental Researches.

1. *Upon the Human Subject.* As before stated, in all recent operations for tubercular glands, I have searched for the phrenic nerve and pinched it below the lower root, with the following results: Each time the corresponding side of the diaphragm contracted, producing a sudden and decided abdominal rising immediately below the costal arch. This has been done in eighteen cases, ten right and eight left. In two cases, one right and one left, there was some pain in the region of the corresponding half of the diaphragm, which subsided in each instance before the end of forty-eight hours. The symptoms commonly ascribed to irritation of the phrenic, *i. e.*, sneezing, coughing, and hic-coughing, were not observed in a *single instance*.

2. *Upon Dogs.* The animal experiments have been made upon dogs only, because they resemble man in their type of respiration, which is costo-abdominal. The method of conducting the experiments has been as follows: The circumference of the thorax was taken at the tip of the ensiform cartilage, and again from one to two and one-half inches above it. The number of respirations per minute were recorded

before and after division of the nerves, and the change in type of respiration noted. Ether was the anæsthetic chosen. The usual antiseptic precautions were observed. After exposing the nerve the interrupted electrical current was applied, and then the nerve was pinched with an artery forceps. The nerve was now either evulsed or resected. Later in this work I weighed the animals before the operation and each week thereafter.

Post-mortems were conducted from one week to four months after the division of nerves. For this purpose in most cases the dogs were deeply anæsthetized with chloroform, the thorax opened, the nerves found and stimulated with the electrical current. The various phenomena were observed, and specimens and drawings made. For the details of these experiments see Table I.

It may not be amiss to state here that before obtaining the results shown in this table, there were eight animals that died, but all of these can be accounted for without detracting in the slightest way from the above results. At first I evulsed the nerve, tearing out from three to five inches of it. Out of four cases only two survived. (See Table I., Cases 1 and 2.) Post-mortems of the two that died showed in one case of double evulsion a double pneumothorax and in the other a pneumopyothorax. In the latter case, I heard the rush of air into the pleural cavity, and, infection occurring, the animal died. Evulsion of the nerve lacerates the pleura, and with an infection is liable to result in death from pneumopyothorax; therefore, I abandoned this method of removing the nerve and resorted to resection outside of the pleura, removing as much of the nerve as was possible in each case.

A third animal died from the anæsthetic before any incision had been made.

Five died as a result of experiments with chloretone as an anæsthetic. These animals were given two-tenths of a gramme per kilo, and operations were begun two hours later. They were not completely under, so they were given two-tenths more per kilo, and operations begun after another hour. By this time they were profoundly anæsthetized, but none of them recovered consciousness. They slept from two to four days, when they died.

Weight. The first seven were not weighed, but my observations would lead me to state that the first week or two they lost in weight, after which they gained and again came back to their normal condition. In the last three it will be seen that they lost in weight for the first week, but came back to normal or nearly so. The loss was not great in any instance.

Measurements. It will be seen that in the single division there was an increased thoracic expansion and usually a slight abdominal retraction, which were more evident on the divided side than on the normal side.

TABLE I.

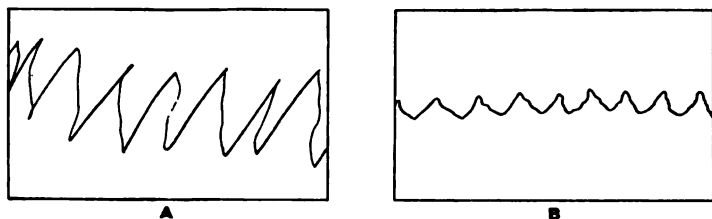
| No. | Date of operation. | Sex, size, and weight. | Measurements in inches. | | | | | | | | Respiration : frequency and type. | | Stimulation of nerve before division. | |
|-----|--------------------|---|-------------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------------------------|---|--|--------------------------------|
| | | | Before operation. | | | | After operation. | | | | | | | |
| | | | At ensi- form. | Above. | At ensi- form. | Above. | At ensi- form. | Above. | At ensi- form. | Above. | Before division. | After division. | Electri- cal cur- rent. | Pinch- ing. |
| | 1900 | | Exp. | Insp. | Exp. | Insp. | Exp. | Insp. | Exp. | Insp. | | | | |
| 1 | Oct. 24 | F. small, | 15 | 15 $\frac{1}{8}$ | 15 $\frac{3}{8}$ | 16 | 15 | 15 $\frac{1}{8}$ | 15 $\frac{3}{8}$ | 16 $\frac{1}{8}$ | All dogs costo-abdominal type. 24 | Costal, abdominal retraction slight. 85 | Tetanic contract'n. | Short well-defined contract'ns |
| 2 | Nov. 5 | M. large, | 25 $\frac{3}{8}$ | 26 | 26 $\frac{1}{4}$ | 27 $\frac{1}{8}$ | 25 $\frac{3}{8}$ | 26 $\frac{1}{4}$ | 26 $\frac{3}{8}$ | 27 | 19 | Pro-nounced costal, inverted. 80 | Tetanic contract'n. | Short well-defined contract'ns |
| 3 | Nov. 24 | M. med' m | 21 | 21 $\frac{1}{4}$ | 22 | 22 $\frac{1}{4}$ | 21 | 21 $\frac{1}{4}$ | 22 | 22 $\frac{1}{8}$ | 23 | Increased costal, abdomen almost passive; no pronounced retract'n. 27 | Tetanic contract'n. | Short well-defined contract'ns |
| 4 | Dec. 3 | F. small, | 14 | 14 $\frac{1}{8}$ | 15 | 15 $\frac{1}{8}$ | 14 | 14 $\frac{1}{8}$ | 15 | 15 $\frac{1}{8}$ | 26 | Decided & somewhat labored costal; inverted. 86 | Tetanic contract'n. | Short well-defined contract'ns |
| 5 | Dec. 3 | M. small, | 21 | 21 $\frac{1}{8}$ | 22 | 22 $\frac{1}{4}$ | 21 | 21 $\frac{1}{8}$ | 22 | 22 $\frac{1}{8}$ | 20 | Costal, slight retraction only. 24 | Tetanic contract'n. | Short well-defined contract'ns |
| 6 | Dec. 7 | M. small, | 20 | 20 $\frac{1}{4}$ | 20 $\frac{1}{2}$ | 20 $\frac{3}{4}$ | 20 | 20 $\frac{1}{2}$ | 20 $\frac{1}{2}$ | 21 | Costal, slight retraction only. 27 | Costal, slight retraction only. 24 | Tetanic contract'n; contr. of left leg also. | Short well-defined contract'ns |
| 7 | 1901 Jan. 23 | M. large, 50 lbs. Feb. 12 51 lbs. Feb. 19 51 lbs. | 30 | 30 $\frac{3}{8}$ | ... | ... | 30 $\frac{1}{4}$ | 31 $\frac{1}{2}$ | ... | ... | 25 | 80 | Tetanic contract'n. | Short well-defined contract'ns |
| 8 | Jan. 23 | M. small, 12 lbs. Feb. 12 10 lbs. Feb. 19 11 lbs. | 13 $\frac{1}{8}$ | 13 $\frac{1}{4}$ | ... | ... | 13 $\frac{1}{8}$ | 13 $\frac{1}{2}$ | ... | ... | 44 | Costal, some abdominal retract'n. 40 | Tetanic contract'n. | Short well-defined contract'ns |
| 9 | Jan. 23 | M. med' m 25 lbs. Feb. 12 24 lbs. Feb. 19 24 lbs. | 19 | 19 $\frac{1}{8}$ | ... | ... | 19 | 19 $\frac{1}{4}$ | ... | ... | 24 | Breath- ing only slightly altered. 26 | Not pro-nounced | Not pro-nounced |
| 10 | Jan. 23 | M. small, 15 lbs. Feb. 12 12 lbs. Feb. 19 15 $\frac{1}{2}$ lbs. | 15 | 15 $\frac{1}{8}$ | ... | ... | 15 | 15 $\frac{1}{2}$ | ... | ... | 30 | Costal, but not pro-nounced. 32 | Tetanic contract'n. | Short contract'ns |

TABLE I.—Continued.

| Division of nerve: right, left, both. | Electrical stimulation before death. | Preliminary to post-mortem. | Postmortem findings. Remarks. |
|---|---|--|---|
| Left, evulsion 5 inches. | Tetanic contraction of right, none of left. | Nov. 14, etherized; an incision was made below ensiform through abdominal wall revealing active contraction of r. half of diaph. and passive of left half; kymographic tracing was taken. See Fig. 5. | Nov. 14. Some atrophy of left half of diaph. crossing the median line; no regeneration of left nerve. Dog was six weeks pregnant; uterus contained five embryos; well nourished. |
| Both, evulsion right $\frac{3}{4}$ in. left $\frac{3}{4}$ in. | April 16. Chloroform was given, thorax was opened, and left nerve found and stimulated; left half of diaphragm contracted slightly, showing partial regeneration; no reaction of right nerve. | Inverted type of respiration. | Marked atrophy of diaphragm, excepting at costal margin; partial arrest of development of abdomen and its contents; no hernia of diaph. Exercised the dog violently Dec. 28, 1900, and Jan. 15, 1901; he did not seem to suffer from labored respiration; still inverted type of respiration; he had been infected. |
| Right, resection 1 inch. | Jan. 17, 1901. Dog was shot and current applied immediately; no reaction of right nerve; normal contraction of left side. | Just before killing the dog, measurements at ensiform on expiration, 21 in.; on inspiration, 21 $\frac{1}{2}$ in.; 2 $\frac{1}{2}$ in. above ensiform, on expiration, 22 in.; on inspiration, 22 $\frac{1}{4}$ in. | Atrophy of right half of diaphragm, which was thin, yellow, semi-transparent; a normal zone $\frac{1}{4}$ inch wide was formed at the periphery; right nerve had regenerated, but the right half of diaphragm did not respond to stimulation. |
| Both, resection 1 $\frac{1}{2}$ inch. | No reaction. | | Dec. 17. Both nerves show some proximal regeneration; atrophy of diaphragm not very decided, because only 14 days passed between the division and post-mortem. |
| Right, resection 1 $\frac{1}{2}$ inch. | No reaction; left normal. | Measurements before killing animal: ensiform, expiration, 21 in.; inspiration, 21 $\frac{1}{2}$ in. | Dec. 15. Some atrophy of diaphragm. |
| Left, resection 1 $\frac{1}{2}$ inch. | Left reaction; right reaction. | Measurements at ensiform, expiration, 21 $\frac{1}{2}$ in.; inspiration, 21 in. | Dec. 14, i. e., 7 days after operation, slight atrophy of left half of diaphragm only. |
| Both, resection 1 $\frac{1}{2}$ inch. | No reaction on either side. | | Feb. 26, 1901. Very decided atrophy of both sides; right side, costal margin, one finds an area which is not atrophic, because it receives a nerve from intercostals. Fig. 6. Costal margin not atrophied. |
| Left, resection 1 inch. | No reaction of left; reaction of right. | | Feb. 19, 1901. Atrophy of left half of diaphragm; none of right. Fig. 7. |
| Both, upper two roots found only. | Reaction of both sides in thorax. | | March 9, 1901. V-shaped portion of diaphragm at ensiform cartilage is atrophied, while the remaining portion is normal. Fig. 8. |
| Right, resection 1 inch. | Reaction in left; none in right. | | Mar. 9, 1901. Atrophy of right half of diaphragm, excepting one centimetre of costal margin. |

In the double divided cases there occurred an inverted type of respiration, *i. e.*, decided abdominal retraction on inspiration, and increased thoracic expansion due to action of accessory respiratory muscles.

FIG. 5.



a. Excursion of right half of diaphragm. b. Passive movement of left or paralyzed half.

FIG. 6.



Resection of both phrenic nerves. Dog No. 7. a. Normal area. b. Non-atrophied margin.

In Case 9, as the upper branches *only* were divided, the animal did not suffer much of a change because the lower branches supply by far the greater part of the diaphragm. (See Fig. 8.)

Electrical Stimulation. In all cases I used the induced current after the Du Bois Raymond method,³ always with a positive result, *i. e.*, tetanic contraction before division and no reaction after the fourteenth day. In Case 6 the animal was killed only seven days after division, and on stimulation there was a slow, feeble contraction, showing either that the nerve was not yet entirely degenerated or that the atrophy of the diaphragm was not yet complete. In Case 9 the current produced a contraction of diaphragm supplied by lower root. (See Fig. 8, *b b.*)

Excursions of Diaphragm. In dog 1, I opened the abdomen in the median line immediately below the ensiform cartilage and inserted a

FIG. 7.



Resection of left phrenic nerve. Dog No. 8. *a.* Non-atrophied margin.
b. Atrophic area. *c.* Esophagus.

glass rod, pushing it against first one side of the diaphragm and then the other, taking a kymographic tracing (see Fig. 5), with the result that the right half contracted normally, producing a rise one-half inch in height on inspiration and corresponding depression on expiration. The left half produced a very small rise, one-eighth inch, representing the influence of the contractions of the right side. There was no contraction of the left half. In other animals, I observed the same phenomena, *i. e.*, that the paralyzed side was passive and only acted upon by the contractions of the normal side.

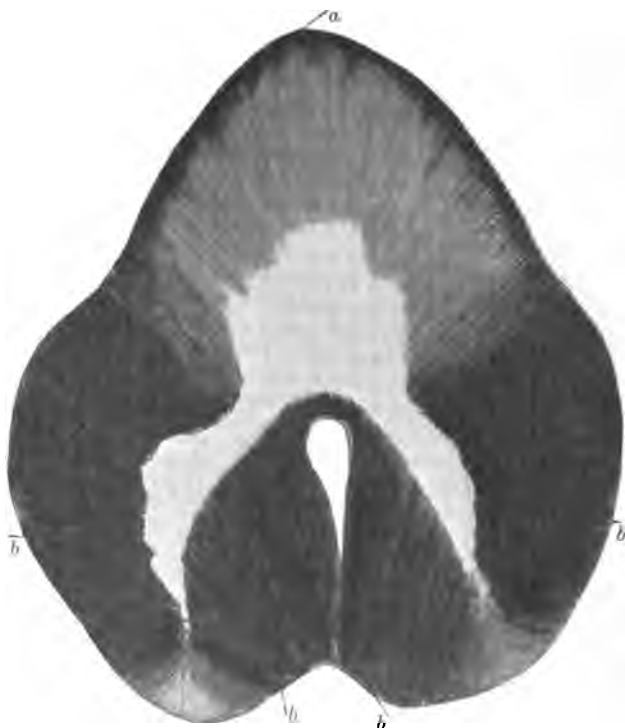
Symptoms. The diaphragm becomes relaxed after the division of the nerve and arches up into the thorax. The type of respiration becomes

increased costal when one nerve only is divided, and inverted when both nerves are divided. The accessory respiratory muscles become very active.

There was no sneezing or coughing in any of the animals. In one case of double division the respiration became labored, but it remained so for two or three days only.

The Post-mortem Findings. Macroscopical. In the cases where the dog was killed from seven to fourteen days after division the atrophy

FIG. 8.



Resection of upper roots. Dog No. 9. a. V-shaped atrophic area. b, b. Normal area. Crura.

was not very great and the color was reddish-yellow. In cases where a long time had elapsed between the operation and the post-mortem, the atrophy was very decided, the paralyzed part was thin and flabby, the diaphragm was pale yellow, and in some of the older cases translucent. In all cases there remained a margin from one-quarter to three-eighths of an inch wide at the costal border, which retained its normal color and thickness. This margin is probably supplied from the intercostal. (See Figs. 2, 3, and 4.) Again, in the specimen taken from dog 7 (Fig. 6, a), it will be seen that there remains a relatively

normal area on the right side, which was probably supplied by a long branch from the intercostal nerve. In all three figures there is a normal area at the crura. These areas evidently receive an independent nerve supply.

In Fig. 8, where the root coming from the upper branch has been divided (dog No. 9), there is a V-shaped area with its apex toward the sternum extending a distance of two and three-quarter inches on each side toward the spine. This is evidently supplied by the upper branches, the lower branch supplying nearly all of the remaining portion of the diaphragm. In dog No. 3, although the nerve had regenerated after fifty-four days, there was no electrical reaction because of atrophy of the diaphragm.

There is very little available literature upon the experimental work on this subject. Sir Astley Cooper⁴ reports having experimented by putting a ligature on the phrenic nerve of a rabbit. The animal suffered from labored respiration and died twenty minutes later. Post-mortem showed that the nerve had been cut.

Henocque and Eloy⁵ reported some experiments in 1883.

Billard and Cavalie,⁶ in 1898, reported on the sequelæ of section of both phrenics in the dog.

REVIEW OF LITERATURE. The literature on this subject is very meagre and confusing, and the conclusions reached by various writers are often diametrically opposite. Thus Gross⁷ says: "The division of the phrenic nerve is nearly instantaneously mortal."

Erichsen,⁸ in his eighth edition, says: "The division of the respiratory nerves on one side only would, in all probability, be fatal in man by interfering with the proper performance of the respiratory act;" but in his tenth edition⁹ he modifies his conclusion, and says: "Division of the phrenic nerve would necessarily, by interfering with respiratory movements, induce a tendency to congestion and inflammation of the lungs; and although such an accident must be a very rare one in cases of ligation of subclavian for axillary aneurism, yet it undoubtedly has occurred as I have myself witnessed in one case."

Ranney¹⁰ says: "Since the phrenic nerve arises in the upper cervical region, any lesion of it will cause respiration to be interfered with and create dyspnoea and hiccup; but respiration will not be arrested, since the pneumogastric nerves continue to excite it, and the auxiliary muscles can expand the chest without the action of the diaphragm. Should the lesion be a surgical one, the respiratory centre of the medulla may be affected and death take place from asphyxia, but I do not think such a result can be explained as a simple effect of paralysis of the phrenic nerves alone."

Hueter-Lossen¹¹ says: "Paralysis of one-half of the diaphragm puts life in immediate danger. This is evident not only from observation

of injuries to the phrenic itself, which are rare, but from injuries to the roots of the nerves in the spinal cord, which determine practically always a fatal termination. These latter may be produced by injuries from the fourth cervical vertebra down, or by degenerative processes spreading from below upward. They are always indicated by paralysis of this principal muscle of respiration, the accessory muscles having been already more or less paralyzed, probably completely. Diaphragmatic paralysis is always easy to determine, since by it alone is inspiration performed under ordinary circumstances."

Diebel¹² calls attention "to the property possessed by nerves and vessels of resisting the encroachments of tumors, just as Langenbeck¹³ had much earlier pointed out in his well-known *Beiträge z. Chir. Path. der Venen*. This is fortunate, since the vagus and phrenic nerves remain apparently uncompromised by a degree of pressure which, *a priori*, it would hardly seem as if they could resist. They permit, moreover, considerable displacement without interference of function."

Brodie¹⁴ says: "If the function of the cord be interrupted above origin of the phrenic nerves, respiration is immediately suspended, and instantaneous death ensues."

Park¹⁵ says: "Cases of unilateral division of the phrenic, or of injury to the course of the phrenic—as, for example, in fractures of the cervical vertebrae—indicate that paralysis of the diaphragm, or at least of one-half of it, puts the patient in immediate danger of life, and also compels the other accessory inspiratory muscles to do an enormous amount of compensatory work."

In a later work Park¹⁶ says: "The section of the phrenic and pneumogastric nerves on one side only is accompanied by respiratory and circulatory irregularities; they are not necessarily fatal, but it is a most serious complication."

Tillmann¹⁷ says: "In case of complete paralysis of the phrenic nerves on both sides, death results immediately from paralysis of respiration, as has been experimentally demonstrated upon animals. Irritation of the phrenic nerve causes continuous coughing and hiccupping in consequence of contraction of the diaphragm."

Later on he says:¹⁸ "After injuries of the subscapular nerve, reflex spasm of the phrenic nerve is sometimes observed with singultus, dyspnœa, and girdle pains."

Gowers¹⁹ says: "If one nerve only is affected, the diaphragm does not descend on that side, but the movements of the other side lessen the resulting defect of movement, and it can then be detected only by close observation. The loss of the action of the diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is said to be readily produced by exertion; the breathing then becomes quick and the voice feeble."

Rose²⁰ and Carless say: "Phrenic nerve division may cause instant death by paralysis of the diaphragm, although where but one nerve is divided the patient can survive. Irritation of the nerve gives rise to spasmodic cough or hiccough."

Medical men have not felt nearly so alarmed at paralysis of the diaphragm as have the surgeons, possibly because the lesion in medical cases comes on slowly, and in this manner patients may adapt themselves to new conditions with less danger. Thus Bernhardt²¹ says: "The prognosis in paralysis of the phrenic nerves and diaphragm is doubtful, to say the least; but, as Duchenne²² has shown, the respiration does not suffer much so long as the patient is quiet because of the action of the accessory muscles, but as one may readily understand, bronchitis and other simple diseases of the respiratory organs become serious in such cases."

Many other statements equally diverse in conclusion may be found in the literature.

TABLE II.

| Case. | Surgeon. | Year. | Nature of injury. | Nerves. | Result. |
|-------|-------------|-------|--|---------|-----------------------|
| 1 | Schürmayer, | 1847 | Stab wound. | Right. | Death in few minutes. |
| 2 | Beck, | 1848 | Bullet wound. | Left. | Death on fifth day. |
| 3 | Bardeleben, | 1882 | Unknown. | | Death in two hours. |
| 4 | Erichsen, | 1884 | Division while ligating subclavian artery. | | Death in few days. |
| 5 | Mackenzie. | 1891 | Rupture from fall. | Right. | Death immediate. |
| 6 | Schroeder, | 1900 | Division during operation. | Left. | Recovery. |

I have been able to find only five cases of actual injury to the phrenic nerve (see Table II.), though there are several others which have been commonly reported as such which, when traced to the original documents, have been found to be erroneous. In some of these the injury to the phrenic nerve is only an assumption on the part of some later writer, who, noticing that the symptoms commonly described as caused by such an injury were present, has inferred that the phrenic nerve must certainly have been injured, while in still others the surgeon has reported an injury to the pneumogastric, but has been cited by others as having injured the phrenic.

The first of the cases of actual injury to the phrenic nerve is that of Schürmayer.²³

Patient was a male, aged twenty-six years, previously well. He was wounded October 16, 1847, by a knife, which penetrated the right side of the neck. The wound began one and one-half inches above the acromial end of the clavicle, passed downward, forward, and inward, ending at the sternal end of the clavicle. In its course the knife-blade

injured the trapezius, splenius capitis, levator scapula, scaleni, and sternocleidomastoid muscles, the fifth cervical, phrenic and branches of the sympathetic plexuses, and the internal jugular vein. Immediately following the injury the patient complained of lameness of the right arm, which hung loosely at his side. Hemorrhage was quite severe. He left the room, going into the kitchen, and after a few complaining words, collapsed and died in the course of a few minutes.

The post-mortem, which was very complete, showed anæmia of vessels in skull and brain, but unusual fulness of vessels in lungs. There were numerous adhesions of the pleura.

Schürmayer says that death was not due to hemorrhage, because of the negative post-mortem findings, but that it was due to division of the phrenic nerve complicated by these numerous adhesions of the lung to the pleura. He believes it probable that cases of unilateral division may recover, and refers to Larrey, but declares that, in the sense of the law, death in this case was due to injury of the phrenic nerve.

The second case, reported by Beck,²⁴ was that of a young man who was shot in the neck during a duel. Upon examination it was found that the bullet had entered two fingers' breadths above the left clavicle at the posterior border of the sternocleidomastoid, and came out on the opposite side at the junction of the upper with the middle third of the neck, anterior to the sternocleidomastoid muscle. The respiration was slow and difficult, pulse weak, pupil of the left eye contracted. On the fifth day he breathed nine to seventeen times per minute. Laryngotomy was performed, during which operation the patient died. Beck performed the post-mortem and found the following: The bullet had contused the left phrenic nerve so that there was an extravasation of blood in the neurilemma, had passed back of the left carotid between the vagus and sympathetic nerves, lacerating the cervical ganglia of the latter, and had then passed through the œsophagus and lacerated the right arytenoid cartilage and the right descendens noni.

In conclusion, Beck states that as the injury to the phrenic nerve resulted in a partial loss of the function of the diaphragm, it certainly complicated a condition.

In some way this case has been attributed to Stromeyer,²⁵ who reported it about 1870, at which time he also reported another case of contusion of the phrenic and brachial plexus with rapid recovery.

In 1882 Bardeleben²⁶ reported a case of division of the pneumogastric and phrenic nerve with no immediate symptoms, but soon the patient's respiration became slow and difficult, and he died in two hours.

In 1884 Erichsen⁶ says: "In a case with which I am acquainted, where the phrenic nerve was divided during a ligation of the subclavian artery, death resulted in a few days from congestion of the lungs."

In 1891 Mackenzie²⁷ reports a case of rupture of the right phrenic nerve with instant death.

In reviewing these five cases it will be seen that in only one, that is the last one, was there an injury to the phrenic nerve alone, while in

all the others there were sufficient injuries to other important structures to account for the unfavorable termination.

In considering the other cases, *i. e.*, those commonly but erroneously considered as phrenic nerve injuries, there are two which deserve special mention, as they have been so widely and frequently quoted, *i. e.*, Bransby Cooper's²⁸ and Hutin's.²⁹

It has been commonly reported that Bransby Cooper, in 1841, while ligating a subclavian artery for aneurism, included the phrenic nerve in the ligature. In reviewing this case carefully, it was found that Cooper states that the patient developed a cough, which persisted more or less violently until his death on the fifteenth day. But the post-mortem, which was incomplete and was done fifteen hours later, showed the phrenic nerve "*in situ* and glued down to the scalenus muscle." Aspiration showed pus in the pleural cavity on the same side.

Hutin's case was operated on in October, 1841, for wound in the axilla. He got up on the sixth day, after which he began to cough. At this time the wound was fetid. On the ninth day he had a slight hemorrhage, and on the tenth day, for fear of a great hemorrhage, Hutin ligated the subclavian artery, but the patient died ten hours later.

Post-mortem showed the artery intact, and Hutin specifically states that no nerves or other important structures were found in the ligature.

Of five cases to which Park¹⁵ refers as injuries of the phrenic nerve, four, three of Demme's³⁰ and one of Riedel's,³¹ were found to be injuries of the pneumogastric, while in the fifth (Hofmohl's)³² the injury was in the cervical plexus below the phrenic nerve.

Referring to the case which came under my observation, one might infer that as the tumor was of slow growth, the gradual pressure upon the nerve had produced a slow paralysis of the diaphragm; but in answer to this, I can say, as Langenbeck¹³ and Diebel¹² have previously shown, that these nerves withstand pressure without much disturbance; secondly, upon pinching the nerve four different times, the epigastric region on the same side showed a sharp sudden rise each time, showing that there had been no paralysis; lastly, the physical examination before the operation showed the diaphragm to be in its normal position and the lungs absolutely normal.

ANATOMICAL RESEARCHES. The subject of the innervation of the diaphragm will be considered under two heads.

1. Innervation from the phrenic nerve. 2. Innervation from any other source.

Under the second heading will be considered: 1. Innervation from the intercostal. 2. Innervation from the sympathetic. 3. Innervation from the pneumogastric.

Innervation from the phrenic nerve. The phrenic nerve or the internal respiratory nerve of Bell is the most conspicuous nerve supplying the diaphragm. It has, by most anatomists, both descriptive and comparative, been considered the sole nerve supply to this muscle. This nerve will be considered under three heads.

1. Its origin: (a) spinal or deep; (b) cervical or superficial. 2. Course and relations. 3. Distribution.

Spinal Origin. Marinesco,³³ in 1898, described a nerve centre in the spinal cord of dogs and rabbits, from which proceeds the nerve fibres of the phrenic nerve.

He describes it as a "group of cells which occupy an intermediate position between the anterior internal and anterior external groups of the anterior horn, extending from the level of the lower border of the fourth cervical vertebra to the lower border of the sixth cervical in dogs, and a little lower down in rabbits. In rabbits these cells are not distinguishable from other cells. In the dog they are smaller and their processes less marked."

Sano,³⁴ in the same year (1898), described a motor nucleus for the phrenic nerve in man, located in the centre of the anterior horn, extending from the middle of the third to the sixth segment. Sensory neurons originate, he says, in ganglia in the posterior roots of the third, fourth, fifth, and sixth cervical nerves.

Superficial Origin. Most of our standard English anatomical authorities give the origin as principally from the fourth cervical, with accessory branches from the third or fifth, or both, but with no figures as to relative frequency of different origins or the proportion in which different roots are found.

Luschka,³⁵ in 1853, published the results of thirty-two dissections of the phrenic roots, showing the following results:

| | | | |
|---|----|-------|-----------|
| Number originating from the 4th alone | 12 | 37.5 | per cent. |
| " " " 4th and 5th | 7 | 21.87 | " |
| " " " 3d, 4th, and 5th | 7 | 21.87 | " |
| " " " 3d and 4th | 6 | 18.75 | " |

Brook,³⁶ of Dublin, in 1887, reviewed Luschka's work and reported sixteen dissections as follows:

| | | | |
|---|---|-------|-----------|
| Number originating from the 4th alone | 4 | 25 | per cent. |
| " " " 4th and 5th | 9 | 56.25 | " |
| " " " 3d, 4th, and 5th | 3 | 19 | " |
| " " " 3d and 4th | 0 | | |

In the past four months, from observations made in the dissecting-room of the Northwestern University Medical School I have obtained the following results:

| | |
|------------------------------------|----|
| Number of cases observed | 52 |
| " " on the right side | 25 |
| " " on the left side | 27 |

These nerves had the following origin :

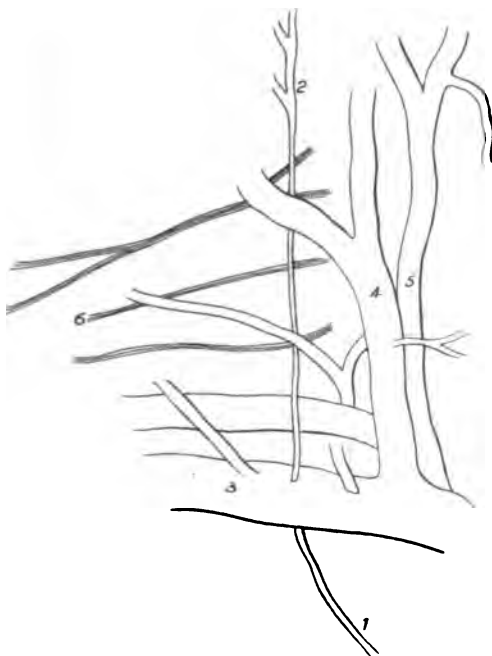
| | | |
|------------------------------------|----|----------------|
| Number of cases from the 4th alone | 9 | 17.3 per cent. |
| " " " 4th and 5th | 21 | 40.38 " |
| " " " 3d, 4th and 5th | 13 | 25 " |
| " " " 3d and 4th | 9 | 17.3 " |

These various results when tabulated show as follows :

| No. of cases. | 4th only. | 4th and 5th. | 3d, 4th, and 5th. | 3d and 4th. |
|---------------|-----------------|-----------------|-------------------|-----------------|
| Loachka, 32 | 12 37.5 per ct. | 7 21.87 per ct. | 7 21.87 per ct. | 6 18.75 per ct. |
| Brook, 16 | 4 25 " | 9 56.25 " | 3 19 " | 0 0 " |
| Green, 52 | 9 17.3 " | 21 40.38 " | 13 25 " | 9 17.3 " |
| Total, 100 | 25 25 per ct. | 37 37 per ct. | 23 23 per ct. | 15 15 per ct. |

Number of roots. When there is a single root it is from the fourth cervical. This occurred in 25 cases in 100. When there are two

FIG. 9.



1. 2. Phrenic nerve. 3. Subclavian vein. 4. Internal jugular vein. 5. Common carotid artery. 6. Roots of brachial plexus.

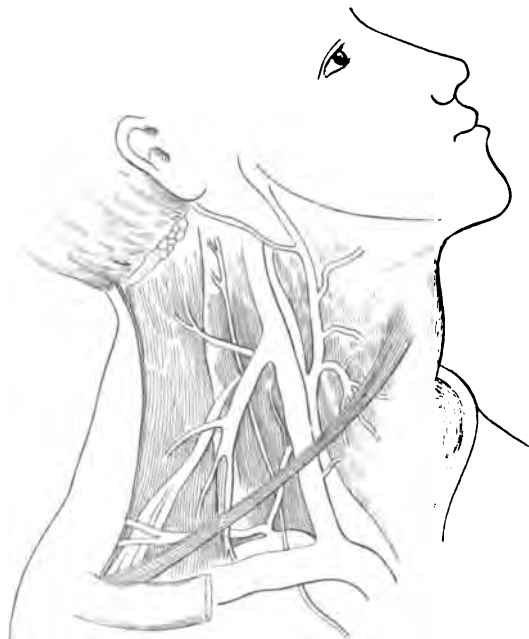
roots they are from either the third and fourth or the fourth and fifth. In 52 cases the nerve had two roots.

In 23 cases the nerve had three roots of origin, viz., from the third, fourth and fifth.

Course. The nerve seems surprisingly constant in its course. It is generally as described in the standard text-books, with few variations. One or two relations, however, are worthy of notice.

To the subclavian vein. I have noted this relation in 51 cases. In 26 cases on the left side, the nerve passed behind the vein in all. In 25 cases on the right side, the nerve passed behind the vein in 22, in front in 2, and in the remaining case passed through the vein. (See Figs. 9 and 10.) Of this peculiar anomaly I have been able to find but one similar case reported in the literature. In Virchow's *Archiv* for 1872, Von Wenzel Gruber,³⁷ of St. Petersburg, reported a case of the left subclavian vein dividing and reuniting so as to form "an island four millimetres long and two millimetres wide, through which passed

FIG. 10.



Course of phrenic (right) in neck, showing relations to adjacent structures.

the left phrenic nerve." Henocque and Eloy, in their article on the phrenic nerve, in the *Dictionnaire Encyclopedique des Med. Scien.*, say that Louget has observed a similar case, but I was not able to find any description of it in his work on the *Nervous System*.

Fawcett,³⁸ Barton,³⁹ Walsham,⁴⁰ Hunter,⁴¹ and Turner⁴² have reported one case each of the phrenic passing in front of the subclavian vein. Of these five cases, three were on the left side and two on the right. It is probably comparatively common.

I found this anomaly present twice in 51 cases, or nearly 4 per cent. It seems worthy of note on account of its relation to the clavicle. When

lying in this anomalous position it is immediately behind the posterior surface of the clavicle, and might be lacerated or divided in fracture of the clavicle or included in the callus.

Relation to the Internal Mammary Artery. *At the origin of the artery:* This relation was observed 22 times on the left side and 20 times on the right, or 42 times in all. In 22 cases (9 right; 13 left) it was external to the origin of the artery. In 13 cases (6 right; 7 left) it was internal. In 7 cases (5 right; 2 left) the nerve passed directly over the origin of the artery.

Relation of the nerve to the course of the artery. This relation was observed in 32 cases, of which 15 were on the right side and 17 on the left side. On the right side there were 14 cases in which the nerve was internal to the artery and one in which it was external. In 17 cases on the left side, the nerve was internal to the artery in 13 cases and external in 4 cases. In a total of 32 cases observed, then, the nerve lay internal in 27 and external in 5 cases.

Distribution. The main distribution of the nerve is to the diaphragm. Small twigs, varying in number, are sent to the superior vena cava, the pleura, and the pericardium. Luschka has described branches to the right auricle. This observation I have not been able to confirm. The branches in the thorax are small and easily destroyed, hence were not demonstrable in many cases on account of the parts having been disturbed before observations could be made. The branch to the superior vena cava from the right phrenic is very constant, and can, I think, be demonstrated in all cases where the integrity of the parts has been preserved. Branches to the pleura and pericardium vary in number from 1 to 4 to each structure.

The course of the main trunk of the nerve is as generally described, the left phrenic being longer and having a course far to the left over the pericardium. It breaks up into its terminal branches about 3 or 4 cm. above the thoracic surface of the diaphragm. It may remain intact until it pierces the diaphragm and divide in its substance or on its lower surface. In 29 cases in which the point of division was noted, the nerve divided above the diaphragm in 18, and in the substance of the muscle in 11 cases.

The statement made in some of our standard English works on anatomy, that the right phrenic passes through the opening for the inferior vena cava, is not borne out by the observations made. In the great majority of cases the nerve passed through a separate opening a few centimetres in front and to the right of the opening for the vein.

The diaphragmatic branches are from 2 to 6 in number, and are distributed to the lateral and anterior portions of the muscle, dividing and subdividing into fine nerve filaments, and soon losing themselves in the substance of the muscle. The exact areas supplied are as yet undeter-

mined. The single case of a dog observed by Dr. Schroeder, in which section was made of the upper roots of origin of the phrenic, the lower being unimpaired, is the only case of the kind I know of. In this dog post-mortem showed degeneration of the anterior and middle portions of the muscle, with the lateral portion normal. I hope at some future time to make some further observations on the exact distribution of these terminal branches.

In 15 cases the right phrenic perforated, in 12 cases branches were observed below the diaphragm, and in 9 cases it was possible to trace branches to the solar plexus. Two very careful dissections on unusually favorable cases showed branches of distribution as described, viz., to the semilunar ganglion, to the suprarenal and hepatic plexuses. These branches of communication in every case observed came from the innermost terminal branch of the right nerve, which pierced the diaphragm close to the inferior vena cava and ran downward and inward, retroperitoneally, to the semilunar ganglion. Whether the branches of distribution in the abdominal cavity are mainly from the phrenic or the sympathetic, or whether the branch connecting the right phrenic with the solar plexus is an afferent or an efferent branch, is as yet undetermined. In no case were abdominal branches observed on the left side.

The question of decussation of nerve fibres is undetermined, and upon this we have conflicting evidence. Kohnstamm,⁴ in his *Studien über den Phrenicus*, says: "Innervation of diaphragmatic halves is distinctly bilateral, inasmuch as each phrenic supplies only the same side of the diaphragm and comes only from nucleus of same side, which in the rabbit lies from 6 to 10.4 segments of cord. A crossing of peripheral nerves in the anterior commissure does not occur in the phrenic. Ventral part of diaphragm is supplied by a cranial trunk and dorsal by a caudal trunk."

On the other hand, Henocque and Eloy⁵ say: "The peripheral branches of the nerve (phrenic) form a plexus which completes the anastomosis between the two median halves of the diaphragm."

The question of nerve communication between the two median halves of the muscle is most important, but as yet undetermined.

Innervation from the Intercostal Nerves. Luschka,⁶ in 1853, published a monograph on the phrenic nerve, in which he says: "The fleshy portion of the diaphragm receives motor filaments from the intercostals from the seventh to the twelfth." Later on, in his *Anatomie des Menschen*,⁷ he says, in describing the last two intercostal nerves: "During their course along the last six ribs these nerves supply not only the intercostal muscles, but also, as I have previously described, the costal portion of the diaphragm. These penetrate into the costal digitations of the diaphragm in company with the terminal branches of

the arteries, and are usually so fine as to consist of primitive nerve-fibres."

Previous to Luschka, however, Bauer,⁴⁶ Meckel,⁴⁶ and Valentine had described branches to the diaphragm in man, coming from the lower intercostal nerves. Their exact origin and distribution had not, however, been determined. Their work, however, seems to have been generally overlooked, or, at least, not emphasized. Cavalé,⁴⁸ writing in 1896, says: "They are not mentioned in any of our standard authors, except a foot-note in Beauner and Bouchard's *Anatomie*."

Among English authorities also these branches seem to have been overlooked. Quain,⁴⁷ in his last edition, refers in a foot-note to Luschka's work in a general way. McClellan⁴⁶ simply says that such branches exist. Gerrish and Morris make no mention of them. Gray⁴⁹ does not refer to them previous to the thirteenth edition, into which is interpolated the statement that filaments from the intercostals have been traced to the diaphragm. In most comparative anatomies there is no reference to this nerve supply. Chauveau,⁵⁰ Mivart,⁵¹ Weiderheim,⁵² Wilder and Gage,⁵³ and Huxley⁵⁴ make no mention of it.

In 1888, Pansini,⁴⁵ of Naples, described a subdiaphragmatic plexus in rabbits formed by "the phrenic and also branches from the three intercostals." This plexus he subdivides into three: anterior, middle, and posterior plexuses. He also describes numerous ganglia in these plexuses, especially in the posterior one.

In 1896, Cavalé,⁴⁸ of Toulouse, published the results of the dissection of six adult human diaphragms, or 12 lateral halves. By dividing the diaphragm in the median line and sawing through the spinal vertebra longitudinally he removed each half of the diaphragm intact, with its costal and vertebral attachments. After macerating the part in a weak solution of nitric acid, he demonstrated diaphragmatic branches from the intercostal nerves, with the following origins:

1. There are no diaphragmatic branches from the sixth intercostal nerve.

2. Branches were observed coming from the eleventh, eighth, and seventh most abundantly, then from the tenth, twelfth, and ninth in the order named, the last being relatively poor in branches.

Course. Diaphragmatic branches arise from the intercostals just as the latter pass over the insertion (costal) of the diaphragm. They either pierce the aponeurosis (of the internal intercostal muscle) or pass through its interstices, and are directed toward the muscular fibres of the diaphragm. They are sometimes alone and sometimes accompanied by the branches of the intercostal vessels. They number 5 or 6 from each intercostal nerve.

Distribution. These diaphragmatic branches from the intercostals supply a narrow margin along the costal border of the diaphragm,

varying from 1 to 3 cm. in width. Cavalié was not able to recognize any anastomosis of these fibres with the terminal branches of the phrenic, nor did he observe any of the ganglia described by Pansini.

In later papers, published in 1898, Cavalié and Billard⁵⁷ report the results of two series of experiments. By the first⁵⁷ they demonstrated the existence of motor fibres in the diaphragmatic branches of the intercostals. In the second series⁵⁸ they proved, by physiological experimentation, that the intercostal nerves cannot assume the rôle of the phrenic after division of the phrenic itself.

Stimulation of the peripheral ends of the divided intercostals in the first series of experiments showed visible contractions of an area of about 3 cm. wide in dogs, and of a length about equal extending in the direction of the muscular fibres. Tracings of these contractions were feeble and of slight amplitude. In the second series of experiments the investigators endeavored to ascertain if the intercostal nerves could assume the rôle of the phrenic in innervating the diaphragm after section of the latter nerve. They decided that it could not, although diaphragmatic contractions on stimulating the intercostals were greater in a dog whose phrenic had been cut a month previous than in one where the phrenic had been divided just previous to the experiment. Instead of indicating any vicarious action of the intercostals, however, Dr. Schroeder is of the opinion, from his extended work on dogs, that this is due to a simple hypertrophy of the muscle fibres of the costal margin of the diaphragm following paralysis of all the rest of the muscle, and not in any way an assumption of the function of one nerve by another. This certainly appears more reasonable.

Cavalié's conclusion from his physiological experimentation, then, is that the diaphragmatic branches of the intercostal nerves cannot, in mammals, assume the rôle of the phrenic after division of the latter.

Later, in 1898, Cavalié⁵⁸ published the results of an extended series of observations on mammals and birds, with four additional human dissections, confirming his previous observations, and bringing his series of dissections up to twenty lateral halves. His observations on mammals were made on dogs, rabbits, guinea-pigs, and rats, on birds, on the cock, pigeon, sparrowhawk, and duck. His observations on dogs are most interesting, and his description of the various branches of innervation of the diaphragm very minute and accurate, but we can only give his final conclusions:

1. The intercostal nerves send branches to the diaphragm in birds and mammals.
2. In birds and mammals these branches contain motor fibres. They probably do in man.
3. They play a certain rôle in respiration, secondary in importance after the appearance of the phrenic.

4. Birds have no phrenic nerve. The diaphragm is principally supplied by the thoracic nerves which correspond to the intercostals in mammals.

5. As one leaves birds and approaches mammals the rôle of the phrenic becomes more important, and the area supplied by it becomes greater; while the rôle of the intercostals becomes less important, and the area supplied by them becomes smaller.

6. In rabbits, guinea-pigs, and rats the last five intercostals send branches to the diaphragm. In dogs the last seven, especially the eighth, ninth, tenth, and eleventh.

7. In man the last six, especially the seventh, eighth, and eleventh.

8. In dogs the area supplied is the costal margin of the diaphragm for a width of about 3 cm. In a man for a varying width of 1 to 2 cm.

I have reviewed Cavalé's work at length, as he has done the largest amount of work along this line and, so far as I know, his investigations have never been reviewed in English.

Sympathetic Nerve Supply. The innermost branch from the right phrenic communicates with the semilunar ganglion of the solar plexus. I was able to demonstrate this communication but nine times, but found it in all cases where careful dissections were made on undisturbed structures. Whether these nerves are afferent or efferent is still undetermined. In no case did I observe any abdominal branches from the left phrenic.

SUMMARY. Our conclusions may be summed up as follows:

1. From the clinical and experimental data it would seem that the diaphragm is not an essential muscle of respiration.

2. That as the symptoms commonly described as caused by an irritation on the phrenic were uniformly absent not only in the operation, but in all of the experimental work as well, it is safe to infer that they may have been due to something other than a simple injury to the phrenic.

3. That while from an anatomical point of view the diaphragm undoubtedly is enervated by branches from the intercostal nerves, this nerve supply is secondary to the phrenic and is insufficient to carry on the action of the diaphragm after a division of the phrenic.

4. That a division of the phrenic nerve, producing a partial collapse of the lower lobe of the lung on the affected side and an atrophy of one-half of the diaphragm, might predispose to infection of the lung or be followed by a diaphragmatic hernia.

5. That a division of one phrenic nerve in man, resulting in paralysis of one-half of the diaphragm only, is not necessarily fatal.

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A NEW FACTOR IN THE ETIOLOGY OF MALARIAL FEVER,
INDICATING NEW METHODS OF TREATMENT.BY A. F. A. KING, M.D.,
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THE now well-known facts that malarial fever is a parasitic affection and that its paroxysms are produced by the sporulation of successive groups of parasites in the blood compel us to reconsider some of our former ideas as to the influence of meteorological factors in the etiology of the disease. Especially is this true with regard to the *heat* of the sun, which has been so long accorded paramount importance as an etiological factor. That solar heat generates fever "miasms" by evaporating swamps, etc., is no longer to be admitted. The dreadful mortality among Europeans—especially of European soldiers marching in the sun—in tropical regions, may have been *partly* due to malarial fever, but no doubt *largely* due to "thermic fever" or insolation. Which of these two components has been most fatal in the past must remain unknown. During the month of August, 1896, there were more than two thousand deaths from sunstroke in nine cities of the United States.¹ The mortality in the tropics must be still greater.

That a certain elevation of temperature is necessary to keep alive and maintain the inoculating activity of infected mosquitoes is true for all seasons and places; but, further than this, there seems to be no scientific evidence that solar heat is in any way a factor in the etiology of malarial disease. The malarial parasite cannot be affected by external temperature, for it is submerged in a fluid—really a blood bath—of 98.2° F. in a healthy human "host;" and in a malarial patient, while the temperature may reach 104°–106° F. during the few hours of the cold and hot stages of a febrile paroxysm, it is, during the much longer period of the intermission, usually *subnormal*, between 97° and 98° F., or thereabout.

The white man is most liable to this disease, the black least so; but the black skin of the negro absorbs heat like a black coat; thus there occurs the anomaly of the hottest individual being the fittest to survive in regions liable to fever from alleged solar heat.

The island of Tahiti is situated almost under the thermal equator and only 18 degrees from the geographical equator; yet the Tahitians are "free from marsh fevers."²

Dr. Manson,³ in his excellent treatise, says the more we know of these diseases the less apparent becomes the rôle of temperature *per se* as a pathogenic factor and the more important the tropical fauna.

Dr. Celli,⁴ in his recent (1900) work, observes that temperature and malarial fever do not run exactly parallel; and he reproduces a table

of Tacchini's covering twelve years, from 1871 to 1882 inclusive, regarding fever and weather factors, in which we see "that in the year 1879, when a true malarial pandemic raged, the mean temperature for the months of July and August was the *lowest* of these years."

If the solar heat theory be untenable, may we not explain the undeniable relation between hot climates and malarial fever by eliminating the term "heat" and substituting that of "light?" Suppose we say it is the light of the sun, not its heat, that determines the periodic sporulation of parasites in the blood? I propose this idea as a working hypothesis, and the evidence in support of it will be comprised in the following statements:

1. *The accumulated experience and observations of centuries, which have been held to prove the agency of solar heat in causing malarial fever, may be held to prove the agency of solar light.* The light and heat of the sun are inseparable. The "burning" sands of Africa must also be "dazzling" sands. So with hot seasons; the cloudless skies of a hot, dry summer diffuse an intense and brilliant light. We cannot conceive how external heat can warm the parasite in human blood; but that light can penetrate the skin and act upon the plasmodium to promote its development—as light is known to act on other amœbæ—is quite intelligible.

2. *Paroxysms of intermittent fever will not, as a rule, take place at night, in the dark.* Numerous authors support this statement. Wood^b says: "It is worthy of observation that when the anticipating and advancing paroxysm reaches the period of darkness, it is either apt to be arrested in its course or to leap over the night backward into the evening or forward into the morning."

Flint^c remarks: "Paroxysms may occur at any time of the day. They very rarely occur during the night."

"It is *entirely unknown*," says Fordyce,^d "upon what this depends; indeed, the observation has been little attended to by any author who has not frequently seen the disease." The successive paroxysms getting an hour or two later may, however, be explained by the patient (languid from the paroxysm of the previous day) lingering in bed an hour or two later than usual, thus depriving the parasites of an hour or two of morning sunlight; as Flint^e tells us, "a paroxysm is sometimes warded off by taking to the bed before the hour."

3. *The relative liability and relative immunity of different races of men to malarial fever depend upon the relative translucency or non-translucency of their skin, and probably of their blood.* All agree that dark-skinned peoples, in which Welch^f includes "Negroes, Arabs, Indians, and Tamils," enjoy a relative immunity; and this has never been explained except by "specific idiosyncrasy" (a term without meaning) or by acclimation. Moreover, hundreds of negroes in the United States have the

disease, notwithstanding their alleged idiosyncrasy. Will the relative transmissibility of the skin to light clear up this difficulty?

In recently examining over a hundred negroes of different colors in Washington, to determine the translucency of their skin (easily done by passing the rays from a pocket electric flash-light through the external ear), I was surprised to find that *light passed through the skin almost as readily as through the skin of a white man*. In only three of the whole lot did I find the skin *absolutely impenetrable* to light. These were very dark, though not as black as genuine Africans, and, while they had lived in malarial places and had been bitten by mosquitoes, they had never in their lives (all were middle-aged) had ague. The affirmative evidence of these few cases, however, becomes of little real value, since there were other individuals with *translucent* skins who had *also* never had the disease.

It appears, therefore, that some negroes are not more immune than white people, because their skin, though of darker hue to an outside observer, is really not much more impenetrable to light than the skin of a white man. If my theory be correct, future experiment in tropical countries, etc., may be expected to demonstrate that individuals enjoying immunity will be found to have skins that will not allow the transmission of light.

White men who become black from malarial melanosis (cases are reported by Falls¹⁰ and Gordon¹¹), and white races becoming black after living for generations in malarial countries, may be considered instances of conservative structural modification to suit new and abnormal conditions, thus conforming to the general principle of adaptation to environment common to living organisms.

Finally, no white skin is impenetrable to light, and immunity in a white person is, so far, unknown.

Whether the *blood* of the negro is too dark, in the tropics, to admit light may be worth investigation. Cartwright¹² gives numerous authorities who affirm that the blood of negroes is blacker than that of whites.

4. *In places where malarial fever prevails the disease is increased by bright, sunny weather and lessened by clouded skies.* Observations on this point are plentiful, but they have been ascribed to "salutary rains" instead of to a clouded sky. Jackson¹³ speaks of fever-stricken troops being benefited by three days of rain, and of a camp "over which *fog hung* until late in the day being better off than another placed in a dry and elevated situation." Of Trinidad, Ferguson¹⁴ says: "It always rains nine months in the year; if it only rained eight, or if at any time there was a cessation of the preserving rains, the worst kind of fevers were sure to appear." In Tacchini's table, quoted by Celli, the year 1879, in which the percentage of fever was *greater* than in any other of the twelve years tabulated, is shown also

to have had the *least cloudiness* of all those years. It appears, therefore, that when the light of the sun is veiled by rain-clouds and fog, the parasites get less light and their sporulation is retarded. Conversely, the brighter the light the greater the liability to fever; hence the great liability of "sailors and fishermen" (Osler¹⁴) and of "berry-pickers" (Welch¹⁵).

5. *It has long been a popular tradition that to prevent the occurrence of ague, or to forestall its recurrence when it has once occurred, it is advisable to keep in the shade and avoid sunlight.* Persistent traditions have a certain amount of evidential value. Manson tells us the peasants of Italy and natives of German East Africa believed for centuries that fevers were caused by the bites of mosquitoes. So, now, the popular idea that sunshine will bring on a "chill," and shade prevent it, may be really true.

Osler, Thayer, and others affirm that intermittent fever is spontaneously curable without medicine. But in these cases of spontaneous recovery the patients were, presumably at least, shaded by their hospital surroundings from the light of the sun. That rest, food, and improved nutrition on the part of a human "host" should kill parasites in the blood seems unreasonable; and that being housed in bed prevents sporulation of the parasite, because of protection from the sun's heat, would force us to the admission that while so housed a man's blood is *too cold* to allow sporulation. Protection from *light* seems to be the only constant factor by which the spontaneous recoveries become explicable.

6. *The malarial parasite is a naked amœba. Red light promotes the vital activities of amœbæ, while violet or purple light restricts them. The color of the light diffused through the blood is necessarily red.* Experiments of Harrington and Leaming¹⁶ on the common amœba proteus show that its protoplasm may be set in motion, or brought to rest, by varying the color of the light to which it is exposed. Their concluding summary is as follows:

1. Amœba streams in the presence of red light.
2. Streaming is retarded, stopped, or reversed by rays from the violet end of the spectrum.
3. Further, the effectiveness of the following kinds of light as inhibitors of protoplasmic flow diminishes in the order named—white, violet, red.
4. Enucleated amœbæ stream in red light and cease to stream in violet or white light.

If the plasmodium malarie be a light-loving organism, red light would seem to be its natural requirement while in the blood, and we might expect to find its activities inhibited, like amœba proteus, by light from the violet end of the spectrum. This, in fact, we *do* find. Prussian blue (an old remedy for ague), and recently methylene blue, seem so far to inhibit protoplasmic movement in the malarial parasite as to pre-

vent its sporulation and thus cut short the ague paroxysms. Cases have been reported¹⁷ by Mya, Thayer, Boinet, Thintignan, Huddleston, of New York, and Mühl, of Basle, and others. If methylene blue stains the parasite or its nucleus, this blue, commingling with the red of the blood, would produce the (to the parasite) disastrous violet or purple.

Possibly the (hitherto enigmatical) curative action of sulphate of quinine may be due to its remarkable *fluorescence*. In solution it intensifies the violet, and even renders the ultra-violet rays of the spectrum perceptible to human vision.

Of the few vegetable products having this quality of fluorescence, another one is esculin, the bitter principle of the horse-chestnut tree bark (*Esculus hippocastanum*). But this also has been successfully used as an antiperiodic for intermittent fever.¹⁸ Do these fluorescent substances act by intensifying violet rays in the blood?

If the etiology now given be correct, treatment is self-evident, viz., keep malarial patients in the dark, or in rooms with purple or indigo windows, and clothe them with garments impenetrable to light; in the tropics, with white clothing lined with purple or black. Give drugs that darken the blood or render it violet, or lessen its translucency.

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ANGINA PECTORIS.

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ANGINA PECTORIS, in its typical form, is in my experience a very rare disease. Pseudo-angina, or what resembles it at times, cardiac asthma, is not infrequent. Although angina pectoris is described among the neuroses of the heart by authors, this view in my judgment

is frequently erroneous. In the great majority of instances where angina is present there exist also organic changes of the coronary circulation, of the cardiac muscular fibres, or a lesion of the aortic orifices.

I should be loath to admit, except in a very exceptional way, the existence of true angina purely of neurotic origin. On the other hand, pseudo-angina is very frequently of this provenance, accompanied by symptoms of flatulent dyspepsia. It must be understood, however, that there are cases on the border-line in which, during life, we have great difficulty in pronouncing as to what symptoms are of nervous origin and what are clearly due to organic changes of the heart or arteries.

In many such instances, unfortunately, even if death occurs, we are not always able to obtain verification of clinical facts by the results of an autopsy. Hence, certain theories are brought to the front which have no basis in actual, reliable observations from the dead-house.

One statement is certainly true of angina, viz. : that it has usually connected with it an element of spasm or sudden intracardiac pressure which separates it notably from conditions which, in many ways, are similar, and yet from this stand-point differ manifestly in a greater or less degree. Of the truth of this statement we shall be convinced later.

In general, it may be stated that pain, properly speaking, does not characterize organic disease of the heart as we commonly meet it. Of course, there may be more or less præcordial anxiety or oppression, or there may be pain in the vicinity which is of stomachal or hepatic origin ; but acute cardiac pain is very rare. For this reason it has become almost an axiom for clinicians to say when acute cardiac pain is complained of, it is more than likely no organic disease of the heart is present. Perhaps this affirmation is too positive or dogmatic, since I am confident functional distress may occur which is dependent upon obvious tissue changes.

In true angina the pain is very characteristic, and as I have said, ordinarily means organic changes of the heart structure. First of all, the pain of angina is marked by its great intensity. In no other disease, perhaps, is this so true. When the anginoid attack occurs, if the patient is walking or exercising in any way, he stops immediately and holds himself as quiet as possible, only taking hold almost involuntarily at times, so great is his distress, of the nearest object which will give him support. Otherwise, he may merely stand rigidly, having come to a short stop, with his arms lying unconsciously by his side, or else one hand is pressed firmly in the præcordial region, as if to ameliorate in a measure the subjective agony by so doing. During the attack the pectoral and other muscles of respiration scarcely move, and, indeed, breathing comes almost to a stand-still for a while. In this we perceive at once the great dissimilarity with an attack of cardiac

asthma, in which the efforts to breathe are so forcible and striking. And yet there are numerous instances in which the cardiac asthmatic features are most notable and where the "*angina sine dolore*" of Gairdner is also present,¹ as Osler states, after a masterly summary of the differential diagnosis of these two states, "when we recall to mind the features of the attack in cardiac asthma and in certain anginal seizures, the similarity of the condition, as Huchard remarks, to an acute emphysema, the views of Von Basch² appear to possess at least a reasonable probability" (p. 85).

The locality of the pain in angina is not always the same. Frequently it is located over the præcordial region. At times, however, it may be situated in the upper portion of the chest, or again, but in relatively few instances, it may stretch like a heavy bar across the xiphoid cartilage and the adjacent structures. Under these circumstances the pain may radiate directly through the chest and be felt even in the back. The pain has been likened to a heavy weight or crushing pressure, as though the thoracic parietes must almost meet. The pain is also said to resemble that of some terrible griping, as though the heart were firmly held in the clutch of some terrible monster. All these sensations, and numerous others, have been described and dwelt upon by writers, who doubtless have used their imagination at times to supply the descriptive powers of the patient. Suffice it to add that the pain is of a frightful sort and quite unlike any other we are familiar with. Often the radiation of the pain is toward the left arm, and in that case is usually carried through the forearm also and to the fingers. The ring and little fingers are said to be usually affected. Very rarely the pain extends to the right arm. When it does it radiates likewise in one or other direction mentioned.

According to Broadbent, the pain of angina often originates in the left wrist, and from there travels upward through the left arm and toward the chest—or again, as Osler states, although originating in the chest,³ "was felt very severely about both wrists." This must be an extremely rare expression of the pain. I have never met it, nor do I find it mentioned by others. It is clear that the relations of brachial symptoms⁴ (neuralgia) to angina pectoris are various. Sometimes, though infrequently, there are no pains in the arms, even in quite severe attacks of angina. Again, the brachial symptoms are very prominent, begin the attack of angina, and last longer than it does. The fact is, however, that the description of pain and its radiation, especially where it has the remarkable intensity of that belonging peculiarly to

¹ Osler. *Angina Pectoris and Allied States*, p. 82.

² These are: "Cardiac dyspnoea follows swelling and diminished elasticity in the lungs."

³ "*Angina Pectoris and Allied States*," p. 42.

⁴ *Boston Med. and Surg. Journ.*, March 14, 1901, p. 256-57.

angina, must be somewhat inaccurate at times. In any event, it could only be obtained after the attack has passed, and I am inclined to believe that only exceptionally the patient could give any graphic and truthful description of it. What is literally true is that the patient has the impression vividly marked of impending dissolution, and it is this sense, together with the character, site, and evident intensity of the pain, which are almost pathognomonic of angina.

I know of few things more remarkable in descriptive medicine than the account given in the life of Dwight L. Moody by his son, of the anginoid attack near the close of the life of the great evangelist. In this case there was no terror or mental distress, as I believe, because his faith and works fixed him, as it were, on a rock. But in very many instances there is unquestionably great terror and mental distress. This is pictured often in the countenance which has that gray, ghastly, drawn look which once seen leaves an indelible impression upon the observers who may be near. The different radiations of the pain in angina are, no doubt, reasonably explained by the position of the cardiac plexus. The site of this plexus near the heart would serve to strengthen this view. In addition, we have its divisions and communications which appear to justify this interpretation.

According to one eminent observer, the pain originates probably in the central nervous system. This writer also explains its radiation and extension by affirming that it proceeds from the spinal cord. One thing is pretty certain, if we may judge by the few thorough observations we have recorded of these cases, and it is that there is no pressure outside of the heart on the plexus from any form of aneurism or other kind of tumor. While the heart may or may not be enlarged according to circumstances, this enlargement, even though present, does not explain rationally the anginoid symptoms.

Cardiac hypertrophy and cardiac dilatation are very frequently met with, and yet in the vast majority of these cases there are no anginoid symptoms properly speaking. Leaving aside these instances, there are a few where the heart is seemingly of normal size and volume, and we must, therefore, seek an explanation of anginoid pains in some other direction. Even in acute cardiac dilatation, no intense pain is felt, and yet we should have in just such cases pressure on or distention of nervous fibres under the endocardium. Moreover, this pressure or distention must be very much greater in patients thus affected than in those suffering with angina where no similar condition exists. During the attack the patient is usually very pale, and the pallor has a certain gray, ashy hue which is indicative of the serious condition which occasions it. Together with this pallor there is extreme weakness, and a faint feeling, which cannot be resisted, overwhelms the individual who is attacked. The pulse shows by its character, oftentimes upon what

this weakness in part depends. It is frequently small, feeble, irregular, as though the poorly acting heart could not send the blood to the extremities. Again, singular to say, it is almost unchanged, at least so far as we can appreciate by our tactile sensations. In the former instance, it is probable that there is present a spasmodic contraction of the peripheral arteries; in the latter, we must assume that no such spasm exists, or, indeed, that arterial changes are so advanced that no marked impression is made upon their contractibility even by the most intense pain and disturbance of the central organ of circulation.

Frequently an attack of angina terminates by a sudden explosion of gas from the stomach. Hence, it is often stated and familiarly accepted that flatulent dyspepsia is an immediate and efficient cause of a true attack of angina. In my experience this is scarcely true, and I am more inclined to the belief that it is especially in cases of *pseudo-angina* that we should expect to find symptoms of stomachal weakness or intolerance. While admitting this, we should also not completely ignore the fact that the stomachal conditions which occasion flatulence may at times appear to be of considerable importance, taken with other exciting factors, in bringing on an attack. There may be, as Broadbent points out, a certain sympathetic relationship between the terminal fibres of the vagi in the stomach and those in the heart. Many facts would serve to demonstrate this possibility. Certainly, even in cases of marked cardiac weakness, where there has never been a true anginoid attack, dyspepsia of an acute and very distressing type will frequently follow undue fatigue of any sort or any severe shock to the nervous system such as distressing or alarming news may readily excite.

A phenomenon which is somewhat curious is the fact of an intense desire to urinate during the period of an attack, even though the effort is vain, simply because the bladder is frequently entirely empty. This statement may not invariably be true. I have known many a time emotional excitement to prevent absolutely for a while the contractile power of the bladder being exerted, and where, as was proven later, the bladder contained a considerable quantity of urine. It requires a very slight degree of annoyance or mental disturbance in men past middle life to prevent frequently their power to void their urine. Of course, the contrary of this is true, especially among women of a neurotic type, and who are still relatively young. The quality of urine of low specific gravity passed by them at times, in a very brief period, is often very great. In the differential diagnosis of true angina with *pseudo-angina*, this point should be borne in mind. During the attack where it is severe, perspiration will flow almost constantly from the patient. His face and neck and hands may be covered with it. It is cold and clammy, and lends additional significance to the gravity of the other symptoms.

The time during which an attack lasts is very variable. Sometimes it is over after a few seconds, although during this short period the agony is fearful. Again, the attack is prolonged for several minutes. Some authors state that the attack may occasionally last throughout an entire night, and that during all this time the patient is unable to move at all on account of the intense pain, and, moreover, is perspiring profusely the entire period. I must confess that I have never seen any attack of this sort, and am inclined to consider them very infrequent.

It is highly probable, moreover, that in a case of true angina depending upon advanced degeneration of the coronary circulation, which would probably be present under such circumstances, the intense pain of anginoid character would terminate life more rapidly. Here, again, I should be disposed to hold the view that a neurasthenic or hysterical element was present, which gave strength and exactness to the true diagnosis, viz.: pseudo-angina.

It must be admitted, of course, that there are instances in which the attacks are certainly anginoid in character, although they do not reach their complete development. This fact may be explained by stating that the patient, having suffered from attacks previously, so soon as he fully appreciates that one is coming on simply stops still and avoids all possible exertion until the attack has completely passed away. In these examples there may be pain in the chest, but without radiations toward the arms or fingers. Of course, if the pain is diminished in violence, there is less dread attached to the seizures, and the patient does not expect to die at any moment. Broadbent and others speak of anginoid attacks *sine dolore*. In such instances the chief danger arises from a syncopal attack in which a patient may suddenly expire. It may be that these attacks had been originally painful, and it was only subsequently that they lost this characteristic feature entirely. I should expect, in such an instance, to find at the autopsy either marked fatty degeneration of the heart walls independent of coronary changes, or else advanced aortic regurgitation. Wherever the coronary circulation is suddenly obstructed with an embolus or thrombus, the breast pang seems to be almost an invariable accompaniment. In those cases where the attack has evidently been brought on by exposure or exertion, it does not usually last long, and when the accidental occasion of the attack has disappeared, the seizure itself is apt to dissipate itself rapidly. Wherever the attack comes on spontaneously, as it were, without any accidental efficient cause being evident, it is apt to last a longer time, and only to pass away little by little and slowly. Occasionally these attacks are the most alarming in reality, and herald a fatal termination in the not distant future.

Among the causes which act efficiently in bringing on an attack of angina are primarily exertion. We are apt to say over-exertion when

the attack has taken place. This over-exertion may be a brisk, rapid walk, or the patient may be walking leisurely and without effort when the seizure occurs. Usually, however, it is when a walk has been prolonged and there is already a feeling of fatigue that the angina is felt. It has been noticed that whenever exertion takes place soon after a meal an attack is more apt to occur. It may be because digestion, if slow and torpid, is thus interfered with, and gases which are generated and accumulate in the stomach press against the diaphragm and indirectly against the heart, and thus, by causing some displacement of this organ, may occasion notable interference with the circulation.

One reason, no doubt, why attacks occur at times during sleep, is because flatus is prone to accumulate in the stomach and intestines during sleep, and considering this together with the fact that in the horizontal position we have more pressure of the abdominal viscera upward, we realize readily conditions which are powerful in causing distress. The liability, under these circumstances, to an attack is increased notably by a feeble circulation. Moreover, as we know, the circulation is always less active in repose, and this state is what prevails during sleep. A loaded rectum is, also, a condition to be avoided, and anyone subject to constipation must see to it that the bowels are properly evacuated. The distended bowel may perhaps act in a reflex manner, as well as by direct pressure.

The influence of cold is sometimes very evident. This is particularly true when a patient is walking against a cold wind. Nothing more is required than this sometimes to precipitate an attack. On the other hand, mild, warm weather is conducive to well-being, and sufferers from angina will often escape attacks during long periods when the weather is free from rapid changes and remains relatively balmy. It is essential at night for a patient to see that the bed is comfortably warm and that no chilling of the surface ensues; otherwise, an attack will often follow. This precaution may be readily attended to with a hot-water bottle or heated bricks. Gentle friction of the surface of the body, perhaps, before the patient retires, is also a proper precaution to take. The wearing of long woollen stockings, and particularly those which are somewhat loosely knit and allow free transpiration, is especially desirable, so as to keep the extremities suitably warm. I know of no small detail so important as this in all affections in which the circulation is notably impaired, and, of course, it becomes doubly imperative in warding off painful attacks which are too frequently occasioned by local chilling of the feet.

In view of the fact that dyspepsia is such a frequent symptom of angina, and appears as an efficient cause, in the judgment of a few writers, quite as often as an effect, it is important to avoid all late or too abundant dinners. The food at this meal should be of the simplest

kind, and no overloading of the stomach should be permitted. In a similar way, no sauces, condiments, or insufficiently cooked food should be tolerated.

Whenever an attack has occurred, it behooves the patient to be more than usually circumspect in all his doings, not to bring on another one. This is especially true of any exertion which seemingly has been the direct cause of an outbreak. And yet this counsel is sometimes almost unnecessary, because the patient's own feebleness, which follows a primary attack, will compel him almost to walk very slowly and deliberately, even if he walks at all, for some hours or days subsequently. No doubt these anginoid attacks would not occur if the heart had sufficient reserve force to respond adequately to the call made upon it. Unfortunately, it has not, and it is therefore evident, in many instances, that the attack is directly occasioned when we reach final causes, by the manifest inability of the heart to respond to the call made upon it for increased vigor.

Angina pectoris is not necessarily connected with any special lesion. Practically, it is almost unknown to have either stenosis or incompetence of the mitral valve appear as a direct, efficient cause of it. It is true, however, when there have been several attacks of angina, it is not infrequent to observe mitral incompetence arise subsequently. In some of these instances it has been noted, where aortic incompetence already existed, that this affection was ameliorated as regards its symptoms, and that the anginoid attacks also became less severe. The explanation appears to be in the lessened blood pressure thus brought about, as shown in the arterial pulse and in the diminished accentuation of the aortic second sound. Musser¹ has insisted upon the importance of this finding, and has reported several examples in his own experience. Broadbent² has also specially emphasized similar instances.

Angina is frequently connected with fibrous myocarditis, and at the autopsy such organic change in the heart muscle is apt to be found. So usual is this condition that Gibson states it is almost a surprise not to find it. When fibrous myocarditis is noted, it is frequently accompanied with evidences of arterial degeneration. The coronary arteries are usually implicated. Especially is this true where aortitis is present. The lesion may be limited to their origin, which is sometimes narrowed and thickened. The arteries may also be affected in a considerable extent, and the organic changes may be considerably advanced. Occasionally they have merely lost elasticity; in more pronounced alteration they may have become markedly atheromatous, or, indeed, calcified.

According to Douglas Powell, fatty degeneration of the heart walls often exists. Sometimes, indeed, the heart is so much degenerated that

¹ Transactions of the Association of American Physicians, vol. x. p. 85.

² British Medical Journal, 1891, vol. i. p. 747. Quoted by Osler.

it is easily torn, and the finger sinks into it on slight pressure. Sometimes, to the naked eye, the fat exists only in patches, affecting merely the papillary muscles or different areas of the ventricles. Even in these instances, however, if we make use of the microscope, we are apt to find considerable degeneration of the walls, where there has been no real change of coloration. In a few instances the microscope shows almost complete disappearance of the muscular fibres.

As a concurrent condition with fatty degeneration, we discover more or less advanced changes of the coronary arteries, very similar to those already mentioned in connection with fibrous myocarditis. These evidences of fatty degeneration are particularly found, of course, where the fatal termination appears to be intimately dependent upon the previous anginoid attack. It should be remarked in this place that we often have both anterior conditions, viz.: that of fibrous myocarditis or of fatty degeneration, without having attacks of true angina. According to Gibson, the relations of angina with endocardial lesions is not so distinct. It is true, of course, that degeneration of cardiac walls may often cause it, and, therefore, it is frequently found at the autopsy. The earlier writers, like Morgagni, certainly attached angina directly to the existence of aortic disease, and in one of Heberden's cases, where the autopsy was made by John Hunter, this affection is duly recorded. On the other hand, we know that the most advanced changes with ossification may exist at the aortic orifice, and yet there may be present during life no morbid symptoms at all.¹ These instances must be somewhat exceptional. What is true is, where aortic lesions have been proven at autopsy, often pain has been noted prior to death. No doubt, this pain has been in part due, at least, to interference with the coronary circulation, caused by accompanying aortitis, with which there may be also a certain degree of dilatation, or, indeed, a sacculated aneurism. Arterial degeneration, especially arterio-sclerosis, is often adjoined to attacks of angina. Where the coronary arteries are degenerated, and where the angina is seemingly dependent upon this condition, we should not lose sight of the fact that the other arteries, being degenerated, are also doubtless contributory. The affection of the coronary arteries through sclerosis and consequent narrowness, prevents a sufficient blood supply reaching the heart, and hence interferes with its nutrition. A thrombus or embolus may obstruct the vessels, but it is doubtful whether one or the other of these conditions causes angina (Gibson). One thing is sure, viz.: we often find calcification of these arteries without previous anginal attacks. Adherent pericardium may be found, but does not occasion anginal attacks unless accompanied by a lesion at the aortic orifice.

¹ Semple, p. 104 et seq.

Angina has been observed following injuries to the chest walls. In these cases the aorta may have been affected. Broadbent states its presence occasionally in malarial fever. It may also be present in advanced diabetes where the arteries may become thickened, thus giving rise to increased tension, and followed by attacks of true angina. It is not infrequent to find anginoid attacks occasioned evidently by the presence of the gouty poison. In these cases the prognosis is only serious where the intracardiac changes are already advanced, as shown by the weakness and irregularity of the heart's pulsations. In some of these cases we find notably fibroid myocardial changes in patches or disseminated. Even in these instances, prior to death, there may have been few or no threatening cardiac symptoms. In a few rare cases neuritis of the cardiac plexus and also of the phrenic nerve has been noted (Lancereaux, Peter) where anginoid attacks have occurred. The view of Semple is, indeed, that angina considered as an idiopathic disease is connected with an affection of the pneumogastric or phrenic nerve. Of course, it is difficult always to pronounce what the precise structural changes are. Still, they are doubtless present, and later will be discovered. Meanwhile we are forced to rank a few such instances among the "neuroses." Frequently, doubtless angina is associated with minute structural changes which only subsequent close investigation will determine. Flint is evidently of the opinion that the connection between angina and organic lesions of the heart is rare. Thus he has only observed fifteen cases in 388 cases of the latter. Again, Flint says that in ten years he has noted only four cases of true angina, that is to say, where the disease was unconnected with cardiac or aortic lesions. It is clear that in all instances where there is present an organic lesion of the heart or arteries, whatever suffering, if any, the patient may experience should be directly explained by them. This leads to the conviction that only those cases in which no such lesion is discoverable should be ranked among the true cases of angina. As a rule, when sudden deaths occur in what has been called angina, pathological lesions sufficiently explanatory are found at the autopsy.

The condition of the heart during an attack of angina has been believed to be one of spasm. At least, this is the opinion of some writers. It is certainly true of Heberden, who first so accurately described these attacks in his commentaries. This does not seem to be altogether a tenable opinion, if one has regard to the fact that the heart has rarely been found thus contracted.

Usually the heart stops in diastole, and is found after death in a relaxed condition. Again, during life, while the pulse at the wrist is sometimes irregular and weak, it never disappears entirely, which it certainly would if the heart were in a state of forcible, spasmodic contraction.

It has been supposed that the heart during an attack presented a sort of hour-glass contraction not dissimilar to that of the uterus. This may be, and yet it would be difficult to prove. Broadbent confesses in this connection that he has a very imperfect notion of what the condition of the heart really is during an attack. The evident fact is that during a paroxysm of angina stress is put upon the heart to which it is quite unequal to respond, and thus it shows its considerable lack of power. Often the stress put upon the heart is due to the continuous high tension of the peripheral arteries. Occasionally, however, there is low tension in the peripheral circulation, and in these instances, if there occur a sudden general arterial spasm, the amount of work thrown upon the heart becomes rapidly much greater, and consequently the heart shows relatively greater distress than where the peripheral resistance is continuously high and exaggerated. In those cases where there is marked and continuous high tension in the peripheral arteries we might suppose that pain in angina was explained by greater pressure thrown upon the heart. This can scarcely be true when we consider how many such cases escape any such pain. Again, in acute dilatation of the ventricles, we have great pressure brought upon the heart walls, and yet no pain results. Neuralgic predisposition is occasionally given as an explanatory cause. This is scarcely true if we mean by that an acquired or evident neurotic tendency, since this disease occurs more frequently with men than with women. It would seem as though from the fact that when the attack occurs immediately, the patient stops doing anything he is occupied with, or exercising, if that be what he is about, that there is a certain pre-ordered protective arrangement internally to guard against these outward manifestations of man's life. (Broadbent.)

Pain, according to Bramwell,¹ is due to irritation of the nerve terminations in the walls of the heart itself. He admits, however, that the theory of irritation due to spasmodic contraction is plausible, and compares this opinion with what occurs in the calf muscles, spasmodically contracted, in ordinary cramp.

Attacks of false angina often resemble those of the real kind. Sometimes the description of the attack by friends will enable us to reach a correct diagnosis. If the patient becomes pale, anxious, and shows signs of great distress, it may not of necessity be true angina. If, on the contrary, there be no such changes evident, we can be very sure that it is only an attack of pseudo-angina. There is much unreliability in the patient's accounts, mainly because they are apt to read up about these attacks, and often give an exaggerated idea of their own sensations. Age will throw some light upon the diagnosis. Under forty

years of age in the male, angina occurs very rarely unless there be a pronounced lesion at the aortic orifice or aortitis. In females it is rare at any age, although attacks of pseudo-angina are not infrequent with them, especially those of an undoubted neurotic temperament. Heberden, for example, in speaking of the cases observed by him, being in number over 100, states that three occurred in women, one in a boy, twelve years of age, and the others in men near or over fifty years.

Usually the first attacks of angina occur during physical exertion. Later on they may come on severely and more readily, and then we may make the diagnosis surely, even though the determining cause is slight. The physical examination will reveal, in case of true angina, the changes mentioned in the aorta or at aortic orifice. Where the attack at first comes on without exertion and at a fixed period after ingestion of food, it would seem to be of digestive provenance or pseudo-angina, and ordinarily due to a dyspeptic attack. Unless history, nature, and onset of attack all concur together with physical signs to establish diagnosis of true angina, we should lean strongly to diagnosis of pseudo-angina, and almost invariably, if we can discover any facts to support this diagnosis. If we leave out attacks of pseudo-angina which are evidently neurotic or of hysterical nature, we can usually find in some digestive disorder, particularly of the stomach, a sufficient explanation of them. There are eructation, marked flatulence, pain, occasional attacks of nausea or vomiting, which all point in this direction. A combination sometimes found is that of dilatation of the stomach, with high arterial tension. If the heart be affected in these cases and they are improperly treated, a fatal result may not infrequently follow. If, for example, digitalis or nitroglycerin be alone used, or the Schott treatment advised and carried out without any care of the stomach, and especially if the subject be old, such a denouement may not be a surprise.

The prognosis of true angina is often uncertain; and yet we have certain conditions which guide us to make it correct. We should estimate carefully the relative predominance of the two factors often producing it—on the one hand, degenerated heart walls, on the other, vascular changes. If there be high arterial tension present, and if at the same time the heart action is forcible and the aortic second sound marked, we may hope by proper treatment to modify these conditions for a while with the use especially of nitroglycerin and the nitrites. Again, if over-exertion and excitement bring on the attacks, or if flatulent dyspepsia be a decided and powerful influence in producing them, we should hope to avoid with care and treatment their natural outcome.

On no account should the patient walk hurriedly, especially in going up hill. He should also never take even a moderate walk until a certain time had elapsed after his last repast.

Attacks of angina which accompany aortic disease may last a considerable time without bringing on a fatal result if carefully watched and guarded. The worst cases are those which recur in the night or at times where no accidental cause is present and avoidable which occasions them. Again, if examination is relatively negative, if the heart is of normal size, without manifest lesion of any kind, and yet its action is feeble, its impulse scarcely felt, and the pulse usually, if not invariably, of low tension, these give great anxiety by reason of the vagueness and uncertainty as to the conditions which may be present and at any time become imminently threatening.

The apparent severity of two attacks may be similar, and yet the relative danger of them may be absolutely different. It is difficult, therefore, at times to make anything like a sure forecast. Of course, where there are pronounced cardiac and arterial changes, and where, in addition, heart failure has followed hypertrophy, the outlook is assuredly very grave indeed. If, at the same time, aortic regurgitation also be present the prognosis becomes even more serious. In a similar way, if chronic renal changes exist the future of the patient must appear dark and imminent. Gibson states that the prognosis of those affected with fatty degeneration is far less serious than the preceding. Provided always the external and avoidable causes of aggravation are prevented, such patients may often live many years. Of course, toxic angina is far less grave. As a rule, with the removal of the cause the case becomes curable. In neurotic cases, while we should expect frequent recurrences of the paroxysms, it is wholly improbable to have a fatal termination.

In this category may be placed frequently the so-called idiopathic cases. They are often extremely painful, but as no incurable lesions exist, they tend to improvement or recovery if properly managed. Of course, we should be careful in making even in these cases too favorable a prognosis, since there may be some underlying structural change of the heart walls or coronary arteries which, during life, could not be determined. There are unquestionably, according to Semple, certain cases of pure angina in which the autopsy reveals no organic changes.

The treatment of angina depends upon what is the apparent or obvious cause. In many instances, owing to the difficulty of tracing accurately to what the attack is primarily due, our treatment must be essentially empirical. First of all, we must consider the general health, and from this point of view our treatment should be hygienic. The means at our command are here what pertain to air and light, rest and exercise, food and drink. After these have all been inquired into and regulated, as far as may be, we naturally seek for the proper medicinal remedies to meet the indications of each special case. In general, also, the efficacy of our treatment will depend much to what degree we may

be able to relieve peripheral resistance to a heart frequently weakened. If, perchance, we find between the regular pulse beats, evidence of increased tension to our tactile sensations, we may often reduce this by appropriate remedies. Still, in order to recognize it, we must at times examine the heart and arteries at different periods, before and after exertion. Not infrequently the arteries are notably degenerated, hard, thickened, tortuous, and even calcareous. We can then do little to affect them directly. Yet, the capillary system, in which there may be notable resistance without excessive changes, and which has caused in a measure the arterial and cardiac changes, may be still favorably influenced by appropriate drugs, and account should be kept of this fact. In gouty conditions the peripheral circulation may show increased tension, although not visibly degenerated, and this condition, of course, may be favorably influenced by appropriate medication.

In these latter cases the ordinary treatment with a mercurial, followed by a saline, once or twice a week, will lower arterial pressure. Between times the use for a while continuously of iodide of potash and colchicum may be of signal benefit. The employment of bitter tonics, if the indication presents, and the proper regulation of the diet is of course useful. According to Powell, hop, columba, and chiretta are better tonics in these cases than quinine and strychnine. In the "neuralgic bouts," to which they are prone, he praises quinine and phenacetin. Where angina occurs with marked aortic disease it is difficult sometimes to know to what extent we may be able to help the attacks by reducing tension of the pulse. Where the pulse remains feeble between the attacks, and the heart has a weak impulse, we should carefully endeavor to help with cardiac tonics, but frequently we can be of little real service in view of the pronounced degenerative changes present in the heart and arteries. Occasionally arsenic, combined with iodide of potassium and nux vomica, is useful where the arterial tension is not too pronounced. Preference may be given in many instances to the sodium salt of the iodide, both between and during the attacks. According to Schott, it is less prone to cause heart failure; but even this salt is "apt to destroy the molecules of albumin" if continued too long or in increasing doses. Milk is the best menstruum for either salt, as in this way stomachal intolerance is less likely to occur. In these and other cases we should try to preserve the use of the nitrites and nitroglycerin for the attacks. Formerly the diffusible stimulants, like brandy, ammonia, lavender, camphor, etc., were much used for these attacks. Now they are almost abandoned for nitroglycerin and nitrite of amyl. These latter are particularly useful in relieving pain, and to accomplish it they dilate peripheral arteries. Nitrite of amyl by the rapidity of its action is preferably employed. Nitroglycerin and the sweet spirits of nitre produce similar effects in different degrees.

All of these are free from dangerous effects, as a rule; not so of nitrite of sodium, which may produce alarming results (Gibson). Nitrite of amyl and nitroglycerin dilate arteries, increase frequency of pulse and respiration, and reduce irritability of the nervous system. Where increased acceleration of the pulse and respiration are already present the nitrites must be employed with great care, as they might possibly cause greater distress. While they are said to be heart stimulants, they mainly cause relaxation of the arteries and also of the cardiac muscular fibres (Broadbent).¹

The nitrites have their drawbacks also in the fact that patients find so much relief from their use that they use them too frequently and injudiciously. A word of warning should be thrown out because life is sometimes shortened by their inconsiderate use. Glycosuria has been produced by them, it is stated. In many instances the nitrites are less useful than iodide of potash. Nitrite of amyl may be carried about with one so as to be used immediately. The nitrite of amyl in glass globules, of 3 to 5 minims, may be in a silk bag and broken upon a handkerchief and inhaled as required. The nitroglycerin tablets, one one-hundredth of a grain, may also be taken in doses of one or two when attack occurs. They do not act as rapidly as the nitrite of amyl, but their effect is more prolonged, and on that account may be more valuable in certain cases.

Some cases, however, are not relieved by nitroglycerin tablets and are relieved by nitrite of amyl. According to Broadbent, such cases have seemed to him to originate in the right ventricle.

It is the belief of Dr. B. Addy² that we have in erythrol tetranitrate a remedy superior even to "nitroglycerin," its effects being very rapid and more lasting. Tablets of one-half grain each were given by him twice or three times a day. They did not cause headache, and the remedy soon checked the attacks. It is true, the patient died after a fortnight of syncope, but during this period great relief from suffering was experienced.

Sometimes, where the heart is weak and the nitrites do not relieve, although they may relax the peripheral circulation, we must recur to the old stimulants. In addition, a turpentine stupe, or mustard leaf, or poultice may be applied over the chest and will occasionally afford a measure of comfort. Whenever these local applications fail, great relief is obtained from a hot-water bag at a temperature of 140° F. to 170° F., "moved with light touches over the whole chest."³ If, despite all this, the attack is prolonged and unrelieved, we must give a hypodermic injection of morphine and atropine, using at first small

¹ This opinion about heart fibres I do not share save very exceptionally.

² British Medical Journal, May 6, 1899, p. 1089.

³ Lancet, September 8, 1900, p. 726.

doses, and later, if need be, becoming bolder, and using larger doses. It is well to make injections deep in the muscle, where the circulation is more active than in the cellular tissue under the skin. In some instances, we should recur to chloroform inhalations as being the only hope of relief to the patient. At times they are undoubtedly dangerous, and especially is this believed to be true if fatty heart is present. As a matter of fact, however, fatty heart cannot always be diagnosed with accuracy. The apex may be strong and the pulse regular and good, and yet fatty heart may exist, and sudden death follow. Again, moreover, it has been shown that chloroform may be given safely where fatty heart later is known to exist by the revelations of the autopsy. We must relieve intense pain, however, even if there be risk, and it can only be done at times by such agents (Balfour).

In cases where there is marked heart failure, ether or brandy should be employed hypodermically in doses of ʒss to ʒj. To each hypodermic injection one or two tablets of one one-hundredth of a grain of nitroglycerin may be added. The latter should be employed with caution, however, as occasionally considerable soreness and even ulceration of the skin may result.

Theodore Schott does not value very highly digitalis or strophanthus in cases where the heart requires stimulation, even in uncomplicated forms of angina pectoris caused by sclerosis of the coronary vessels.

Oxygen inhalations are often also useful, not only to satisfy the air hunger, due to obstruction of circulation in the lungs, but also to stimulate cardiac circulation and help nutrition of its muscle, and thus get rid of effete material which interferes with proper metabolism (Powell). In these cases the oxygen must be used with a funnel near the nose and mouth, so that it may be inhaled frequently and without effort. As corroborative of the extreme value of inhalations of oxygen in the treatment of some severe cases of angina pectoris, I would refer to one recently reported in the *British Medical Journal* for December 1, 1900, p. 1568.

Rest in bed for a time is often desirable after acute paroxysms have passed, but later it is useful, as far as possible, to get the patient back to his ordinary life with judicious restrictions. The same rules apply here, however, as in other heart affections. We must remember, also, that exertion which one day may seem all right, another day may cause distress and oppression. This is one of the objections to Oertel's system of treatment (Broadbent).

Physical therapy is undoubtedly useful in some instances, but it must be utilized with great care. This counsel pertains particularly at the present time to the treatment as instituted at Bad Nauheim, where the resistant movements in conjunction with carbonic baths are prac-

tised. In advanced arterio-sclerosis every increase of the blood pressure which is the result of this treatment might lead to fatal consequences (embolism, apoplexy, rupture of aneurism of heart, or aorta). "Advanced sclerosis is, therefore, a contraindication for this treatment" (Schott). The value of many medicaments in angina comes from producing low blood pressure. The balneological and gymnastic treatment exercises a tonic influence, and "by strengthening heart muscle, as well as by acting on cardiac nerves, distressing symptoms of angina are either removed or reduced" (Schott).

Guidance should be had in regard to the bad effects of winds, great heat or cold, or rapid changes. Also, an atmosphere heavily laden with moisture is injurious. Internal conditions of dyspepsia and constipation must be warded against. A great deal of tact and good judgment are required, and the patient's disposition should be thoroughly known. Rest, particularly after meals, should be insisted upon, as patients are particularly liable to attacks at these times.

In general, supervision and counsel must be employed about exercise. Where an attack has lately occurred, it is wisdom to refrain from exertion for a while, especially if the heart is weak and fluttering, and afford it time to re-establish itself.

THE PATHOLOGY OF THE HEALED FIBROUS ADHESIONS OF THE PERICARDIUM.

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In a total of 1048 autopsies upon the bodies of adults, the records of which are among those of the Pathological Laboratory of Rush Medical College (in affiliation with the University of Chicago) and of the Cook County Hospital, some inflammatory change was found in the pericardium in 128. These changes included everything in the nature of a pericarditis, from the frank serofibrinous and tuberculous processes to those with but a thin film of fibrin over a small area, or a fibrous band uniting the parietal and visceral layers at some point. Of these 128 cases 57 showed changes that were essentially chronic, the lesion consisting of fibrous tissue uniting the opposed surfaces for a greater or less extent. These figures agree closely with those of Leudet,¹ who found in 1003 autopsies adherent fibrous pericarditis 61 times. The remaining 71 were acute, and represented all the conditions usually seen in acute pericarditis.

Of the 57 cases with fibrous adhesions the cause could be attributed to rheumatism in 8; in 6 the lesion was frankly tuberculous, as shown

by the presence of caseous masses or miliary tubercles in the adherent pericardium. There remain 43 cases of complete or partial synechia in which the cause cannot be thus determined from the anatomical findings or the history—certainly an impressively large proportion; 24 of these 43 were cases of total obliteration; in 14 the adhesions covered but part of the heart, and in 5 more a single firm band united the apex of the heart to the opposite pericardium. The large number of such cases of dry, fibrous synechia of the pericardium, for the most part lying latent during life and found only at autopsy, directly calls attention to the exactly similar condition that is still oftener found in the adjacent serous cavities, the pleural. Synechia of the pleural sacs, either total or partial, is an extremely common occurrence, and was recorded in 63 per cent. of all the 1048 autopsies from which these cases were gleaned, which figure is undoubtedly too low. In all but 3 of the 43 cases of “idiopathic” pericardial synechia there were fibrous pleural adhesions also present—that is, in 93 per cent. Furthermore, in a considerable number of cases in which the pleural adhesions were limited it was expressly noted that the pleura was adherent over the external surface of the pericardium. In several cases the pleural adhesions, where abundant, were expressly stated by the examiner to be especially firm over the external surface of the pericardium.

From these observations it would seem probable that the cause of adhesive fibrous pericarditis is in many cases the same as that of adhesive fibrous pleuritis. In addition, it is quite probable that the pericardial changes are secondary to those of the pleura much oftener than the reverse is true, for being a closed sac the pericardium has but limited chances of infection. The possible sources of infection of the pericardium are: 1. Through the blood stream. 2. Through the lymph vessels. 3. Direct infection by trauma. 4. By extension, which must be from (a) heart or vessels within the pericardium; (b) mediastinum; (c) pleura; (d) peritoneum.

Infection from the bloodvessels means a bacteraemia, which is bound most generally to produce an acute process in the pericardium, which is usually fatal and does not often terminate in the formation of fibrous adhesions unless the infecting agent be rheumatism, tuberculosis, or syphilis. Rheumatism affecting the pericardium usually affects the endocardium also, and these cases and other cases with a history plainly indicating rheumatic origin for the trouble have been excluded in this series. Of our 43 “idiopathic” cases, but two, so far as the history sheets tell us, had ever suffered from rheumatism. Syphilitic infection of the pericardium is one of the rarest of its manifestations, and when present is usually secondary to a gumma of the myocardium, which leaves sufficient traces to explain the source of any adhesions that might result. Mracek,² who has investigated the literature on this subject,

says: "Syphilis has hardly ever caused an exudative serofibrinous pericarditis." A. Genersich⁷ has recently reported such a case. Nor does syphilis often affect the pleura. Among the autopsies studied there were 35 cases of marked syphilis, but in none were the pleura or pericardium evidently affected by it. Tubercle bacilli reaching the pericardium, or any other place, by the bloodvessels produce almost necessarily distinct tubercles, so that should the resulting pericarditis heal—which cannot be common in a tuberculosis of vascular origin—the characteristic fibroid, caseous or calcareous nodules would usually give evidence of the source of the adhesions.

From the lymph vessels the most common infection would be tuberculous. This is favored by the fact that the lymphatics of the pericardium empty, in common with those of the pleura, either directly or *via* the glands at the base of the pericardium, into the glands about the large bronchi and the root of the aorta. The frequency with which tuberculous lesions affect these glands makes them the possible source of pericardial infection. To be sure, in thus explaining infection of the pericardium we must invoke a reversal of the normal current in the lymph vessels; but this phenomenon has been shown to occur by v. Recklinghausen,⁸ and its results have been observed especially in the transmission of malignant neoplasms (*vide* article by Witte⁹ on "Retrogressive Lymphatic Transportation of Carcinoma"). The choked condition of the lymph spaces in the tuberculous peribronchial glands would favor such a reversal of the direction of the lymph flow. Here, again, we would expect to find, at least frequently, miliary tubercles or their remains, as in vascular tuberculosis; but a lymphatic tuberculosis is likely to be a much more trivial affair, recoverable, and leading often to a simple sclerosis of the perilymphatic tissue. Hence it is possible that an adhesive pericarditis might result in this way, extensive or limited, and a few years after recovery no morphological evidences of its tuberculous nature remain. The connection of such a pericarditis with fibrous pleuritis is plain.

Direct infection of the pericardium by trauma can scarcely come under consideration in this matter of simple fibrous pericarditis. The occurrence of a fibrous mediastino-pericarditis due to repeated pressure on the sternum in certain trades, while affirmed by a few, is not generally credited. Roberts¹ states that he has met with no such cases. M. Labbé² reports a case of fatal cardiac incompetence following a pericarditis which he considered due to a blow on the chest. Such instances are extremely rare.

Of sources of direct extension the heart and the parts of the large vessels within the pericardium must be least common, since they are so rarely the seat of any extensive inflammatory processes. No relation between the simple diffuse chronic myocarditis and pericarditis could

be found in the cases studied, except in those cases in which the pericarditis, being primary, had affected the underlying myocardium. Localized myocarditis, acute or tuberculous, would leave sufficient evidences to determine the source of the secondary pericarditis. From the mediastinum infection might travel readily enough to and into the pericardium. Except in the lymph glands, infections in this space are rare, and a resulting process in the pericardium would have its course plainly indicated. But in the mediastinal lymph glands we have probably the commonest site of quiescent tuberculosis. These glands are in the closest relation to the pericardium possible, especially over its base. Another place of close contact is where the pericardium is carried along the left branch of the pulmonary artery, passing between the artery and the left bronchus. A gland seems to lie between this extension of the pericardium and the bronchus quite constantly. The small glands of the anterior mediastinum are also to be remembered, as they are often tuberculous. There can be no question of the relationship of these glands to tuberculosis of the pericardium. This relation has been consistently mentioned by every writer on tuberculous pericarditis, but the manner of infection has not been so clearly indicated. The possibility of dissemination of the bacilli by way of the lymphatics has already been considered. Occasionally a caseating gland breaks into the sac and erupts into this space, resulting in a rapid, caseating, or purulent inflammation of the entire surface. Such cases have been reported by Kast⁷ and Mickle.⁸ But generally the mechanism of the infection is not so evident. Usually it is simply found that a number of caseous glands lie upon the external surface of the pericardium, united to it by adhesions which sometimes are dense, but often only slight, and there is no continuity of the tuberculous lesions of the gland and those of the sac. In some cases the inflammation between the membranes is typically tuberculous, but in many it presents simply a mass of fibrous tissue of varying thickness, and neither macroscopically nor microscopically can a single feature warranting a diagnosis of tuberculosis be found. Such a case is the following;

CASE I.—J. M. G., colored, male, aged thirty-two years. Died with a general anasarca and marked albuminuria, giving rise to the diagnosis of chronic parenchymatous nephritis. No history of rheumatism or other acute inflammatory process was obtained. At autopsy the diagnosis was sustained; but, in addition, an extensive tuberculosis was found, with caseous and ulcerative lesions in the lungs, intestinal ulcers, and caseous and miliary tubercles in the abdominal and mediastinal lymph glands. There was also found an extensive bilateral fibrous pleuritis and a gray thrombus in the right internal jugular vein. In the right auricular appendix was a small mixed thrombus infiltrated with leucocytes. Both layers of the pericardium were thickened about equally, each being perhaps 2 mm. thick, and uniting them throughout was a layer of soft, bluish-gray connective tissue, which was easily torn,

though firmer at the base than lower down. Nowhere could anything resembling tuberculous lesions be found in the pericardium or in the exudate. The glands at the base of the pericardium, particularly over the right auricle, were closely adherent to the external surface of the membrane; but a careful dissection of these glands, supplemented by microscopical examination of the intervening tissues between these glands and the pericardium failed to show any extension of the tuberculous lesions outside of the gland capsule. A piece of the pericardium, with the soft exudate, was placed in the abdominal cavity of a guinea-pig; the animal remained in good health, and, when killed, eighty days later, showed no tuberculous lesions. Sections were made through many parts of the pericardium and examined microscopically, always negatively as to any lesions resembling those of tuberculosis. Everywhere the structure was that of a poorly vascularized, new-formed connective tissue, with few cells, which nowhere showed any tendency toward grouping. A few sections were stained for tubercle bacilli, with negative results. On the contrary, the lymph glands immediately adjacent to the pericardium showed all the characteristics of tuberculosis.

This illustrates a case which is almost certainly tuberculous, the origin probably from the glands in contact with the pericardium, but in which the manner of extension to the inner surface of the sac cannot be found, and in which the resulting lesion bears none of the characteristic anatomical evidences of its tuberculous origin. This case is but one of a number. Of the twenty-four cases of total obliteration of unexplained etiology, tuberculous lesions of the glands immediately adjacent to the pericardium were noted in ten. Inability to locate any direct extension from the tuberculous glands to the pericardium was likewise the result in two cases of tuberculous pericarditis, with abundant characteristic nodular lesions, that I have examined.

From this it is evident that a condition of total or partial synechia of the pericardium may and often does come from tuberculous mediastinal glands, without the healed lesions of the pericardium bearing any traces of their tuberculous origin. Two possible explanations suggest themselves. One is that the changes in the pericardium, although due to infection by the specific organism, at no time presented the anatomical characteristics of such an infection. It is by no means rare for tuberculosis in other localities to fail to produce the usual structural changes, and this is particularly true of serous surfaces. A. N. Perón⁹ has shown that in tuberculous pleurisy the lesions in the early stages are not different from those of a simple inflammation except for the presence of the bacillus, and even later distinctive tubercles often do not form, so that in the healed condition there is no evidence of tuberculosis. The second explanation is derived from a comparison with the conditions about tuberculous glands other than those of the mediastinum. It is well known that the diseased glands of a chain in the neck are frequently fused by dense masses of firm fibrous tissue. Microscopical examination of this shows it to consist simply of fibrous tissue,

poor in cells, and nowhere presenting any of the evidences of tuberculosis. The capsule of the glands is found to be outside of all characteristically tuberculous cellular groupings. On the other hand, this process of sclerosis may extend for considerable distances from the nearest tuberculous glands. Since the sclerosis is seen outside of glands that are still increasing in size, it cannot be considered as the results of a healed tuberculosis. A distinct resemblance between this process and that seen in the pericardium is evident. The explanation that is most reasonable, and one quite generally accepted, is that the periglandular fibrous hyperplasia is due to the action of the products of the tubercle bacilli that have diffused outward into the tissues. The sclerogenic action of tubercle toxin is marked, especially when it is so diluted or so composed that the necrogenic action is excluded. J. Auclair¹⁰ claims to have been able to obtain from cultures of tubercle bacilli two different substances, one soluble in ether, the other in chloroform, the first of which produces the necrogenic effects, the other only the sclerogenic. It would seem quite reasonable to assume that a similar diffusion of sclerogenic substances occurring from the peribronchial and other mediastinal glands would invade the pericardium or pleura and lead to a simple connective-tissue formation in these cavities. In favor of this view is the fact that the two sacs and the mediastinal tissues are often found fused together by a mass of firm fibrous tissue enclosing tuberculous glands; and, further, that in many of the specimens of fibroplastic pericarditis the lesions are definitely older over the base—i. e., in close proximity to the glands, than elsewhere, as shown in Case I.

The pleura is probably the most frequent source of non-tuberculous pericarditis, except for the rheumatic form, if we consider the frequency of pericarditis in pneumonia, which caused twenty-nine of the seventy-one acute cases found at autopsy. It is not to be denied that rheumatism is a more common cause clinically, but the rheumatic cases do not often come to autopsy in the acute stage. Pneumococcus pericarditis, while perhaps sometimes hæmatogenic, is oftenest produced by direct extension from the pleura. It is not uncommon to see the inflammatory process just beginning over the inner surface of the pericardium in closest relation to the inflamed pleura. Such a process, healing, might leave pleural and pericardial adhesions; undoubtedly it sometimes does. But it is universally admitted that tuberculosis is the most frequent cause of pleural inflammation, and it seems proper to assume that many of the pericardial adhesions result from the extension of this process through the pleuropericardial wall. This extension may be by growth of the tubercles through the wall, or perhaps by diffusion of the sclerogenic products of the organism. A roundabout route may also be assumed, the mediastinal glands becoming infected from the pleura, and in turn infecting the pericardium in the ways before mentioned.

Occasionally cases of tuberculous peritonitis are accompanied by tuberculous pericarditis, which seems due to an extension through the lymph vessels of the diaphragm. More often this condition is accompanied by tuberculosis of the pleura, sometimes also of the meninges and joints, seeming to be a matter of a general tuberculous serositis. Rarely a simple peritonitis produces inflammation of the pericardium. None of the cases in this series, however, gave evidence of relation to the peritoneum.

By the above process of exclusion it has been attempted to show that *many cases of adherent pericardium are probably of tuberculous origin, although not showing any anatomical characteristics of tuberculosis.* However, it is to be understood that this is not the only cause. Many other affections of the pericardium may heal and leave adhesions, and this may occur as well in an individual with tuberculous peribronchial glands as in anyone else, so the existence of caseous or calcified glands is not conclusive evidence that adhesions found in the pericardial sac are due to tuberculosis. However, when the patient's history shows freedom from rheumatism, pneumonia, and other acute processes, and autopsy reveals a pericardial synechia accompanied by a similar condition in the pleural cavity, or healed or active tuberculosis in the mediastinal glands, or both, the assumption that tuberculosis has caused the pericardial adhesions cannot often be wrong.

Returning to the forty-three cases of fibrous pericarditis in which no etiology has been assigned, it is found that forty showed pleuritis, and in eighteen healed tuberculosis was demonstrated. Of these eighteen it seems proper to consider tuberculosis the most probable cause of the pericarditis in thirteen, for the aforementioned reason. The following cases are cited to illustrate these points:

CASE II.—A negress, aged thirty-five years, who died of uræmia, was found to have, in addition to an advanced chronic nephritis, a total obliteration of the pericardial cavity by firm fibrous adhesions. Both pleural cavities were similarly obliterated. In the lungs were a few small areas of caseating tuberculosis near the apices, and a few miliary nodules were present in the spleen. There was a general tuberculous lymphadenitis, affecting principally the mesenteric, retroperitoneal, anterior mediastinal, and peribronchial glands. The glands about the pericardium were intimately adherent to it. No caseation, miliary nodules, or other evidences of tuberculosis were seen in the layers of the adhesions. The heart weighed 440 grammes, the enlargement being in the left ventricle, and explained by the renal disorder.

CASE III.—A man, aged twenty-four years; death from phthisis and amyloidosis of the abdominal viscera. The pericardium was adherent over the entire surface of the heart, the adhesions showing no caseous or other distinctly tuberculous alterations. Both pleural cavities were likewise obliterated, and the lungs were firmly bound to the external surface of the pericardium. Advanced ulcerative and nodular tuberculosis affected both lungs extensively. The peribronchial lymph glands

were much enlarged and contained tuberculous areas. It is recorded that the heart was "somewhat enlarged," but there are no details as to the changes.

CASE IV.—Man, aged sixty-six years; death from general tuberculosis of pulmonary origin. Both pleural cavities obliterated by fibrous adhesions, and the lungs thus bound to the pericardium. Uniting the heart to the pericardium beneath the adherent pleura is a band of fibrous tissue.

Of the twenty-two remaining cases of fibrous synechia in the series without assignable etiology there are fifteen accompanied by a fibrous pleuritis, without other anatomical changes being found to explain the pericardial adhesions. Of the other seven a ready explanation is found in four. One was due to extension of a sarcoma, two to extension of aortic aneurism, and one from a healed myocardial inflammation.

The cases accompanying fibrous pleuritis, and without indications of the cause of either the pleuritis or the pericarditis, are fourteen in number. Of these only the following are of special interest:

CASE V.—Man, aged thirty-eight years; died from asystole. The clinical diagnosis was: Chronic adhesive pericarditis; cardiac hypertrophy, with dilatation; pressure on recurrent laryngeal nerve, with resulting paralysis. At autopsy the obliteration of the pericardial cavity by adhesions was found to be complete, and externally it was firmly united to the pleura and to the diaphragm. The heart was hypertrophied, there was chronic mitral and tricuspid endocarditis, and both auricles and the right ventricle were dilated. General passive congestion, with anasarca, was present, and a renal infarct was found, the embolus arising in a thrombus in the left auricle. Both pleural cavities were obliterated by firm adhesions, and where the left recurrent laryngeal nerve passed beneath the aorta it was compressed for a distance of one-half an inch by dense fibrous tissue. No evidences of tuberculosis were found, and the history states that the patient had not had rheumatism or other acute infection. This case was reported by Dr. J. B. Herrick¹¹ before the Chicago Medical Society, February 9, 1898.

CASE VI.—Male, aged fifty-seven years; death from asystole, secondary to chronic interstitial nephritis, arterio-sclerosis, and myocarditis. A few thin, fibrous adhesions passed from the right ventricle to the pericardium. A few fibrous adhesions were present in the pleural cavities. No tuberculosis. In the left auricular appendix was a mixed thrombus.

CASE VII.—Man, aged sixty years; died from cardiac failure. The pericardial cavity was obliterated by firm, old adhesions. Adhesions were abundant and strong in the pleural cavities. No tuberculosis. Clinically, the case was diagnosed as chronic endocarditis, but at autopsy there were no changes in the valves, although the heart was hypertrophied and dilated.

CASE VIII.—Man, aged sixty-one years; death from an uncompensated aortic stenosis. Adhesions obliterated the pericardial cavity over the base of the heart. In the left auricle was a mixed thrombus. Both pleural cavities contained fibrous adhesions, and in the right apex were calcareous nodules.

There were four cases in which no explanation whatever of the finding was possible, there being no healed tuberculous or fibrous pleuritis, nor yet any history of any acute infectious process. There are, therefore, some eighteen cases of fibrous pericarditis in which it is impossible to find the cause, except that in all but four it was associated with more or less extensive similar changes in the pleural sacs. To attempt to state any one thing as the cause in any of these cases would be mere guesswork; but the frequency of the co-existence of pleuritis is, perhaps, of some significance. If we are to consider the cause of the two conditions as the same, the occurrence of fibrous pleuritis as the result of tuberculosis without recognizable lesions must be considered, and also the relation to previous pneumonia and simple acute pleuritis. Nephritis accompanied many of these cases, and its relation to fibrous pericarditis, although doubtful, is of interest. Serous inflammations in nephritis generally mark the termination of the chronic interstitial affection, and Bosc¹² seems to consider it as altogether a terminal occurrence. He states that it does not seem capable of regression, and is followed by death in some days, or, rather, it is present at the moment of death. On the other hand, W. Ewart¹³ believes that effusion may occur transiently, without evidences of acute pericarditis, in Bright's disease, as well as in rheumatism and cardiac troubles; usually this is of mechanical origin, but, as in the case of analogous pleural effusions, they may be dependent upon a subacute inflammation. If Ewart's ideas are correct, then nephritic pericarditis may occasionally be recoverable, and adhesions may originate in this way. But allowing for uræmia, pneumonia, and all the evident sources of pericardial adhesion, there remain a few cases the origin of which can only be conjectured from the anatomical findings. In these cases the history is often of help, giving evidence of pneumonia, rheumatism, acute infections of other sorts, which offer suggestions, while not permitting a positive conclusion.

RHEUMATIC PERICARDITIS. Eight cases of fibrous synchia in this series were directly attributable to rheumatic pericarditis, the following being illustrative:

CASE IX.—L. G., aged fifteen years, female; St. Elizabeth's Hospital, service of Dr. Sanger Brown, autopsy by Dr. Le Count. This girl entered the hospital with severe chorea and acute articular rheumatism. Death resulted from asystole. At autopsy general œdema and passive congestion of cardiac incompetence were marked. The heart was greatly enlarged, weighing 840 grammes with the adherent pericardium, and the cavities were much dilated. The mitral valves were incompetent, and the aortic cusps were the seat of a recent verrucous endocarditis. The pleural cavities contained no adhesions, and no traces of tuberculosis could be found either in the lungs or in the lymph glands. Although the peribronchial glands were enlarged and soft, they contained nothing that could be interpreted as tubercles. The pericardial sac was completely obliterated by firm adhesions, which

when torn left a reddish surface, sprinkled with small grayish and yellowish points, some of which protruded. This appearance was identical with that of an acute miliary tuberculosis. Microscopically, the "tubercles" were found to correspond to small foci of round cells embedded in the adhesions, which nowhere had the structure of tubercles, although many pieces from different parts of the heart were examined. A piece of this tissue placed in the abdominal cavity of a guinea-pig did not produce tuberculosis, and sections stained for tubercle bacilli did not reveal their presence. Sections of the lymph glands showed the enlargement to be due to a simple endothelial and connective-tissue hyperplasia, with no evidences of tuberculosis.

CASE X.—Man, aged twenty-five years; died with asystole. Five months before death this patient had an attack of acute articular rheumatism, and one month later the cardiac symptoms became manifest. The clinical diagnosis was chronic endocarditis, cardiac hypertrophy, and acute dilatation. On opening the thoracic cavity the pericardium was found to present an unusually large surface, extending 10 cm. to the left and 6 cm. to the right of the median line. On the left side there were a few adhesions between the lungs and the pericardium, but *there were no adhesions to the chest wall.* On opening the pericardium it was found that although the external layer was not noticeably thickened it was united to the epicardium by firm fibrous adhesions, separated with difficulty. The heart was much enlarged, weighing 700 grammes; the ventricles were dilated, the right being 11 cm. in depth, the left 10½ cm. Both auriculo-ventricular orifices were much enlarged. The myocardium showed no macroscopical changes. All the valvular surfaces were normal. General anasarca and passive congestion of the lungs, liver, spleen, and kidneys gave evidence of the valvular incompetence. No evidences of tuberculosis could be found in the lungs, pleura, glands, or elsewhere. There were no pleural adhesions except those uniting the left lung to the pericardium, which presumably originated from the latter.

CASE XI.—A woman, aged twenty-two years; death from asystole, with the following clinical diagnosis: Mitral regurgitation and stenosis, dilatation of the heart, double hydrothorax, hydroperitoneum, thrombosis of the left axillary vein. Her trouble all dated to an attack of rheumatism five weeks before death, with evidences of involvement of the heart, although no positive evidences of pericarditis were obtained while she was in the hospital. Three years previously she had had an attack of articular rheumatism, not accompanied or followed by cardiac manifestations. The anatomical diagnosis was as follows: Acute mitral and aortic endocarditis; mitral stenosis; hypertrophy and dilatation of the heart; thrombosis of the left auricle and of the left subclavian and axillary veins; general anasarca; healed splenic infarct; passive congestion of the liver and spleen; fibrous internal and external pericarditis. In this case the external surface of the pericardium was found adherent to both lungs where they were in contact with it, and over the apex to the chest wall. The cavity was completely obliterated by very firm fibrous adhesions, showing no nodules, caseous or fibrous. No adhesions were found in the pleural cavity except those about the pericardium, and the glands and lungs showed no evidences of tuberculosis.

CASE XII.—Man, aged thirty-five years. Clinical diagnosis: Cerebral embolism, chronic degeneration of the kidneys, left heart hyper-

trophy, cedema of the lungs. Anatomical diagnosis: Chronic mitral, tricuspid, and aortic endocarditis; hypertrophy and dilatation of the heart; softening of the left basal ganglia and left cerebral cortex; renal infarct; general passive congestion; cirrhosis of the liver; chronic fibrous pericarditis; and localized fibrous pleuritis. On the outer surface the pericardium was attached to the left lung by one fibrous band; internally the cavity was totally obliterated by firm fibrous adhesions, simple in character. In this case cardiac symptoms were first observed during an attack of rheumatism four months before death, with dyspnoea, which had been marked up to the time of death, and by cerebral embolism.

CASE XIII.—Woman, aged forty-five years, dying with asystole. Anatomical diagnosis as follows: Acute and chronic mitral and aortic endocarditis; mitral stenosis; thrombus in right auricular appendix; infarcts in the lungs, with localized fibrinous pleuritis; general passive congestion; obliterative fibrous pericarditis. The pericardial sac was obliterated by simple fibrous adhesions, and externally adherent to the right lung. No tuberculosis. This patient had repeated attacks of rheumatism during the last twenty years of her life. First noticed cardiac symptoms twelve weeks before death, after exposure to cold, which exposure resulted in no other trouble besides that of the heart.

Comparing these cases with those of tuberculous origin, the following anatomical differences are seen: *In the rheumatic there is a total absence of tuberculous lesions in the mediastinal glands and lungs; the pleuritis existing is generally localized at the pleuropericardial surfaces, seeming to be secondary to the pericarditis; endocardial changes are usually present, in all but one of those recorded; death is usually due to cardiac failure in the rheumatic, while this is the exception in the healed tuberculous pericarditis; the age of the subjects is, on the whole, younger in the rheumatic.* So far as the changes in the pericardium are concerned, they differ not at all from those of the simple fibroplastic changes sometimes produced by tuberculosis, as described previously. On the other hand, in one case (Case IX.) the changes resembled macroscopically so closely those of a miliary tuberculosis of the pericardium, with sclerotic changes predominating, that it required considerable microscopical and experimental evidence to exclude tuberculosis. It is of interest that all but the first of these eight cases was classified as of probable rheumatic origin on the autopsy findings alone, and the verification of this diagnosis by the history when subsequently investigated showed that the differences above described are sufficiently characteristic to be of value. The relative effects of rheumatic and tuberculous pericarditis on the heart itself, which are of much clinical interest, will be discussed later.

CALCIFIC PERICARDITIS. Of this rather unusual form of pericardial lesion there are four examples found in this series.

CASE XIV.—Man, aged thirty-five years; death from chronic nephritis. The pericardial change evidently attracted the attention of the examiner, for the clinical diagnosis made before autopsy reads: "Chronic nephritis; pleurisy, with effusion; pericarditis or medias-

tinal tumor." The anatomical findings were as follows: General anasarca; fibrous pleuritis and peritonitis; fibrous pericarditis, with calcareous plates; serofibrinous pleuritis; dilatation and hypertrophy of the heart; interstitial pneumonia; cirrhosis of the liver, with congestion of the spleen and gastro-intestinal tract; chronic diffuse nephritis. Externally the pericardium was bound firmly to the diaphragm and lungs; internally it was found obliterated completely, and embedded in the adhesions were large, thick, calcareous plates. No tuberculosis found anywhere.

CASE XV.—A "middle-aged" woman, who died after an operation for empyema. Unfortunately the record of this case is incomplete, it having been done at a private autopsy outside the hospital, and it is impossible to improve the data. The anatomical diagnosis reads: Right pleuritis and thoracotomy wound; left fibrinous pleuritis; adhesive fibrous pericarditis, with calcareous plates; atrophic cirrhosis, with passive congestion of the spleen. It is stated that the pericardial layers were adherent, and in the adhesions were found irregular, calcareous masses; one, on the anterior surface, measured 3 cm. by 2 cm.; on the left border was one one-half the size of the fist; over the diaphragmatic surface was one 2 cm. in diameter. The heart was the seat of some hypertrophy and more marked dilatation, the left ventricle measuring 9.5 cm. in depth. No tuberculous changes were found in the lungs. Here the pericardial change seems definitely older than in the pleura, for the latter is described as fibrinous.

CASE XVI.—A man, aged thirty-five years, who died from asystole, with the following clinical diagnosis: General arterio-sclerosis; chronic endocarditis; insufficiency of the mitral valves; chronic parenchymatous nephritis. The anatomical diagnosis: Chronic adhesive pericarditis, with calcification; chronic mitral endocarditis, with stenosis; hypertrophy and dilatation of the heart and tricuspid incompetence; chronic adhesive pleuritis; general passive congestion, with slight anasarca; multilobular cirrhosis; diffuse nephritis. The pericardial sac was completely obliterated; there were calcareous masses in the right and upper border and a large collection of "stony masses" in the sac proper. Externally it was adherent to the left lung. No tuberculosis found in the body.

CASE XVII.—Man, aged fifty-five years, who died of acute nephritis. At autopsy were found: Chronic fibrous pleuritis; chronic fibrous pericarditis, with calcification; atheroma of the aorta; acute parenchymatous nephritis; cirrhosis of the liver, with jaundice; acute myocarditis, with myocardial segmentation. The pericardial cavity was found completely obliterated by friable fibrous adhesions, greatly thickened, and between the layers of the pericardium toward the base was a large calcareous plate. There were no changes in the lungs, and traces of tuberculosis were not found.

Calcification of pericardial exudate is rare enough to be of considerable interest, although the occurrence of four cases in such a relatively small number of autopsies leads to the belief that its rarity lies chiefly in the infrequency with which it has been reported. In 1899 Fritz Diemer¹⁴ was able to collect but ten cases, beside two of his own, in which the calcification was extensive, more or less completely covering

some part of the heart. Three of the four cases mentioned would come under this description. Perhaps the most remarkable instance of this condition was reported in this country in 1890 by Drummond.¹⁵ In this case the greater part of the heart was surrounded by a stony covering; the right ventricle had to be opened with a saw. The calcification extended through the right auricle and through the entire thickness of the left ventricle. It varied from one-quarter to one inch in thickness, and seemed to involve the epicardium only, not the parietal layer, which could be stripped off.

The etiology of this condition is very obscure. Diemer states that in his twelve cases it was hinted at in six only. His series certainly throws no light on the subject, except in pointing out the fact that in only one was any tuberculous lesion found, and that not related to the pericardium. We know that calcification occurs only in dead or dying tissues, or inspissated exudates, which, from analogy, are probably purulent. Calcification of adhesions is seemingly the less common of the two. In all the four cases described in this series it seems most probable that these were instances of calcified inspissated exudates. What form of exudative pericarditis is most likely to heal and leave the proper conditions for calcification? Certainly not tuberculous, for a caseating tuberculosis of the pericardium would seldom last long enough for this, and if it did it would be accompanied by characteristic lesions locally or remotely; but in none of these four cases, and in only one of Diemer's twelve was tuberculosis found anywhere in the body. However, Püschmann¹⁶ has reported a case of calcareous myocarditis of tuberculous origin. The purulent exudate which is least malignant and most prone to become sterile and inspissate is that arising in pneumococcus serositis, and, further, this is one of the most common causes of acute pericarditis. It also agrees with the conditions observed in most cases of calcareous pericardium, in usually leaving no traces of its origin in the lung except fibrous pleuritis. In the first case of this series the existence of a marked interstitial pneumonia is of significance, as also the pleural thickening in three of Diemer's cases. Case XV. gave a history of pneumonia eleven years before death, and also of attacks of rheumatism, the last two years before death. Unfortunately, in the other cases the history is incomplete. Ewart¹⁷ has reported a case of extensive calcification of the pericardial adhesions resulting from rheumatism.

A most important as well as interesting observation is that in *each and every one of these four cases of calcareous pericarditis a marked cirrhosis of the liver was present*. On looking over Diemer's¹⁴ cases it is found that in two of the twelve no reference as to the liver can be obtained. Of the remaining ten, in seven a distinct atrophic cirrhosis was observed; in one a fibrous perihepatitis, combined with advanced

syphilitic cirrhosis, was found ; in another a fibrous perihepatitis alone ; in but one was there no fibrous change in the liver. This striking feature seems to have been overlooked by Diemer. In only one of these nine was any reference made to alcoholism, this individual being rated as a moderate drinker ; in one case—that of Drummond¹⁵—the macroscopical appearance of the liver suggested tuberculous cirrhosis, but this was not confirmed microscopically. In the remaining cases there is nothing to explain this cirrhosis. Further reference will be made to this matter later in considering the results of pericardial synechia.

RESULTS OF PERICARDIAL ADHESIONS. For the most part, synechia of the pericardium seems to be altogether without effect on the patient, and it is not usually recognizable during life, even in the cases in which it does produce some effects that are readily referable to the heart or circulatory system. Like the fibrous pleuritis, it is essentially an autopsy finding. Of the fifty-seven cases of fibrous pericarditis, of whatever nature, in the entire series, in thirty-six of which the pericardial obliteration was total, the condition was recognized during life in but three. For the consideration of the effects of pericardial adhesions the cases may be conveniently divided into three classes :

1. Those with but one or a few bands of fibrous tissue connecting the parietal and visceral layers.

2. Complete or nearly complete obliteration of the pericardial sac, without adhesions between the external surface and the surrounding structures.

3. Pericardial sac completely obliterated and adherent externally to the adjacent structures—that is, combined internal and external pericarditis.

It is hardly probable that a few bands such as are often seen lying upon the auricles, or attaching the apex of the heart to the parietal pericardium, interfere in any appreciable degree with the action of the heart.

More extensive adhesion may, and in many cases most certainly does, prevent perfect cardiac function. It is quite generally recognized that in the third group of cases, in which the heart is bound to the pericardium, and this in turn to the chest wall, the lungs, the posterior mediastinum, and the diaphragm, that cardiac dilatation, hypertrophy, and ultimately incompetence often result. This process is seen particularly in rheumatic cases, and is one of the most rapidly fatal of the cardiac complications of rheumatism, if not the most so. From the nature of the changes present, an incompetence established in this way is absolutely unaffected by treatment, and the course is rapidly and progressively downward. Pericardial obliteration from causes other than rheumatism less often leads to cardiac failure. It will be recalled that in the cases of this series ascribed to tuberculosis there were no deaths

at all from asystole, whereas in those of rheumatic origin the cause of death lay in the heart alone, either from the pericardial lesion or from valvular changes, or both, in all but one, which terminated prematurely from cerebral embolism (Case XII.). English writers have considered this matter of asystole of pericardial origin most extensively, and ascribed to it the proper importance. The reason that the rheumatic pericarditis is so much more prone to cause fatal incompetence of the heart is ascribed by them quite generally to changes occurring in the myocardium during the rheumatism; in other words, rheumatic pericarditis is generally a pancarditis affecting valves, muscles, and pericardium together. From this condition dilatation results, and Dickenson¹⁸ argues that the chief effect of the pericardial adhesion is to prevent this dilatation from being overcome as the heart gains strength—*i. e.*, the dilatation is primary, and not due to the adhesions. Hypertrophy follows, but under disadvantages, and compensation is never attained. "Thus it is," he says, "that adhesions following upon non-rheumatic pericarditis, which even in children is attended by little enfeeblement of the heart, proves generally harmless."

While not questioning in any way the logic of Dickenson's reasoning, nor doubting the correctness of the inference, there is another factor that does not seem to have been brought forward. This is based upon the function of the pericardium as determined by H. L. Barnard.¹⁹ Barnard has shown that its function is, beyond that of providing a smooth surface to permit motion, to limit dilatation of the heart and to relieve strain upon the muscle, especially upon the right ventricle during diastole. He compares its function in relation to the heart muscle to that which the fibrous adventitia of the arteries bears to the muscular coat. First, he found that normal pericardium is practically inextensible. Then he demonstrated that, while the heart of a dog within the pericardial sac ruptured under a minimum pressure of $1\frac{1}{2}$ to $1\frac{3}{4}$ atmospheres, when freed from the pericardium it ruptured from a pressure of $\frac{1}{2}$ to 1 atmosphere. Rupture within the pericardium is preceded by a rupture of that sac, which gives way at its attachment to the great vessels. (Barnard says that this part of the pericardium seems to be the only part that contains nerves, and suggests that this may bear some relation to the subjective symptoms met with in pericardial effusions, heart dilatation, etc.) He further showed that a heart which within the pericardial sac could be made to take 10.5 c.c. of fluid, when removed from the sac dilated to hold an additional 16 c.c., showing the support of the pericardium to the heart in dilatation. In pericarditis with effusion, the alterations of the pericardial tissue by the fluid and by inflammatory changes permit it to yield under the pressure of the accumulating exudate. If no adhesions form without, it returns to its normal size as the fluid is absorbed; but when it is fixed to the

chest wall, the diaphragm, and other structures, as the fluid subsides it cannot return to its original dimensions, at least not entirely, and so the heart, whether inflamed or not, lacking any support from the outside, dilates as the fluid disappears until the two pericardial layers are again in contact. Furthermore, in a pericarditis with effusion, the heart and pericardium are separated by a considerable space over most of their surfaces. The heart cannot well become adherent until the fluid is absorbed, and if the pericardium is adherent externally adhesion between its two surfaces implies a dilatation of the heart to render this interpericardial adhesion possible. Now, rheumatic pericarditis is often a pericarditis with effusion, while the form of tuberculous pericarditis under consideration in this paper is presumably a simple plastic process, occurring as it does slowly and without symptoms. Hence the occurrence of cardiac incompetence often in one and seldom in the other. Myocardial degeneration and valvular lesions such as occur in rheumatism would, of course, favor the dilatation of the heart to a dangerous degree, from which it is ordinarily prevented by its own musculature. The chief difference between this theory and that of Dickenson is that in the latter the dilatation of the heart is considered primary; in the former it is considered as secondary to the distention of the pericardium.

Adhesions on the outer surface of the pericardium are most commonly thin filaments attaching it to the lung on either side, especially the left; such adhesions are of little importance, because of the elasticity of the lung. It is when the bands are firm and numerous, or the adhesion is of complete surfaces, and the pericardium is united to the chest wall, or to the lung, when it is in turn firmly united to the chest wall, or, perhaps most important of all, when the pericardium has spread over the diaphragm for a considerable distance, that the heart is affected. It is by no means unreasonable to consider that by the traction of these different points of attachment during inspiration, the diaphragm and the chest wall pulling in different directions, that a dilatation begun by the contraction of new-formed connective tissue would be increased. This traction during inspiration is accepted by many as the correct explanation of the *pulsus paradoxus* that is sometimes seen in the condition of pericarditis accompanied by mediastinitis. Of causes of simple plastic pericarditis with mediastinitis, other than rheumatism, pneumonia is the most prominent, for the inflammation, beginning outside of the pericardium, naturally leaves external adhesions. The simple plastic form of tuberculosis is probably seldom accompanied by much adhesion to the chest wall or diaphragm, unless occurring with a mediastinitis. J. H. Sequeira²⁰ has reported an interesting example of this condition occurring in an infant fifteen months old.

That adhesions limited to the pericardial cavity itself can so interfere with the action of the heart as to lead to asystole has been a much-

debated question. Many of the English writers are convinced that internal pericarditis alone, especially when of rheumatic origin, may do so. For example, A. G. Barrs²¹ has reported four cases, all in young people, in which there were no adhesions to the anterior chest wall, and in one only a few adhesions to the lungs. Theo. Fischer,²² writing on the importance of pericarditis in children, states that of a total of 196 cases of adherent pericarditis occurring in Guy's Hospital only eleven had adhesions to the anterior chest wall. As he found that in seven years' time, while there had been but four deaths due to valvular disease in children under fifteen years of age, there had been thirteen from adherent pericardium, it is evident that most of these fatal cases of adherent pericardium must have been without external pericarditis. Carl Hirsch²³ has made a valuable study of cardiac hypertrophy by ascertaining the ratio between the weight of the body and the weight of the heart and of its individual parts. He arrives at the conclusion that simple obliteration of the pericardium cannot bring about hypertrophy. This conclusion is, however, based on the findings in but six cases, without reference to the cause of the adhesions, nor to the existence of external adhesions. Dickenson¹⁸ takes the same view, and states that in nearly all cases of rheumatic pericarditis there are adhesions externally, although in his own series of cases this condition was not marked in about half. However, it is to be remembered that in removing the sternum as ordinarily done at an autopsy, any abnormal adhesion of the pericardium anteriorly would be overlooked, as a rule, unless quite extensive.

An illustration of asystole, without external adhesions of consequence, is afforded by Case X. During life this was considered a case of broken compensation from valvular lesions. At autopsy the valves were found quite normal, the cavities dilated, walls hypertrophied, and the pericardial cavity was obliterated by strong adhesions without much thickening of the pericardial walls. There were a few adhesions between the left lung and the pericardium, but, although searched for, no other adhesions were found. Here, apparently, a fatal incompetence had resulted from simple internal adhesions. Macroscopically, the myocardium was unchanged.

How an internal obliterative pericarditis can interfere with the heart to any extent is difficult to see. Romberg²⁴ has suggested that the fibrous tissue, contracting, interferes with diastole, which supposition hardly agrees with the prominent dilatation in all these cases. Myocardial change in some instances is, perhaps, responsible, and this is sometimes to be distinguished only microscopically, as in a case reported by Jaccoud,²⁵ who considers that secondary changes are responsible for the incompetence of synechia more often than mere mechanical traction. In a number of cases that I have examined the myocardial lesions have

been slight and limited to the tissues immediately beneath the pericardium. This applies also to the fatty infiltration which is often seen to cut out the superficial part of the musculature. The mere inelasticity of the adherent pericardium is not sufficient to cause any significant interference with cardiac action, and "there is abundant evidence that the mere fact of obliteration of the pericardial cavity around a normal heart is a trivial impediment to its action, and excites neither hypertrophy nor dilatation." (Dickenson.¹⁸) The fatal termination of the process produced by the pericarditis is not generally long delayed; often it is within a year of the beginning of the acute inflammation. In the case of Jaccoud,²⁵ previously referred to, death occurred about three and a half months after the onset.

An interesting and important condition which results from pericardial synechia is that of "pericarditic pseudocirrhosis" of the liver. This was first described by F. Pick²⁶ in 1896, and has attracted much attention since that time. He stated that the cases resembled much the mixed form of cirrhosis of the liver, with enlargement of the liver, and ascites, but without jaundice, occurring especially early in life. In all these cases the pericarditis is latent. The cause of the symptoms he considered as eminently a cirrhosis due to connective-tissue proliferation resulting from obstruction in the circulation of the liver, and to intense ascites through portal congestion. R. C. Cabot,²⁷ in 1898, reporting a case, collected seventeen others from the literature, and the reports have increased at a corresponding rate since that time. V. Eisenmenger²⁸ most correctly objects to the relation between passive congestion and cirrhosis assumed by Pick, for little hyperplasia of connective tissue results from passive congestion in the liver, and that which does form is in the centre of the lobule. The ascites, without oedema of the legs, he attributes to conditions outside the liver, chiefly torsion, compression, or angulation of the inferior vena cava by the pericardio-mediastinal adhesions; or else through a localized peritonitis, with some possible etiological relation to the pericarditis, at the transverse fissure of the liver. This last is, perhaps, most marked in the perihepatitis or "zuckergussleber" of Curschmann. One interesting observation is the following: In seven of Diemer's²⁴ ten cases of calcification of the pericardium, in which the condition of the liver was described, atrophic cirrhosis was present; in an eighth there was extensive perihepatitis and syphilitic cirrhosis; in the ninth there was perihepatitis alone; in one only of the ten there was no cirrhosis, but simple passive congestion. Only one gave a history of alcoholism, and that but moderate. In two of the three cases of pericarditis in Pick's first report the exudate was calcareous, and in both of these there was a slight but demonstrable cirrhosis of the liver; the third, in which the adhesions were not calcified, showed no cirrhosis. C. Bacaloglu²⁹

reports a case of calcareous pericardium, not included among those of Diemer, showing a slight atrophic cirrhosis; no history given. In all four cases of calcification of the exudate in the writer's series a marked atrophic cirrhosis was present, although without ascites. Of these four, one gave a history of alcoholism (Case XVI.), but the other three were not alcoholics. From a study of the autopsy reports and history-sheets of these cases I am unable to ascribe any relationship between the pericarditis and the cirrhosis; but the coincidence is striking. However, it may be said that *while in fibrous pericarditis we do get a symptom-complex entitled to the name of pericarditic pseudocirrhosis, in calcareous pericarditis we frequently, in the great majority of reported cases, in fact, get a real atrophic cirrhosis.* This association of cirrhosis is not seen in cases of simple fibrous pericarditis. A case reported by Ewart,¹⁷ of calcareous pericarditis, presented marked cedema and conditions leading to the diagnosis of atrophic cirrhosis, which did not exist. In this case the operation for establishment of collateral circulation by producing plastic peritonitis was performed.

An individual with both cardiac and tuberculous lesions may present them combined in an interesting form in the liver, as the so-called *cardiotuberculous liver*. This is simply a marked chronic passive congestion of the liver, plus tuberculous changes, which may be, according to Cousin,³⁰ either specific tuberculous lesions, or fibrous or fatty changes with or without tubercles. Tuberculous pericarditis offers the proper conditions for the development of this form of hepatitis, and it has been seen in many cases. Hutinel³¹ has reported several cases in children, and considers the subject extensively.

W. H. Welch³² has written of *thrombosis of the systemic veins* occurring in cardiac disease. In twenty-seven such cases collected by him the pericardium was found adherent three times, all being rheumatic. This has been observed in two of my series (I. and XI.); in both of these and in four others (V., VI., VIII., and XIII.) there were thrombi in the auricles. The snaring off or immobilization of parts of the auricles, particularly the appendix, furnishes most favorable conditions for thrombus formation, and embolism from this source is frequent. This is, therefore, a dangerous complication. Welch ascribes the thrombosis of the systemic veins in part to the eddying or whirling movement of the blood that may be set up in heart disease in the lower end of the jugulars and the adjacent parts of the innominate and subclavian veins, in part to the anatomical disposition and structure of the veins, but is inclined to think that infection is the important factor. In his first case, which resembled Case XI. of my series, in almost every detail, streptococci were found in the thrombus. In Case XI. the thrombus in the left axillary vein contained staphylococci, both aureus and albus, also the bacillus coli communis, and a small, strictly

anaërobic organism, the exact nature of which was indeterminate; it stained by Gram's method. From the heart's blood only *S. pyogenes aureus* and *B. coli communis* were obtained. The other case of thrombosis (Case I.) yielded *S. pyogenes albus* in cultures from the thrombus, which was not obtained in cultures from the heart's blood.

TERMINATIONS. It is quite probable that fibrous adhesions when once formed in the pericardial sac are not necessarily permanent. It is known that they are often removed from the peritoneal cavity in course of time, and this is probably true of the pericardial cavity. As the new-formed connective tissue becomes older and assumes its fibrillar structure the inevitable contraction that follows obliterates most of the bloodvessels, as in all scars. To this is added the incessant tugging of the heart, drawing them out and causing them to finally separate and form simple tags on the surface of the pericardium. This conception is formed from the frequent finding of the various stages of this process. As a rule, the parts of the adhesions that remain longest are those over the surface of the right auricle and also between the parietal layer and the apex and ventricular septum. Sibson³³ states that the reason for this is that these are the portions of the front of the heart which move least. As the "tugging" effect, as well as the interference with circulation by contraction, would be manifestly less in a completely obliterated sac than in isolated adhesions, the chances of complete absorption of adhesions will vary inversely with their extent, other conditions being equal. A healthy and strong heart would, of course, most readily free itself from the bands. Calcification is another possible termination.

The fibrous adhesions may also act as a "*locus minores resistentiæ*." Rubino³⁴ has shown the effect of injury to the pericardium on localization by first injuring the pericardium by heat, mechanically, and by severe cooling, and then injecting pus microbes into the general circulation. In this way he produced experimentally a pericarditis, which is the result that would be expected from what we know of the general laws of bacterial localization. That fibrous adhesions may produce in a slighter degree the same effect is not improbable, and it is not uncommon to see an acute pericardial inflammation engrafted upon a chronic adherent process in which the pericardium is but partly obliterated. On the other hand, total obliteration of the cavity would render secondary infection very unlikely to occur. McPhedran and Caven³⁵ in reporting a case of diffuse tuberculous hepatitis state their opinion that a coexisting pericarditis was implanted in this way on an older fibrous pericarditis.

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A CASE OF FIBROMA MOLLUSCUM.

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In July of this year, G. J., a young, unmarried woman, aged twenty-six years, came to the Skin Dispensary of the University Hospital for advice concerning a disease of the skin characterized by the presence of a large number of variously sized tumors distributed over the entire cutaneous surface. These tumors were covered by normal skin, and varied in size from a hempseed to a pigeon's egg; they were soft and inelastic to the touch, the largest feeling like empty sacs, and, according as they were small or large, they were hemispherical and sessile, or pear-shaped and pedunculated. While all parts of the skin were affected, the tumors were most abundant and largest upon the arms. The history of the affection, very briefly, was that the tumors began to appear in infancy—it was asserted most positively by other members of the family that none were present at birth—and rapidly increased in number, new lesions continuing to appear up to the present time. Her general

health was in no way affected. The patient, while a physically well-developed woman, was evidently defective mentally.

Sections were made of a small tumor excised from the back, and examination of these showed that the epidermis was in no way altered, except that the interpapillary prolongations of the *rete mucosum* had to a large extent disappeared over the centre of the growth, owing to the flattening out of the underlying papillary layer of the corium. The

FIG. 1.



substance of the tumor consisted of fibrous tissue which at the periphery was arranged in a loose meshwork, while the central portion was quite compact. Many round and spindle-shaped deeply staining nuclei were observed, which were appreciably larger at the periphery of the growth than in the centre. With the employment of appropriate staining a considerable number of "mastzellen" could be seen. A few apparently normal hair-follicles and sebaceous glands, and a moderate number of small bloodvessels were present.

While not an extremely rare disease, fibroma molluscum is nevertheless quite uncommon, since, according to the statistics of the American Dermatological Association, it comprises but 0.090 per cent. of all diseases of the skin.

The etiology of the affection is extremely obscure, but heredity seems to play some part in its production, since it has been observed to occur in several members of the same family and in successive generations. Virchow has reported that a patient having fibroma molluscum stated that his grandfather, father, brothers, and sisters were likewise affected. The subjects of this form of tumor are often imperfectly developed physically and mentally, Hebra stating that all the cases observed by

FIG. 2.



him "were stunted in bodily growth and of more or less limited mental capacity." Later observations, however, have shown that there are numerous exceptions to this rule.

The number of tumors varies from a few to many hundreds. Mr. Hutchinson has reported a case, with a portrait, in which the face was the seat of hundreds of lesions, producing hideous deformity.

While there is a general agreement among those who have studied these tumors as to their anatomy, there is considerable diversity of opinion as to the part of the skin in which they have their origin. Rokitansky traced them to the deep part of the corium; Virchow believes that they originate in the connective-tissue framework of the

fat-lobules, and v. Recklinghausen asserts that they start from the fibrous sheath of the nerves. It is extremely likely that each one of these structures may serve as the starting-point for the fibrous hyperplasia, and that in some cases all these are involved.

Where the number of tumors is limited they may be removed by excision or the galvano-cautery, but when there are hundreds of them the treatment must be limited to the removal of the largest and most annoying.

HERPES ZOSTER AND ITS RELATION TO INTERNAL INFLAMMATIONS AND DISEASES, ESPECIALLY OF THE SEROUS MEMBRANES.

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IN September, 1890, I read a paper in the city of Denver, before the American Climatological Association, entitled "Is Herpes Zoster a Cause of Pleurisy and Peritonitis?" In that paper the histories of two cases were reported, one associated with pleurisy with effusion, and the other coming on with an attack of localized peritonitis. After reporting these cases I asked the following questions:

1. Was the internal disease a zoster eruption of the serous membrane?
2. Was the inflammation of the internal filaments of the nerves communicated to the pleura and peritoneum?
3. Did the internal inflammation cause the zoster?
4. Were both troubles independent and simply coincident?

I have since that time had the opportunity of observing other cases which may assist in settling at least two questions, and perhaps the third. I will repeat the two cases given in that paper, and then proceed to give other histories collected since which serve to bear upon this subject.

CASE I. *Herpes zoster with pleurisy and effusion.* (Quoted from my former paper.)—Some time ago I had under my care a maiden lady who, in answer to the question "How old are you," replied, in a low voice, "To you I am sixty, but to other people I am fifty-six." She was in the last stage of locomotor ataxia, so that she could scarcely walk. While in this condition she was attacked with a short, intermittent, neuralgic pain in her side, at the left base of the chest. A careful examination failed to develop any physical signs of pleurisy. In time the pain was followed by a well-marked zoster, which satisfied me that the pain was preliminary to the herpetic eruption. About the time this eruption appeared I noticed a pain with every inspiration, like the "catching pain" of pleurisy, associated with considerable constitutional disturbance, and a dry, hacking cough. A little later my attention was called to the diminished movement of the

left side of the chest, which was almost immobile, and a careful physical examination revealed an effusion which half-filled the side of the chest. This effusion continued until the chest was entirely filled. In this case the zoster and the pleurisy seemed to be coincident.

CASE II. *Herpes zoster with dry pleurisy.* (Reported by Dr. H. H. Doan, Resident Physician of the Philadelphia Hospital.)—M. D., aged forty-two years, white, painter, single; weight, 120 pounds. Family history revealed no facts of any interest.

Previous History (November 29, 1891). Used whiskey to excess. Had initial lesion which, from history given, was evidently specific; malarial fever seven years ago; erysipelas twice; inflammatory rheumatism; friction rub heard on right side of chest five years ago; has been a patient in the drunkard's ward three times for delirium tremens. Two weeks ago he had a severe catching pain in the right side; it felt like a stitch. He never went to bed. Five days later a number of lesions appeared along the track of the seventh or eighth intercostal nerve of the right side, evidently an attack of "shingles" following pleurisy. Temperature was taken for one week. It reached 99.3° F. once. In this attack, which was an evident case of pleurisy at the seat of a former pleurisy, which he had five years before, it would be difficult to tell positively whether the pain of four weeks ago was from the affected nerves or the inflamed pleura.

CASE III. *Zoster and pleurisy.*—Prof. George H. Rohe, of Baltimore, having had his attention drawn to the subject under consideration by my first paper, in 1890, informed me that he had in his hospital a case of herpes zoster of the chest, with a marked pleuritic friction sound underneath the seat of the eruption. The friction sound was so well marked that he used the case to demonstrate the sounds of pleurisy to his students.

Dr. Rohe could not tell which was the initiatory affection in this case.

CASE IV. *Herpes zoster and pleurisy with chronic Bright's disease.* (Reported by Dr. P. Janney, Resident Physician, Philadelphia Hospital.)—Bridget McC., white, female, aged sixty years, born in Ireland, housewife, married.

Family History. Father and mother died of old age. One brother died at the age of seventeen of "enlargement of the liver." Four sisters and one brother living and healthy.

Previous History. Healthy, as a child. In 1845 had influenza with the rest of her family. In 1848 had typhus fever, and was sick three or four months. In 1856 had jaundice for three months. No history of biliary colic. Has had one living child and three miscarriages. No venereal history. Five years ago her feet became swollen, had pains in back, passed frequently small quantities of urine. Ever since has had more or less swelling of feet. Admitted to the Philadelphia Hospital about two years ago with diagnosis of Bright's disease.

History of Present Disease. In the middle of October, 1891, she felt a catch at intervals in the right side in the nipple line. October 30, 1891, had a constant severe pain in the seventh and eighth interspaces on the right side, and a friction rub was heard on November 2d. Chest was examined and a herpetic eruption was found in the seventh and eighth interspaces, extending around almost to the sternum and over

the seventh and eighth dorsal vertebrae. There was constant pain and a slight moist crackle heard on inspiration and expiration.

Examination of Urine. Urine passed in large quantities; pale and of low specific gravity, containing a slight trace of albumin, but no casts; acid in reaction. All physical signs disappeared in two weeks, showing that the attack of pleurisy was acute. The pleurisy in this case seemingly preceded the external eruption.

CASE V. Herpes zoster complicating phthisis and chronic Bright's disease. (Reported by Dr. H. H. Doan, Resident Physician, Philadelphia Hospital).—J. H. Diagnosis, phthisis and Bright's disease. A native of Ireland, tailor by occupation, single, tall and thin.

Family History. Mother asthmatic for many years. Father died of Bright's disease. No tuberculous history.

Health usually good; drank very heavily and used tobacco to excess. No venereal history. Smallpox in 1866.

Had cough for two years, losing flesh; expectoration very slight until May, 1891. In January, 1891, had a continued attack of diarrhoea. July 20th, noticed eruption on left side of the upper part of the abdomen; no pain. Before this, although following the appearance of the eruption, there was severe pain, lasting two weeks. At one time during this attack he had slight hæmoptysis, lasting for one day. Lost appetite; bowels constipated. On admission to hospital there was an herpetic eruption extending from spine at junction of fourth rib around to the nipple, with points of tenderness. No friction sounds heard, nor could fluid be demonstrated. Since admission he has had frequent chills, occurring at irregular intervals, with decided irregularities of temperature. No plasmodium found in blood.

Urine, acid and albuminous, with many granular and hyaline casts. Numerous bacilli found in sputum.

With tubercular phthisis we have more or less plastic pleurisy, and many observers have reported cases of zoster complicating tubercular disease of the lungs, and in their cases the pleurisy antedated the eruption.

CASE VI. Herpes zoster with localized peritonitis. (Quoted from my former paper).—I had a case which served to bear upon the subject under discussion—apparently an inflammation occurring in another serous membrane simultaneously with zoster.

A woman, aged thirty-five years, who was about four months pregnant, had what I had diagnosticated as a localized peritonitis, occurring on the right side of the upper part of the abdomen, just below the margin of the liver. There appeared later at the same position a well-marked, clustered eruption. At about the same time a deep-seated soreness and tenderness appeared, extending to the median line. The febrile symptoms were marked; respiration and movement caused a great increase in the pain, which was constant; and pain was caused by the flatus which was present passing through the bowels. She barely escaped aborting. Later on the uterus increased in size and caused much pain, which was relieved, but not entirely so, when she was delivered. It was some months before the soreness entirely disappeared. The diseases seemed to be coincident in this patient.

CASE VII. Herpes zoster following catarrhal appendicitis.—M. L. had a sharp attack of catarrhal appendicitis with high fever, which ran its course in about seven days. The next week a sharp, lancinating pain

appeared, followed later by a zoster eruption on the right side of the abdomen, the right hip, and the upper part of the thigh. The localized peritonitis preceded the skin affection in this case.

CASE VIII. *Herpes zoster following arthritis and effusion of right knee.*—Judge A. Right knee-joint was much swollen and painful from rheumatic arthritis. This condition had continued for several weeks when an eruption of herpes zoster came on the outside of the knee and popliteal space, and ran the usual course of that affection. The eruption did not encircle the knee.

Here we find a case of hydro-arthritis of the knee-joint of six weeks' standing, the skin around it being secondarily affected by zoster.

CASE IX. *Influenza; meningitis; pleuropneumonia followed by herpes zoster.*—F. L. had an attack of influenza followed by meningitis and later on by a pleuropneumonia of the right lung. Late in convalescence he had a marked attack of herpes zoster situated on the right side of the lower anterior chest and upper part of the abdomen.

CASE X. *Hæmaturia and zoster.*—S. A. had, in May, 1899, an eruption of well-marked zoster over and above the position of the left kidney, extending around to the umbilicus and below it. In the third week of August, two and a half months later, he began to pass blood in his urine. This continued for months. There was no pain or other discomfort over the kidney or bladder except when he passed a clot, and then he felt a slight pain and discomfort at the symphysis pubis.

The quantity of blood constantly varied and continued for months. The color of the blood was generally dark or quite black. He had no constitutional disturbances. Two years ago, September, 1897, he had a similar eruption on his face, which lasted about ten days or two weeks; at the same time he told the doctor that his urine was high colored. The doctor examined it and said it was blood. This hæmorrhage lasted four or five weeks. He never had any eruption or hæmaturia at any time other than these two occasions.

RECAPITULATION. 1. In Cases I., II., III., and VI. the herpes zoster seemed to come on at the same time as the internal inflammation.

2. In Cases IV., VII., VIII., and IX. the affection of the serous membrane preceded the eruption.

3. In Case V. the zoster appeared during the course of a chronic pleurisy of tubercular phthisis.

4. This résumé seems to indicate that inflammation of the serous membrane precedes the zoster; therefore, we may reason that the inflammation caused the eruption.

5. These conditions occur together or follow one another too frequently to be simply coincident.

I feel sure that the cases here reported settle at least some of the problems that I suggested in my former paper:

1. Was the internal disease a zoster eruption of the serous membrane?
2. Was the inflammation of the internal filaments of the nerve communicated to the pleura and peritoneum?
3. Did the internal inflammation cause the zoster?
4. Were both troubles independent and simply coincident?

While on this subject of zoster and internal diseases I will take the opportunity to state that I had a case, in 1874, of œsophageal cancer that had, late in the disease and over its seat, a complicating zoster eruption encircling the left side of the chest at the margin of the ribs. The eruption, it seemed to me, might be caused by the cancer, as the œsophagus is located to the left of the border of the vertebræ, near the roots of the nerves.

In 1890 to 1893 I had quite a number of cases of influenza and catarrhal fever complicated with zoster, generally coming on with the less acute influenzal attacks. A number of my friends reported to me one or more attacks of the same kind in their cases of acute and sub-acute influenza.

The subject being, I believe, a new one, I shall feel repaid if the attention of future observers is called to this interesting condition.

THE CHANGES OCCURRING IN STRIPED MUSCLE IN THE NEIGHBORHOOD OF MALIGNANT TUMORS.¹

BY FRANK P. ANZINGER, A.M., M.D.,
OF ANN ARBOR, MICH.

By far the greater part of the investigations made during the last decade into the nature of malignant tumors have been along the lines of their supposed parasitic origin. Yet, with all of the immense effort and labor extended in this direction, we are to-day not one step nearer the solution of the problem of the etiology of these growths. There is no sufficient evidence to prove that the peculiar bodies found in carcinoma cells are parasites, either bacterial, protozoic or blastomycetic, each of which they have from time to time been confidently proclaimed to be. It must be acknowledged that we are no nearer the demonstration of this problem than we were ten years ago, and the oft-repeated failures should convince us that if malignant neoplasms are parasitic in origin we must arrive at the discovery of the parasite by following lines different from those hitherto pursued. In the meantime, while this field of research is being so largely worked, other aspects of the tumor problem are being more or less neglected, particularly those along chemical lines.

Very little work has been done with the comparative chemistry of tumor cells and cells of normal tissues, or with the chemistry of the products of tumor-cells. The changes occurring in the cells in the

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neighborhood of malignant tumors have also received but scanty attention.

In 1867, Otto Weber made observations on cancer in the pectoral and sternomastoid muscles. He described the manner of growth of carcinoma into the muscle, the presence of cancer-cells in the muscle-fibres, and the destruction of the muscle as the result of suppuration, but the finer structural changes were not observed. As a result of his study he concluded that cancer-cells were derived from muscle-elements. In 1870, Volkmann described the appearances of cancer in the pectoral muscle, the manner of invasion of the muscle by cancer-cells, the formation of cancer nodules, and a peculiar change in the muscle protoplasm, to which he applied the term *lacunar erosion*. He pointed out the fact that this change was due to the pressure of cancer-cells, and concluded that carcinoma cells invade muscle, but do not originate from muscle-elements.

The first careful study of the minute changes occurring in striped muscle in the neighborhood of malignant growths was made by Schaeffer in 1886. The material examined by him consisted of 3 cases of epithelioma of the lip, 2 cases of carcinoma of the pectoral muscle, 3 cases of carcinoma of the tongue, and 4 cases of sarcoma. The manner of invasion of the muscle and the gross appearances of muscle changes are first described by him, then the finer changes as produced both by sarcoma and carcinoma. The special changes observed were: Distortion of the fibres, atrophy, cloudy swelling, fatty degeneration, waxy change, fissuring, changes in staining, nuclear proliferation, hypertrophy, small-celled infiltration, and increase of the perimysium. He compared these changes with those found in other conditions, and concluded that the changes produced by tumors were not characteristic, but could be produced by other pathological agencies.

Since the work carried out by these three observers represented the sum total that had been done in this direction, it seemed worth while to extend this research with a view to settling several questions left untouched by them, particularly with reference to a comparison of the changes produced by sarcoma and carcinoma. While this work was in progress there appeared in April, 1900, an article upon the same subject by Fujinami. As this is the last and most important contribution in this line, I give his conclusions somewhat at length. These are based upon the study of 20 cases of sarcoma and 17 of carcinoma infiltrating muscle. The manner of invasion and the changes occurring in the contractile substance and muscle nuclei, and the fate of the primitive muscle bundle, are considered in detail. The results of this study are as follows:

1. The invasion of the muscle takes place in different ways, both through the lymph-spaces, lymph- and bloodvessels, and the sarcolemma.

The invasion of the sarcolemma by tumor-cells occurs most frequently in the case of carcinoma, and is especially pronounced when the direction of extension of the tumor is parallel with the axis of the muscle-bundle. It is seldom seen in sarcoma, occurring only in the case of the small round-cell variety. The penetration of the sarcolemma is dependent upon the biological relations of the tumor-cells, their direction of growth, and the variety of change in the muscle-substance.

2. The changes in the primitive muscle-bundle are of manifold variety, all possible changes occurring. Simple atrophy is the most frequent; and the most interesting are the nuclear proliferation, "ampullar atrophy" and formation of giant-cells. Those changes formerly regarded as regenerative in nature are to be regarded as being essentially degenerative, the primitive muscle-bundle in a certain stage of retrogression assuming relations similar to those at the beginning of its development.

3. The muscle-fibres show varied and irregular changes in thickness and length.

4. The changes produced in the primitive muscle-bundle by tumor-cells cannot be referred alone to mechanical pressure, but the changed biological relations of these cells must also produce chemical effects.

5. In the interstitial connective tissue there is often a reaction, as shown by leucocyte infiltration, fibroblastic proliferation, and proliferation of the intima of the vessels. These changes may also secondarily affect the primitive muscle-bundles. The infiltration of the perimysium is usually much more marked in the periphery of carcinoma than of sarcoma.

6. Cell-forms (sarcolytes, sarcoplasts) arise from the changed muscle-bundles, and under certain conditions it is probable that these take part in the formation of tumor-cells.

7. A connective-tissue-like change of the muscle-fibres is assumed.

8. The reaction of muscle to the invasion of a malignant tumor may be very varied according to the local conditions. The muscle does not play a passive rôle simply.

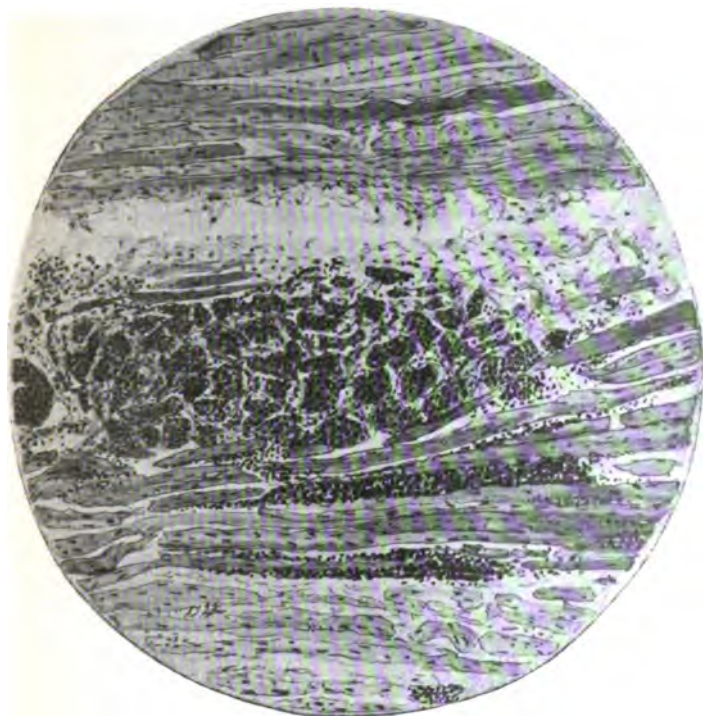
9. The fact that the muscle-fibres possess a characteristic appearance and show various changes is of practical value in the diagnosis of tumors, in so far as it aids in establishing the biological properties of the tumor-cells and their direction of growth.

10. There is no absolute difference between the muscle changes produced by sarcoma and carcinoma.

As the work begun by me before the appearance of Fujinami's paper had led me to conclusions differing from some of those of his, it was continued with renewed interest, and the results are given below. For the original suggestion of the work I am indebted to Dr. Warthin, who gave me access to the material of his laboratory, and my research has,

from time to time, been controlled by him. The material examined consisted of 30 cases of cancer of the breast infiltrating the pectoral muscle, 15 cases of epithelioma of the lip, 1 of cancer of the tongue, 1 case of lupus-carcinoma of the cheek, 2 secondary carcinomas of the diaphragm, 2 cases of sarcoma of the pectoral, 2 fibrosarcomas of the rectus abdominis, 2 sarcomas of the inferior maxilla, 1 sarcoma of forearm, 3 of femur, 1 of tibia, and 1 of general sarcomatosis of the skin. Examinations were made both of hardened and fresh material. Alcohol,

FIG. 1.



Carcinomatous nodule in pectoral muscle, showing manner of invasion.
(Leitz obj. 3, eye-piece III.)

mercuric chloride, Flemming's, Müller's, and Zenker's fluids were used as fixing agents. The muscle was cut both transversely and longitudinally. Celloidin and paraffin embedding were both employed; and the following stains were used: hæmatoxylin and eosin, Van Gieson, carmines, polychrome, methylene blue, orcein, Russell method for cell-inclusions, Weigert nerve method, Mallory's reticulum method, and Plimmer's method.

MANNER OF INVASION. 1. *Carcinoma in Pectoral Muscle.* The primary carcinoma of the mammary gland advances by direct expan-

sion until the fascia of the pectoral muscle is reached. The latter serves as a barrier to further advance, and the carcinoma usually reaches the muscle through the lymphatics. Uninvolved fascia may separate the main mass of the tumor from the muscle when in the latter there are metastatic foci of tumor-cells in the lymph vessels about the bloodvessels. Serial sections show that these are often not metastases, but direct extensions of the growth through the vessels. When the nodules in the muscle reach a large size the muscle-fascia may be destroyed, but in the earlier stages it is rarely involved. Its normal appearance is, however, no evidence that there are no tumor nodules in the muscle beyond it. The muscle may be strewn with nodules varying in size from a pin-point to a walnut. They resemble the primary in structure. Arising in the interstitial tissue, they push the muscle-fibres apart and produce in it the most varied pathological changes. As the nodules increase in size there is more or less complete destruction of the fibres. Remains of these may be found in the cancer nodule. In infiltration with scirrhus carcinoma there is a marked proliferation of the perimysium, and this leads to the production of marked changes in the primitive bundle. Small islands of muscle-protoplasm and small groups of cancer-cells are surrounded by a dense mass of connective tissue. Invasion by way of the bloodvessels is rare. When the muscle is once reached further extension takes place through the lymph-spaces of the interstitial tissue and also through the sarcolemmar tubes.

2. *Epithelioma of the Lip.* The invasion of muscle is favored in this region by the structure of the muscle-fibres, these being short and irregular and the perimysium of a very loose texture. The irregular columns of epithelial cells fill in the meshwork of the perimysium; the younger cancer-cells infiltrate between the separate muscle-fibres and destroy these as they increase in size. Leucocyte infiltration is usually marked, and it is difficult to separate the leucocytes from the young cancer-cells. Cells undergoing hyaline change or cornification may also be mistaken for remnants of muscle. Small fragments of muscle-plasma and nuclei may sometimes be found in the central portion of a cancer nest; this appearance is, however, explained by the plane of the section, and is not due to an encapsulation of muscle by cancer-cells as has been supposed. Penetration of the sarcolemmar tubes by the young cancer-cells also occurs, but is more rare than in the case of breast cancer.

3. The invasion of muscle by secondary carcinoma in other regions is similar to that in these two places, the difference depending upon the local conditions in the muscle and the character of the tumor-cells.

4. *Sarcoma.* The invasion of muscle by sarcoma takes place by direct extension in the form of outgrowths of the main mass penetrating the muscle-bundles, extension of slender cords of cells between the

muscle-fibres, and also by lymphogenous and hæmatogenous metastasis. There is always much less reaction in the perimysium of muscle invaded by sarcoma than there is in the case of carcinoma. The ingrowths of sarcoma replace the perimysium, at first separating the muscle-bundles, which may show but slight change. As the proliferation of the tumor progresses there is gradual atrophy and disappearance of the muscle-fibres. These may, however, persist for a long time, and not infrequently small bits of muscle may be found scattered throughout the tumor. Many of these may appear as regeneration forms. In the case of universal sarcomatosis of the skin, large subcutaneous nodules were present which resembled greatly enlarged lymph-glands. On section these were found to contain in their central portion the remains of muscle-fibres. Round-cell sarcoma produces a more rapid destruction of muscle than the spindle-cell variety. On the whole the effects of the invasion of muscle by sarcoma appear to be chiefly mechanical, as the surviving bits of fibres usually show but little change in structure. The rapidity of growth in some cases causes a rapid disappearance of the muscle without preceding retrograde changes.

CHANGES IN MUSCLE PRODUCED BY INVADING MALIGNANT TUMOR.

1. *Atrophy.* This is the most frequent and constant change. It may be either mechanical or chemical in nature. In the case of scirrhus carcinoma, epithelioma, and sarcoma, the atrophy is limited to the area of muscle invaded by the growth, and is chiefly the result of pressure. In the case of non-scirrhus cancers of the breast, the atrophy may extend into the pectoral a long distance from the advance cells of the tumor, and may affect large bundles of muscle in which no cancer-cell is present. This cannot be explained as mechanical in origin; on the other hand, it is probable that it is due to disturbed biological or chemical relations. Both forms of atrophy may be present in the same case, but the chemical form is more characteristic of carcinoma. In scirrhus carcinoma, epithelioma, and sarcoma the length of the muscle-fibre is very frequently preserved, while its thickness is reduced to that of a mere thread. In the rapidly growing forms of breast cancer the fibres of the pectoral show great variation also in length.

2. *Hypertrophy.* The occurrence of hypertrophic fibres in the neighborhood of malignant growths was maintained by Schaeffer, who made measurements of the fibres in 2 cases of fibrosarcoma. But even in the case of actual measurements it is hardly possible to make such a diagnosis, inasmuch as the standard of size shows such individual differences. Large fibres occur very frequently among the atrophic ones, but this enlargement in the majority of cases is only relative. The size of the fibre is also increased in certain forms of degeneration, notably cloudy swelling.

3. *Changes in Morphology.* Very marked and varied changes in the morphology of the fibres occur. The parallel arrangement of the fibres

may be changed; they may be bent at various angles, often assuming a spiral form. The sarcolemma may be retracted from the muscle-substance, the latter being formed into wavy bands or broken up into scroll-like figures.

4. *Changes in Muscle-substance.* (a) *Alteration in the Striation.* In the atrophic fibres the cross striation is usually very sharply defined, the alternating light and dark bands being very narrow. Very frequently there is a longitudinal fibrillation with loss of the cross striation. The plasma is divided up into its fibrillar elements, and often at the cut end of a fibre these may be seen protruding from the sarcolemma in a brush-like manner. Except in the case of cloudy swelling and Zenker's necrosis, the longitudinal striation persists as long as the plasma remains intact.

(b) *Vacuolization.* This is a frequent change, and in some cases may be the only one present. It occurs often as a very early sign of degeneration. The vacuoles appear as clear or cloudy spaces in the plasma, which contrast strongly with the surrounding stained plasma. They may be sharply outlined as if punched out, or their border may be irregular, in many cases appearing as if fringed. They vary much in size and shape, and at times communicate by delicate grooves or channels. When located at the periphery of the fibre the sarcolemma may bridge over the vacuole or become invaginated into it. In the majority of cases they are to be interpreted as due either to hydropic degeneration or Zenker's necrosis.

(c) *Fissuring* Fragmentation of the fibre may occur in any plane, transverse, longitudinal, or irregular. These changes may be partly of the nature of an artefact, but are undoubtedly pathological to a large extent, as they occur in the most carefully fixed specimens.

(d) *Waxy Degeneration.* Zenker's necrosis is of very common occurrence, particularly in the case of mammary cancer invasion of the pectorals, where it very often occurs at some distance from the tumor. Schaeffer maintained that this form of necrosis was not common near tumors, but in my cases it has been found both of frequent and extensive occurrence.

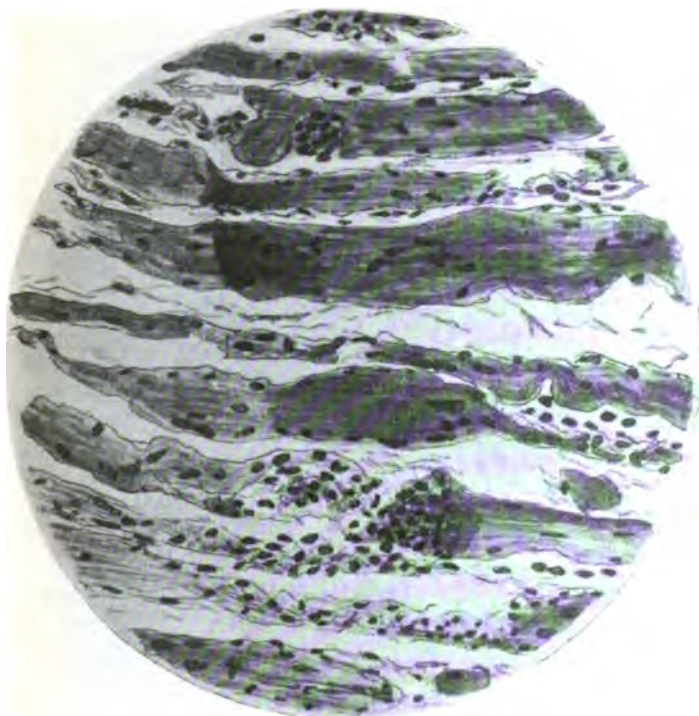
(e) *Cloudy Swelling.* This is of very frequent occurrence. The fibres are swollen, granular, stain heavily, and show a tendency to take both nuclear and diffuse stain (polychromatophilia).

(f) *Fatty Degeneration.* No fatty change was found in muscle in the vicinity of tumors, though repeated search was made with the use of osmic acid. Weber and Schaeffer state that it is of common occurrence under these circumstances, but Fujinami found only traces of it in the examination of fresh material.

Lacunar Erosion. The appearances described by Volkmann under the head of "lacunar erosion" occur not infrequently in invading

carcinoma of the pectoral. Muscle-fibres which otherwise may appear normal present one or more cup-shaped indentations of the plasma at some point along its margin. These depressions usually contain eight to twelve nuclei, which are small and irregular in outline, but from general appearances must be considered to be muscle nuclei. Other fibres may be found in which the muscle-substance is intact except at one or both poles, where it is interrupted by a concavity which may be shallow or deep. In these depressions are numerous deeply staining

FIG. 2.



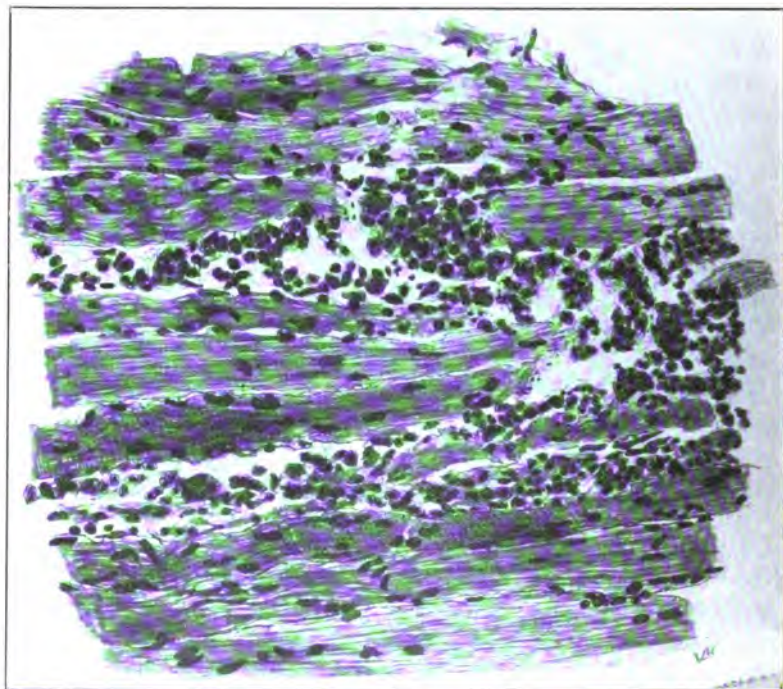
Pectoral muscle near periphery of mammary carcinoma, showing lacunar erosion and ampullar atrophy. (Leitz VII., eye-piece III.)

nuclei packed so closely that, with the low power, their character cannot be made out. Examination with high powers shows that the sarcolemma is prolonged for some distance beyond the muscle-substance, the two lines representing the cross section of the sarcolemma finally converge and meet. These apparent erosions are, therefore, explainable as being portions of the fibre where the muscle-substance has entirely disappeared as the result of degeneration, while the muscle nuclei have undergone proliferation. Other fibres show earlier stages of this process; beneath the sarcolemma the remains of the muscle-substance,

in the shape of granular detritus tinged with diffusely staining chromatin, are found. These apparent erosions are in the majority of cases not due to the presence of tumor-cells, but in some instances a group of the latter is found pushing the sarcolemma into the depression and presenting the appearance which led Volkmann to believe that the erosions were due to an "eating-out" action on the part of the tumor-cells.

5. *Changes in the Muscle Nuclei.* (a) *Proliferation.* This is a very common change, and presents many features of unusual interest.

FIG. 3.



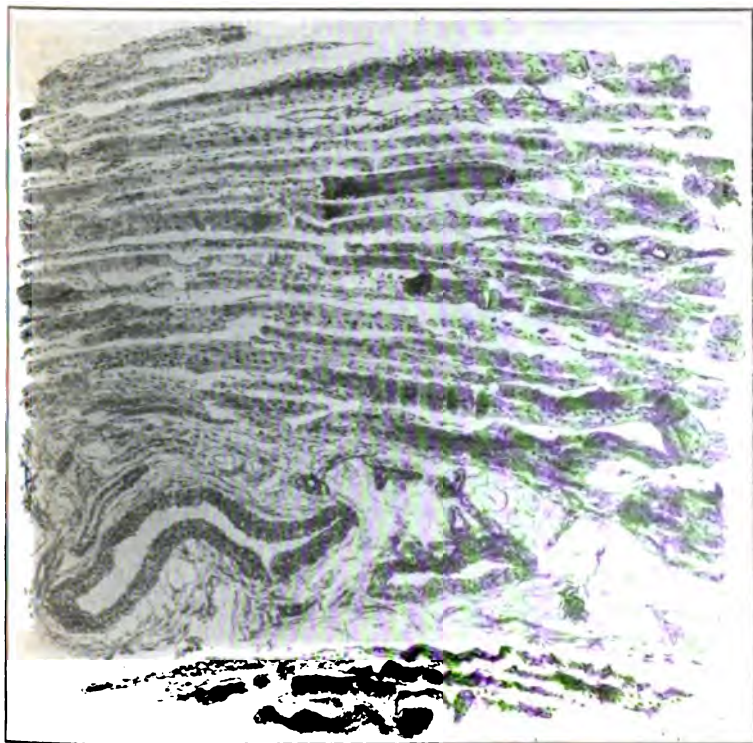
Invasion of pectoral muscle by mammary cancer, showing changes in muscle-substance and nuclei. (Leitz VII., eye-piece III. Camera lucida, reduced 1/3.)

Increase in the number of the nuclei leads to the formation of long nuclear chains, often sixty nuclei being counted in one chain. At other times they may be so numerous as to form a solid cord. In transverse sections the nuclei are often seen arranged in clumps replacing a portion of the plasma just beneath the sarcolemma. The central portion of the fibre may contain many nuclei. The collections of nuclei found in the cup-like erosions of the muscle-substance beneath the sarcolemma are undoubtedly muscle nuclei which have multiplied at these

points. Waldeyer has described appearances in diseased muscles in which the protoplasm is entirely replaced by muscle nuclei (Muskelschläuche). In a few instances these were found by me, but were not so typical as suggested by some observers. No mitotic figures were found in any of my specimens, the proliferation appearing to be by direct division.

(b) *Changes in Morphology.* The normal muscle nucleus is oval in shape, and the average measurement is 7 to 11 μ in length and

FIG. 4.



Changes in pectoral muscle at some distance from edge of carcinoma. (Leltz III., eye-piece III. Camera lucida, reduced 1/3.)

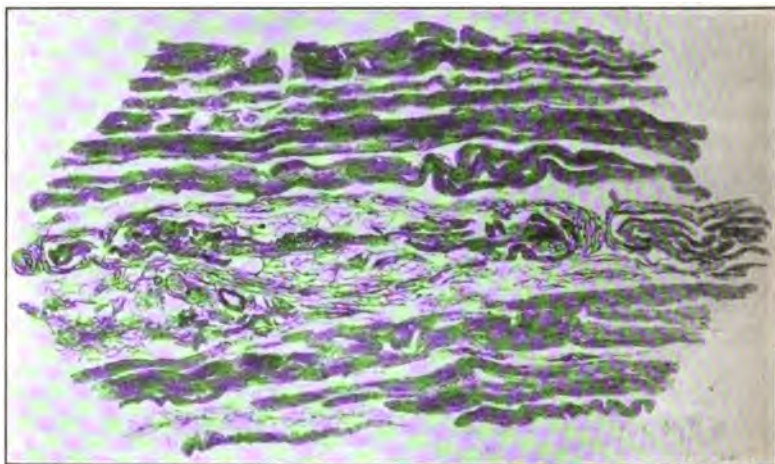
3 to 4 μ in breadth. Near malignant tumors they become very much elongated, some nuclei measuring 40 μ in length. Others are very short, some atrophic, and others hypertrophic. They may be very much altered in shape and staining power. The nuclei survive the destruction of the muscle-substance, but ultimately disappear also.

6. *Changes in the Sarcolemma.* The behavior of the sarcolemma during the destruction of the muscle protoplasm has already been men-

tioned. Its final fate is degeneration and complete disappearance though some investigators believe that it may take part in the proliferation of the perimysium. After the disappearance of the muscle substance the sarcolemmar tube may collapse or it may be entered by leucocytes and tumor-cells. Not infrequently it appears to have been distended by fluid.

7. *Changes in the Perimysium.* In infiltration of the perimysium with cancer-cells the connective tissue takes part in the formation of the stroma of the cancer nodule. The proliferation of the perimysium takes place, however, to a much greater extent in the case of spindle

FIG. 5.



Muscle spindle in pectoral muscle near invading carcinoma, showing nuclear proliferation in intrafusal fibres near equator. Neighboring muscle shows retrograde changes. (Lentz III, eye-piece III. Camera lucida, reduced 1/3.)

cell and fibrosarcomas, epithelioma, and scirrhus cancer than in carcinoma simplex or medullare. Secondary changes such as fatty infiltration, leucocyte infiltration, hemorrhage, œdema, necrosis, etc., are of common occurrence in the interstitial connective tissue.

8. *Vascular Changes.* Obliterative endarteritis and periarteritis are of very frequent occurrence, and in the majority of instances are due to local changes. Proliferation of the endothelium of the lymph channels was seen in a few cases. The vascular changes are most pronounced in the larger vessels.

9. *Changes in Nerve Elements.* Sections stained according to Weigert's method showed no change. Fujinami describes a connective-tissue increase in the nerve-trunks, but this was not seen in my material.

Special search was made for possible changes in the muscle-spindles, as these organs have not hitherto been considered in this connection. Forster studied the behavior of the spindle in many pathological conditions, such as acute myelitis, progressive muscular atrophy, etc. With the most marked change in the muscle he found the spindle remaining normal. Sherrington found no changes in the spindle to be produced by cutting muscles and tendons. Batten found a spindle which stained poorly in a case of tabes dorsalis, and in a case of atrophy and degeneration of the upper arm muscles, due to injury of the brachial plexus, the spindles were atrophic, the intrafusal fibres showed granular degeneration, the spindle sheath hazy, and the nerve-fibres of the spindle degenerated. Grünbaum found a peculiar hyalin deposit around the intrafusal fibres in a case of pseudohypertrophy. In my own work many preparations were examined for the study of the spindle. The nerve-trunks leading to the spindle were normal in every case examined. In one case of pectoral carcinoma longitudinal sections of a spindle showed a marked nuclear proliferation in the intrafusal fibres near the equator, but as similar appearances have been seen by several observers in apparently normal spindles the significance of this appearance is not clear.

10. *Small-celled Infiltration.* Leucocyte infiltration is found to some extent in or about all malignant growths. It is very marked in the case of epithelioma of the lip, but occurs to a very slight degree about invading cancer of the pectoral. The leucocytes are for the greater part collected about the bloodvessels and along the border-line of the invading growth. They are usually found intermingled with young tumor-cells, and it is difficult and often impossible to distinguish between the two.

11. *Occurrence of Mast-cells.* Both leucocyte and connective tissue mast-cells were at times found abundantly in the areas where small-celled infiltration was most marked. In a few instances mast-cells were found inside the sarcolemmar tube in company with other forms of leucocytes. In some of the cases of mammary cancer large numbers of eosinophiles were present in the areas of small-celled infiltration of the perimysium.

12. *Regeneration of Muscle.* In two-thirds of the cases of sarcoma and in three cases of epithelioma cell-forms identical with those seen in the repair of muscle were found in large numbers. They were never seen in the pectoral muscle in carcinoma, but were very numerous in a case of spindle-cell sarcoma of that muscle. The new fibres are found in the stroma of sarcoma and in the granulation tissue at the periphery of lip-epithelioma. The newly-formed fibres are usually short, and are spindle or club-shaped, or irregular in form. Their protoplasm stains well with eosin. The nuclei are numerous and are usually

grouped at one extremity. Many of the new fibres are vacuolated, their nuclei large and vesicular, and their protoplasm taking both nuclear and diffuse stains. These evidences of degeneration are exactly similar to those occurring in the new fibres after repair of muscle wounds. Fujinami, however, interprets the forms found near malignant tumors as being essentially degenerative in nature and not regeneration forms. He explained their formation as follows: The degenerating fibre is constricted at regular intervals, and this constriction goes on until the fibre is broken up into fragments, the nuclei at the same time proliferating ("ampullar atrophy"). The muscle-fragments he calls "ampullæ," and these he considers to be larger and to possess larger and more deeply-staining nuclei than the true muscle-buds in regeneration. While it is very probable that muscle giant-cells may be formed by the disintegration of the fibre, this explanation will not hold for my cases. In these, serial sections show that the giant-cells are really muscle-buds and not muscle-fragments. Their arrangement is not at all in accord with the idea that they are degenerating remains of old muscle. The presence of vacuoles in protoplasm and nuclei is also no argument that they are essentially degeneration forms, as it is well known that the newly-formed fibres in the repair of muscle wounds also quickly undergo degeneration, and do not lead to the production of muscle capable of functioning. I regard the appearances found in my cases as identical with those found in muscle repair.

SUMMARY OF CHANGES WITH REFERENCE TO THE DIFFERENCE BETWEEN THOSE PRODUCED BY SARCOMA AND CARCINOMA. 1. *Atrophy*. This is more marked in the case of carcinoma. In sarcoma the muscle may show an extreme degree of pressure atrophy at the border of the tumor, but in carcinoma there is usually a general atrophy, even of fibres at some distance from the cancer. This can be interpreted as chemical or toxic atrophy. I have not seen it in sarcoma.

2. *Hypertrophy*. This was not seen in any of the cases of carcinoma, but in a case of spindle-cell sarcoma of the pectoral the muscle-fibres appeared unusually large.

3. *Morphological Changes*. These are much more marked in the case of carcinoma, the muscle-fibres often assuming very grotesque forms. In sarcoma this distortion occurs to a very slight degree. The changes in carcinoma are so constant, even with the most careful technique, that they cannot be regarded as artefacts. The finding of mast cells and leucocytes within the sarcolemma is also evidence that these changes occurred during life.

4. *Change in Striation*. Longitudinal fibrillation was present in all of the carcinoma cases; in sarcoma it was seen only rarely.

5. *Vacuolization*. This is a frequent and early change in both sarcoma and carcinoma.

6. *Retrograde Changes.* Early stages of cloudy swelling are common in the case of sarcoma, but in carcinoma the degeneration is more frequent and much more severe. Zenker's necrosis is of much more frequent occurrence in the case of carcinoma, in the pectoral often occurring at a distance from the tumor.

7. *Lacunar Erosion.* This form of degeneration of the muscle-substance is almost characteristic of carcinoma, being found only in one form of sarcoma, small round-cell of the skin. It may be emphasized once again that this so-called erosion is independent of any mechanical action of tumor-cells; but is a primary change in the muscle-substance.

8. *Nuclear Proliferation.* An increase in the number of the muscle nuclei is very commonly found in carcinoma, but often absent in sarcoma, even when other degenerative processes are present. This is in accord with the fact that the muscle-substance in sarcoma shows a much less tendency to retrograde changes. Nuclear proliferation goes hand-in-hand with degeneration of the muscle-substance. This apparent paradox may be explained on the hypothesis of a reversion to an embryonal state antecedent to degeneration, or that the normal muscle-substance has an inhibitory influence over the nuclei, the removal of which in degeneration of the former permits an unrestrained proliferation of the latter. Changes in size and form of the nucleus go hand-in-hand with nuclear proliferation.

9. *Sarcolemma.* Changes in this are more manifest in carcinoma because of the more marked changes in the protoplasm.

10. *Vascular Changes.* These are very common in both carcinoma and sarcoma; if anything, more marked in the case of the former. Hypertrophy of the endothelium occurs more commonly in sarcoma.

11. *Small-celled Infiltration.* This is very marked in lip-epithelioma, but in pectoral carcinoma it is very often very slight or entirely absent. In sarcoma the leucocytes were not numerous in the muscle and were usually limited to the perivascular areas of the perimysium. The enormous infiltration in lip-epithelioma may be due to secondary inflammation or infection, the location favoring the occurrence of these events.

12. *Muscle Regeneration.* As mentioned above, this was found only in the cases of sarcoma and lip-epithelioma. It was never seen in pectoral carcinoma.

From this summary it is evident that the muscle changes occurring near carcinoma and sarcoma are not identical, as claimed by Schaeffer and Fujinami. This observation is of great interest because of the significance which may be attached to it. The essential difference between the two may be briefly stated as follows: Retrograde changes are marked in the case of carcinoma; in sarcoma they occur to a very slight degree, or may be entirely absent. In sarcoma the atrophy is apparently only mechanical, while in carcinoma it occurs at a distance

from the tumor under conditions which make it very probable that it is of a toxic nature or due to a withdrawal of nutrition. The morphological changes in the muscle-fibre are more marked in carcinoma than in sarcoma, and this may be explained in the same way. Nuclear proliferation is marked in the case of carcinoma, in sarcoma it occurs to a slight degree, and in the majority of cases is absent. Regeneration forms are numerous in sarcoma, but were never found in pectoral carcinoma. Their presence in lip-epithelioma may be due to the extensive inflammatory changes present.

Comparing the changes produced in muscle by malignant tumors with those due to other causes, we find that the differences are of degree rather than of kind. Krauss, in experimental work on animals, found that sections of the nerve-trunks produced atrophy, nuclear proliferation, cloudy swelling, loss of striation, and vacuolization. Normal and diseased muscle-fibres were found side by side. Changes similar to these occur in acute anterior poliomyelitis, the bulk of the muscle being replaced by fat and connective tissue. In primary myopathy the fibres are atrophic or completely degenerated, occasionally hypertrophic (?) fibres are seen. The cross striation is indistinct, longitudinal fibrillation is marked, and fissuring, vacuolization, increase of connective tissue, fatty infiltration, and leucocyte infiltration are also present. In progressive muscular atrophy the fibres are much reduced in thickness, their striation is coarse or indistinct, with a tendency to fissuring; longitudinal fibrillation is common, and there is marked fatty degeneration, the sarcolemma often being filled with fat-droplets.

If the changes produced by malignant tumors show nothing specific in character, to what may the great difference in degree between those due to carcinoma and sarcoma be referred? The local changes in muscle invaded by tumors may be explained as due either to direct pressure of the tumor-cells or to chemical and nutritive influences. The difference between the two types may be the result of their different biological relations. Carcinomata are epithelial in origin, sarcomata mesoblastic, the invaded tissue muscle is also mesoblastic. An abnormal proliferation of any tissue means marked metabolic changes, the consumption of much nutritive material, and the giving off of much waste product. Tumor-cells behave in a manner similar to that of parasites, invading another complexus of cells, and flourishing at the expense of the latter. The tissue invaded suffers both from loss of nutrition and from the effects of the metabolic products of the more actively proliferating tumor-cells. Whether specific poisons are produced by the latter we are not yet in a position to say, but the changes about carcinoma are for the greater part to be interpreted as toxic in nature. In the invasion of muscle by sarcoma the antagonism between the two tissues must be less marked than in the case of carcinoma. The

sarcoma cells are genetically mesoblastic, and have similar capacities and tendencies to those of muscle. Hence, a sarcoma may infiltrate the entire perimysium and produce no changes except the simple atrophy due to mechanical pressure. In the central portion of a large sarcoma mass remains of muscle-fibres may still be present and may present no retrograde changes. Such an occurrence I have never seen in carcinoma; here the invaded muscle is quickly destroyed. The attempts at regeneration found in sarcoma are also an evidence of the less injurious effects of the latter. Carcinoma is, therefore, more antagonistic of muscle than sarcoma, because of its different biological relations, the different nutritive and metabolic conditions, and also probably because of the formation of poisonous products by the cancer-cells.

CONCLUSION. 1. The changes produced in striped muscle by invading malignant tumors are :

A. Retrograde.

1. Atrophy.
2. Morphological changes.
3. Changes in protoplasm.

Vacuolization.

Fissuring.

Longitudinal fibrillation.

Lacunar erosion.

Cloudy swelling.

Simple necrosis.

Zenker's necrosis.

Polychromatophilia.

4. Changes in nucleus.

Morphological changes.

Proliferation.

Vacuolization.

5. Perimysium.

Atrophy.

Hyperplasia.

Leucocyte infiltration.

Hemorrhage.

Edema.

Necrosis.

6. Vascular changes.

Endarteritis.

Periarteritis.

Hypertrophy and hyperplasia of endothelium.

B. Regenerative.

1. Hypertrophy (?).

2. Regeneration forms.

2. These changes are not specific, but are produced by conditions other than malignant tumors.

3. The changes produced in muscle by invading carcinoma are more marked than those produced by sarcoma, but do not differ essentially in kind. Those produced by sarcoma are chiefly mechanical; those due to carcinoma are mechanical, nutritive, and metabolic. Evidences of toxic action are more marked in the latter than in sarcoma. Carcinoma acts more as an active injurious foreign body upon muscle than does sarcoma.

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A CASE OF OPHTHALMIA NEONATORUM CAUSED BY THE DIPLOBACILLUS OF MORAX AND AXENFELD.

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THAT the gonococcus is not the only cause of ophthalmia neonatorum is a conceded fact. The diplococcus, the streptococcus, the bacillus coli communis, and the micrococcus aureus also produce purulent conjunctivitis in the newborn. The last statistics of Groenouw show that in one hundred cases of ophthalmia neonatorum the gonococcus was present in only forty-one. All observers agree, however, in the opinion that when the disease is due to the gonococcus it usually assumes a very severe character.

Among the several micro-organisms described in connection with purulent conjunctivitis of the newborn the bacillus of Weeks and the diplobacillus of Morax and Axenfeld have never been mentioned. Having met with an unusual case of ophthalmia neonatorum caused by the last-named bacterium, which as a general rule is only found in cases of subacute catarrhal conjunctivitis, I have deemed it of sufficient interest to report.

Clinical History. E. L., female, three days old, child of healthy parents, presented slight œdema of the lids and a small amount of purulent exudate in both eyes, the symptoms being more marked in the right eye. There was no chemosis. The parents asserted that their two other children had shown, shortly after birth, similar symp-

toms, which, in the case of the elder one, were so severe as to threaten the destruction of one eye.

I prescribed protargol in 10 per cent. solution, increasing to 20 per cent. solution, and frequent washings with warm boric-acid solution, with the result that the oedema of the lids rapidly disappeared and the purulent exudate decreased very noticeably. I instructed the mother to continue the protargol instillations, and did not see the child during the following ten days. When called again I found that there was yet some purulent exudate in both eyes, and that the stump of the cord and the left ear were also suppurating. Otherwise the child seemed in perfect health. A much-desired bacteriological examination could not be made at the time. The suppuration of the ear and stump ceased under appropriate treatment, but the ophthalmia was rebellious. Noticing that not even applications of nitrate of silver in 2 per cent. solution had any effect, I made, at the first opportunity, a bacteriological examination of the conjunctival exudate, and found to my surprise a pure culture of the diplobacillus of Morax and Axenfeld. Knowing the almost specific action of zinc salts on this micro-organism, I prescribed sulphate of zinc in a 2.5 per cent. solution, to be instilled once a day. Under this treatment the ophthalmia, which had resisted all medication, got rapidly well. The right eye was cured in eight days, the left one in twenty. The disease had lasted in all nearly three months.

The history of this case will show, I hope, the importance of making an early bacteriological examination in all cases of ophthalmia neonatorum, and demonstrates also the fact that the diplobacillus described by Morax and Axenfeld as the cause of subacute catarrhal conjunctivitis can produce other forms of infectious ophthalmia.

FORWARD DISLOCATION OF THE HEAD OF THE FIBULA.

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FORWARD dislocation of the head of the fibula is very rarely met with, and for very simple reasons. First, it is firmly bound in its articulation with the tibia by strong ligaments; and, second, there are no very powerful muscles attached to it anteriorly to either in whole or in part pull it from its place.

Hamilton reports four cases observed by Savournin, Jobard, Thompson, and Dr. J. Hawley; while the *American Text-book of Surgery*, edited by Drs. Keen and White, says there have been seven cases of this injury reported.

It seems that while seven cases of forward dislocation have been reported at various times, there have been only five cases reported of backward dislocation of the same joint. This would appear at first thought to be the more common of the two, seeing the biceps femoris

is attached to the head of the fibula, and is a very powerful muscle. To my mind, it is extremely improbable that any case of dislocation of the fibula has ever occurred through muscular action alone in persons in whom the ligaments of the knee were in normal condition. In muscular action played a very important part in its production the backward dislocation would be much the more common of the two, while in reality it is not so often reported.

The treatment laid down by Hamilton for cases of forward dislocation is to flex the leg upon the thigh and the foot upon the leg at the same time pressure is made upon the dislocated bone. He says Thomson and Jobard were unable to effect reduction when the leg was extended.

In a case of my own I found the following :

The cause was direct violence, the limb having been caught between two freight cars in such a way that the tibia was pressed backward and the fibula forward. The symptoms were absence of the bone in its proper place and undue prominence in front of where the articulation should have been. The X-ray showed that the bone had been completely torn from its articulation and pulled forward. It was impossible to effect the slightest change in the position of the bones by traction upon the foot either alone or accompanied by pressure upon the head of the fibula. Reduction was accomplished by flexing the leg upon the thigh, the foot upon the leg, both as completely as possible, by making firm pressure upon the dislocated bone and by having extension gradually made while this pressure was increased. The bone slipped into place with considerable noise as soon as extension of the leg was begun.

A NOTE ON OSTEOPHYTES OF THE NASAL CHAMBERS.

BY ALEXANDER W. MACCOY, M.D.,
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It is some years since the subject of osteophytes in the nasal chambers first began to interest me. During the progress of an operation for deflection of the nasal septum, while I was making explorations in the nasal chambers, I became aware of certain sharp, rough, coral-like deposits situated within the nasal passages. As they proved to be neither necrotic nor rhinolithic aggregations, nor exostoses, I considered them osteophytes. These osseous bodies found in the nasal chambers have the anatomical arrangement and composition of osteophytes. Their formation being arranged in laminated or radiating structures, such anatomical arrangement determines the fitness of the name.

I have neglected to investigate the literature on this subject, having

found only one plate where such bodies in the nasal chambers were illustrated.¹

To enable one to determine definitely the existence and location of osteophytes, a *digital* exploration of the nasal chambers is necessary. Some of us can remember a paper by Dr. Harrison Allen in which he advocated *digital* manipulation of the anterior regions of the middle turbinated bodies for certain diseased conditions present. Following this suggestion, I have for some years used the little finger as an intelligent exploratory probe. In all my operations for deflection of the nasal septum I have found it necessary in such cases to use the finger far back in the nasal cavities to enable me to get a clear idea of the trend of the septum, and also of its thickness. While exploring in this manner I have found in many instances these bony concretions, which I have named osteophytes.

The clinical fact is interesting that bony deposits do in a certain number of cases exist in the nasal chambers without definite anatomical relation to the normal osseous formations.

The presence of osteophytes in the nasal cavities materially interferes with the success of an operation for nasal deflection by preventing free respiration and drainage, and, especially when they are large, by hindering complete manipulation of the septum after the incisions have been completed.

Osteophytes generally are to be found well back in the nasal chambers, situated underneath the lower turbinated bone, on the bony septum, and on the floor of the nose. In size they vary from a sharp spicula to a small, irregular mass. While formulating these notes a young woman came under my care who was a subject of osteophytes in various parts of her body. Large masses were present on the tibia, as well as on the lower end of the radius. The subject consulted me for deflected septum. An operation had been performed upon her some time before for deflected septum, but it had been unsuccessful. Examination showed a marked deflection of the triangular cartilage, associated with deformity of the lateral cartilages. In the triangular cartilage plaques of bony tissue could easily be felt, which were undoubtedly osteophytes. After the second operation there was troublesome bleeding which was not controlled by the tubes, but required tamponing. This incident was, evidently, the result of the interstitial change over the cartilage involving the coats of the bloodvessels and preventing their contraction. The results of the second operation were most satisfactory, and, after a lapse of over a year, the alignment of the septal wall is perfect. In several other cases of osteophytes present in cases of deflection of the nasal septum coming under observation the bony concretions were

¹ Zuckerkandi, "Anatomie der Nasenhöhle," Table I., Fig. 2, Description, p. 187.

well back in the nasal passages, and were broken off and removed during the operation for deflection. I recall one case in a young woman where the osteophytic deposit was near the posterior naris, where it could barely be reached by the tip of the little finger. If this bony mass had not been discovered, complete restoration of the respiratory function of that side would not have resulted, as was the case after the osteophytes had been removed and the deflection corrected.

SUMMARY. 1. Under an anæsthetic, with the little finger as an intelligent probe, certain osseous structures foreign to normal nasal conditions may be found.

2. That these osseous masses are osteophytes, having the anatomic structure of such bodies.

3. That osteophytes, clinically, are *new* conditions to be considered and studied.

4. That in operations for deflection of the nasal septum, lack of complete success may result from their presence.

5. That osteophytes are loose in structure and readily removed by proper instruments.

A REVIEW OF SOME RECENT LITERATURE ON CERTAIN INFECTIVE DISEASES.

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SYPHILIS. The occurrence of fever as a manifestation of syphilis has been known for some time, it having been commented upon especially by Musser and by Osler. More recently, Fletcher,¹ writing on the subject, states that in all cases of fever of obscure origin the possibility of the fever being syphilitic in nature should be borne in mind and that careful examination of the long bones, skin, and viscera should be made, with a view to detecting manifestations of syphilitic infection. He states, also, that the fever of syphilis may occur at different periods during the course of the disease: 1. It may occur in very rare instances three or four weeks before the onset of the secondary skin eruption. This fever is very puzzling and, until the appearance of the eruption, it is likely to be attributed to some other cause. 2. It may precede or be coincident with the appearance of the secondary eruption. This is the so-called "fever of invasion," and it is a very common and important symptom of secondary syphilis. 3. The fever

¹ Syphilitic Fever. New York Medical Journal, 1901, vol. lxxiii. p. 1065.

may occur at any time during the course of the secondary or tertiary stages. The late occurrence of the fever is the most interesting feature. In one of the cases reported it occurred twenty-nine years after the disease had been contracted. It is said that the fever of invasion is rarely absent at the onset of the secondary symptoms. It usually precedes the appearance of the secondary eruption by a week or ten days; rarely by more than two weeks. It is generally believed to be a symptom of the invasion of the system by the organism the cause of syphilis or by its toxic products. The degree of fever varies in different cases; it may be moderate (101° F.), but sometimes it is much higher (104° to 105° F.) in the afternoon. The fever of invasion, as well as the syphilitic fever associated with the late manifestations of the disease, may present any one of the three following clinical types: (1) A mild continuous pyrexia, in which the temperature ranges in the neighborhood of 101° F.; Osler states that this type is not uncommon in the fever that ushers in the constitutional symptoms; (2) a remittent type of fever, with morning drops toward normal and evening exacerbations—the usual fever of invasion; (3) a definite intermittent fever; this is the most remarkable form of all, and it is the type that is most likely to lead to diagnostic errors. It is sometimes accompanied by chills and fever. The febrile diseases for which syphilis is often mistaken are the malarial fevers, typhoid fever, tuberculosis, and sepsis. When general pains and joint pains accompany the fever the diagnosis of rheumatism may be made. In doubtful cases the true nature of the fever may be demonstrated readily by instituting anti-syphilitic treatment.

Carriere,¹ writing of syphilitic fever, states that it is extremely rare (a statement that will not meet concurrence on the part of those who see much of the disorder), and that during the past ten years he has observed but two instances of it. The fever is said to develop usually within forty to sixty days after the initial sore, but sometimes much later. Women especially are likely to have the fever, probably, it is believed, because the initial lesion may go undetected and untreated, and a severe infection thus occur. The febrile affections for which the syphilitic fever may be mistaken are malaria, psittacosis, cerebro-spinal meningitis, miliary fever, pyemia, verrucose endocarditis, miliary tuberculosis, recurrent fever, and influenza. It is only in the most unusual cases that diagnostic difficulties are likely to arise, but the differentiation from typhoid fever may be perplexing. Attention to the following will facilitate the diagnosis: 1. A coated, moist tongue in an individual that presents a typhoid aspect suggests an abnormal typhoid fever or another disease. 2. Extraordinary intensity of the headache suggests syphilis. 3. Very profuse perspiration bathing the body of the patient

¹ La typhose syphilitique. Gazette des Hôpitaux, 1901, vol. lxxiv. p. 65.

is unusual in typhoid fever. 4. Extensive and generalized eruption suggests that the disease, if it be typhoid fever, is a very unusual form. The failure to detect the typhoid bacillus in the stools or in the fluid obtained by puncture of the spleen (to my mind an unjustifiable procedure) suggests the absence of typhoid fever. Examination of the blood is of great value; in syphilitic fever there occur greater oligocythæmia, microcythæmia, and oligochromæmia than in typhoid fever, and hypoleucocytosis occurs in syphilitic fever, and hypoleucocytosis in typhoid fever. 6. The absence of the Gruber-Widal reaction in all stages of the disease is of great value. Finally, one should seek for evidences of syphilis—the initial lesion or a cicatrix (sometimes difficult to find especially in women), and evidences of syphilis in the viscera and the skin, etc. In case it be suspected that a syphilitic patient has acquired typhoid fever, the following are of diagnostic value: (1) The absence of the Gruber-Widal reaction; (2) the absence of the typhoid bacillus in the stools and the fluid obtained by puncture of the spleen; and (3) the therapeutic test—the result of anti-syphilitic treatment. It is believed that the syphilitic fever is an expression of syphilitic infection of an organism of lessened resistance.

Scheib¹ reports the case of a man, aged fifty-one years, who, three months before his death, suffered an injury in the region of his stomach. Soon thereafter he complained of pain, vomiting, loss of appetite, hæmatemesis, and the passing of blood by the bowel. Examination revealed the ordinary signs of bronchitis, and, in addition, pain, tenderness, and rigidity about and below the umbilicus—all of which were relieved by evacuation of the bowels. Subsequently fever, right-sided pleuritis, hæmoptysis, collapse, and death occurred. The necropsy revealed a half-litre of fresh blood between the stomach and the liver, and another half-litre between the stomach and the spleen; a funnel-shaped, perforating ulcer, the size of a dollar, on the anterior surface of the stomach, toward the hilum of the spleen; a dense infiltration with central necrosis, about the size of a dollar, in the region of the pylorus; similar areas of infiltration at eight different regions of the small intestine—some perforated, but all closed by adhesions with the surrounding structures; and many areas of infiltration, grayish-white in color, of firm consistence, without central necrosis, varying in size up to that of a hen's egg, and situated in the lungs. The possible nature of the affection is discussed with especial reference to the likelihood of its being neoplastic or tuberculous, and it is pointed out that it cannot be the one or the other; that tubercle bacilli could not be detected; that microscopically the infiltration consisted of granulation tissue with giant cells; and that the bloodvessels revealed marked endarteritis.

¹ Syphilis mit gumöser Affektion des Magens und Darms so wie der Lungen bei einem 51-jährigen Manne. *Prager medicinische Wochenschrift*, 1900, vol. xxv., Nos. 45, 46.

teritis proliferans that in some places had progressed to complete closure of the vessels. From these facts it is concluded that the case is one of visceral syphilis.

Einhorn¹ divides syphilitic affections of the stomach into three groups—syphilitic ulceration, syphilitic tumor formations (gummas), and syphilitic stenosis of the pylorus. Illustrative cases of each group are cited. Of interest is the fact that two of the cases of obstruction of the pylorus, in consequence of the failure of the ordinary methods of treatment, would have gone on to operation had not the possibility of their syphilitic nature been borne in mind. It is pointed out that syphilis of the stomach is more common than is generally thought; that tertiary syphilis may give rise to severe disturbances of the gastro-intestinal tract; that in all severe disturbances of the gastro-intestinal tract the possibility of syphilis should be considered; and that such disturbances are amenable to antisypilitic treatment. A review of cases reported in the literature is given.

Mracek² states that dactylitis is uncommon in acquired syphilis and in adult life, whereas, as a manifestation of hereditary syphilis in infancy it is quite common. Among 50,000 syphilitic patients he has observed five cases of dactylitis—two in adults and three in children. He reports in detail a case occurring in a man, aged thirty years. Careful investigation, including the use of the X-rays, convinced him that the disease develops primarily in the bone, which becomes rarefied, with the coincident formation of peculiar spaces filled with colloid infiltration and exudation masses; and that, secondarily, the joints and the softer tissues become implicated. The deformities are due principally to inflammatory and oedematous infiltration of the softer tissues. The affection is almost painless, chronic in its course, and tends to heal, with the production of deformities—ankylosis or flail joints. Treatment should consist in administering large doses of potassium iodide (75 grains daily) for a number of weeks. Mercury may be combined with the iodide, but care must be exercised in its use, as in syphilitic diseases of bones its employment is sometimes attended by unfavorable results.

SYPHILIS AND TUBERCULOSIS. Dieulafoy³ reports two cases of concomitant syphilitic and tuberculous disease of the lung. He states that when a subject for a long time syphilitic (in one of his cases twenty years) becomes affected with tuberculosis of the lungs the prognosis is good, and especially is the tuberculosis likely to pursue a favorable course. He warns against the use of potassium iodide and urges the use of mercury. He suggests the alternate use, for periods of fourteen days, of

¹ Ueber Syphilis des Magens. *Archiv für Verdauungskrankheiten*, 1900, vol. vi. p. 150.

² Zur Dactylitis syphilitica. *Wiener klinische Wochenschrift*, 1900, vol. xiv. p. 432.

³ Association de la tuberculose et de l'syphilis. *La tribune médical*, 1901, No. 13.

daily injections of biniodide of mercury (0.004 to 0.008 gramme) and of daily injections of cacodylate of sodium (0.05 gramme).

TUBERCULOSIS. At the final session of the recent British Congress on Tuberculosis the following resolutions were unanimously adopted: 1. That tuberculous sputum is the main agent for the conveyance of the virus of tuberculosis from man to man. Indiscriminate spitting, therefore, should be suppressed. 2. All hospitals and dispensaries should present every out-patient with a leaflet on the prevention of consumption, and insist on the use of a pocket spittoon. 3. Notification of tuberculosis should be established when possible. If compulsory notification is impracticable, voluntary notification should be encouraged. 4. The provision of sanatoria is an indispensable part of measures for the diminution of tuberculosis. 5. Medical officers of health should use all their powers and relax no effort to prevent the spread of tuberculosis by milk and meat. 6. In view of the doubt thrown on the identity of human and bovine tuberculosis, the government should be requested to institute an inquiry into the subject. 7. The educational efforts of the great national societies for the prevention of tuberculosis are deserving of support. 8. A permanent international committee should be appointed to report on the measures for the prevention of tuberculosis in different countries, to publish a popular statement of these measures, and to keep and publish a record of scientific research in relation to the disease. 9. Overcrowding, defective ventilation, the damp and insanitary dwellings of the working classes diminish the chances of curing consumption and are predisposing causes of the disease. 10. The attention of governments and charitable persons should be called to the necessity for establishing anti-tuberculosis dispensaries.

Friedmann¹ publishes the results of an extensive series of investigations concerning the significance of the tonsils as a portal of entry of tuberculosis in young children. The investigations comprise the examination of the tonsils obtained at 91 necropsies, and of tonsils removed by operation from 54 children, with one exception under five years of age. In cases 1 to 3 the tonsillar tuberculosis probably was not primary; in cases 4 to 7, and in case 9 the tuberculosis was most likely primary in the tonsils and secondary in the lymphatic glands, intestine, and bone; in case 8 the tonsils, which contained many tubercles, with giant cells and bacilli, were the only seat of tuberculosis in the body; in cases 10 to 16 (2 with and 5 without tuberculous lesions elsewhere in the body) the tonsils contained typical giant cells, but bacilli could not be demonstrated; in cases 17 to 19 the giant cells in the tonsils were attributed to some process other than tuberculosis; in cases 20 to 30 (cases

¹ Ueber die Bedeutung der Gaumentonsillen von jungen Kindern als Eingangsporte für Tuberculöse Infektion. Ziegler's Beiträge zur path. Anatomie, etc., 1900, vol. xxviii, p. 66.

extensive tuberculosis) the tonsils revealed no tubercles, but the presence of old cicatrices suggested the probability of previous tuberculosis; in cases 31 to 34 tuberculosis of the internal organs was unassociated with tuberculosis of the tonsils; in cases 35 to 37, although the tonsils presented no tuberculous alterations, tubercle bacilli were demonstrated in smear preparations made from the surface of the organs; in the remaining 54 cases tuberculosis was found nowhere in the body. In only one of the 54 cases of operative removal of the tonsils from living children were tuberculous lesions detected that could be looked upon as primary. It is believed that although strong children with chronic hypertrophy of the tonsils rarely have tuberculosis of the tonsils, tuberculosis of the tonsils in children in general is quite common; and that it is not likely that infection occurs by means of the lymph-stream, the blood-stream, or by inhalation, but rather by means of the food. Following the primary infection there occurs a descending tuberculosis of the lymph glands, the result of infection through the lymph-stream.

INFLUENZA. Michel,¹ writing of the pulmonary complications of influenza, gives expression to some opinions that evidently are founded more on speculation than on morbid anatomy. In the pathogenesis of these complications especial significance is attached to atmospheric influences as well as to predisposition, such as infectious and intoxications, more especially alcoholism. Distinction is drawn between primary complications (due to the influenza bacillus) and secondary complications (due to mixed infection). The primary complications comprise the simple catarrh, the acute bronchitis, "fluxion de poitrine," a congestion simulating diaphragmatic pleuritis, and a "hæmoptytic congestion;" the secondary complications comprise splenopneumonia, bronchopneumonia, and pneumonia. To many of us such terms are unusual, at the least. According to Michel, the "fluxion de poitrine" is of sudden onset, and implicates the lungs, the pleura, chest wall, and the nerves. The patient complains of marked pain, aggravated by deep inspiration, coughing, and pressure, and mucous, mucopurulent, and sometimes sanguinolent expectoration. Examination reveals dulness, diminished or rough vesicular breathing, and friction-sounds. Pleural exudation may occur, and the congestion may progress to hepatization of the lung; the process, however, usually is confined to congestion. This "fluxion de poitrine" is not uncommon in French literature, and is said to be a process midway between "congestive bronchitis" and bronchopneumonia. In America, at least, such condition is not recognized. The ordinary "fluxion de poitrine" we should recognize probably as a bronchopneumonia, and should the condition progress to hepatization we should probably interpret the phenomena

¹ Des complications pulmonaires de la Grippe. Le Bulletin Médical, 1901, vol. xv. p. 285.

as indicating the confluence of bronchopneumonic foci. Nor can it be said that a form of congestion of the lungs simulating diaphragmatic pleuritis is recognized in America. Michel recognizes it by the occurrence of pains at the lower portion of the thorax (the insertion of the diaphragm), pain in the neck (from implication of the phrenic nerve), hiccough, sometimes uncontrollable vomiting, and more or less evident inflammation of the lower portions of the lungs and pleuræ. " hæmoptytic congestion " is of interest in that in case of severe hæmorrhage the condition may be mistaken for pulmonary tuberculosis. It is difficult to understand what is meant by " splenopneumonia." Attention is directed to certain peculiarities of the course of influenza pneumonia, especially to the insidious onset and to the difficulty sometimes experienced in distinguishing it from typhoid fever, especially pneumotyphoid. The Gruber-Widal reaction is said to be of value, and to this might have been added the examination of the expectoration for the influenza bacillus. Mention is made of the occurrence of delayed resolution, of abscess of the lung, of gangrene of the lung, and of a condition designated " bronchoplegie á signes pseudocavitaires." This condition, which we should probably designate bronchiectasia, is termed bronchoplegia because it is thought that the stagnation of the bronchial secretion and the excessive suppuration in the dilated bronchi are due to paralysis of the bronchial muscles. The condition is of importance in that it may be mistaken for abscess or tuberculous cavity formation in the lung, both of which conditions have a much more serious outlook than has the " bronchoplegia."

Castellani¹ has examined bacteriologically nine cases of complications and sequels of influenza—pneumonia, bronchopneumonia, pleurisy, empyema, suppuration of the ear, meningitis, arthritis, and enteritis. The investigations comprised bacteriological examination of the expectoration, pleural exudate, joint fluid, cerebro-spinal fluid, lung " juice," and the blood. Influenza bacilli were found only in the expectoration and in the pus from the ears, and not in pure culture, but mixed with pneumococci in excessive numbers. Animals inoculated with these fluids died of pneumococcic sepsis. Examination of the cerebro-spinal fluid led to indefinite results. The remaining secretions and the blood revealed only pneumococci. In addition, Castellani examined the blood in twenty-one cases of typical influenza, and in a number of other cases with complications. In all cases of pneumonia and bronchopneumonia he found only pneumococci. It is believed that the influenza bacillus may be the cause of the complications of influenza, but that, as a rule, this bacillus does not enter the circulation, and that many complications are produced by the pneumococcus.

¹ Contributo allo studio bacteriologico di alcune complicanze dell' Influenza. Riv. clin. med., 1901, No. 3. Fortschritte der Medizin, 1901, vol. xix. p. 781.

In contrast with these opinions, the opinions of Jehle¹ may be cited. This writer details the results of his investigation of a number of cases of scarlet fever, measles, varicella, pertussis, diphtheria, and a number of different diseases in adults, with reference to mixed infection with the influenza bacillus. It was ascertained that influenza is a common secondary infection in childhood, and that it may occur early in the course of the primary disease. The influenza infection usually is in the lower respiratory tract, but it may remain localized to the tonsils, and it may assume an epidemic character. With the occurrence of influenza mixed infection an increased morbidity and mortality develops. In the acute exanthemas there occurs regularly an invasion of the blood-stream with the influenza bacillus, which may enter from the tonsils (this statement is of especial importance and interest as contrasted with the observations of Castellani, previously cited). This bacteræmia may occur very rapidly, and in scarlatina it may be observed even before the eruption. In the non-exanthematic diseases and in adults the influenza bacillus is but rarely found in the blood. If, however, influenza bacteræmia does occur, severe organic lesions—carditis, endopericarditis, pleuritis, brain abscess, etc.—usually occur also. It is believed that the exanthemas in some manner predispose the body to infection with the influenza bacillus and facilitate the entrance of the influenza bacillus into the blood-stream.

Franke² states that he has observed with great regularity in influenza a striated redness of the anterior faucial arch, whereas the uvula, the lateral part and about two-thirds or three-fourths of the velum palati, and sometimes the free edge, retain their normal color. This is said to be especially valuable as a diagnostic sign in cases of chronic influenza. He has observed also swelling of the anterior papillæ of the tongue—a swelling exceeded in degree only in cases of scarlatina and in a few cases of measles. This swelling may occur on the second or third day of the disease, and possesses the same diagnostic significance as does the redness of the faucial arch. Finally, attention is directed to the diagnostic value of enlargement of the spleen, said to occur especially in the chronic forms of the disease.

Federn³ states that in influenza the blood pressure (measured with von Basch's sphygmomanometer) is abnormally low, that this results from disturbance of the cardiac nerves, and that it occurs so constantly that it is of diagnostic value. Inferentially, it is said that most of the symptoms in influenza are due to defective circulation—that they are quasi congestive symptoms. The blood pressure is thought to have

¹ *Zeitschrift für Heilkunde*, 1901, vol. xxii, p. 190.

² Ueber ein typisches Influenzasymptom, die Influenzaangina, und über Influenzazunge und Influenzamilz. *Deutsches Archiv für klinisches Medicin*, 1901, vol. lxx, p. 280.

³ Ueber Influenza. *Wiener medizinische Wochenschrift*, 1901, vol. i, p. 1162.

prognostic value in influenza, in that those patients that have high pressure before the attack (in consequence of increased resistance of the vessel wall) are less resistant and recover from the disease slower than do others.

Petrucchi¹ reports a number of cases of influenza during the course of which there occurred hemorrhages of different sorts—epistaxis, hæmoptysis (of all grades from slight tinging of the expectoration to profuse hæmoptysis), metrorrhagia, hæmaturia, etc. He believes that one is justified in speaking of a hemorrhagic form of influenza in addition to the catarrhal, the nervous, and the gastro-intestinal forms, and he finds justification for this not only in the occurrence of the hemorrhages, but also in the fact that we already speak of hemorrhagic typhoid fever, hemorrhagic scarlatina, etc., and in the fact that the development of a hemorrhage in influenza is indicative of a malignant type of the disease and influences materially the prognosis. In these cases convalescence is much prolonged if the hemorrhage does not lead directly to the death of the patient, as occurred in one of the cases reported.

Huber² states that recently following an epidemic of influenza in which all of the seven members of a family were affected, four suffered with suppurating buboes and lymphangitis. Of these, three (not the father) previously had been vaccinated, and the buboes were confined to the glands of the axilla. It is presumed that these glands were the locus minoris resistentiæ from the vaccination. The father's bubo was in the inguinal region. The buboes contained only streptococci—influenza bacilli or plague bacilli. Recovery ensued in all cases. It is presumed that, aside from rendering the glands less resistant, the vaccination had nothing to do with the infection—a hæmatogenous general infection with the streptococcus. This opinion is borne out by the situation of the bubo in the father. The case is reported as an instance of heterochronous polyinfection—that is, mixed infection with the influenza bacillus and the streptococcus.

Ford³ recently has written of acute aortitis occurring in the course of influenza, of which condition he has observed eighteen cases. In its manifestations it is said to resemble the condition as it occurs in other acute infectious diseases, notably typhoid fever. It affects especially patients past thirty years—those, therefore, whose arteries perhaps already show some of the consequences of wear and tear. It involves especially the arteries of the legs; usually those of one leg, but sometimes those of both. It is most likely to develop during convalescence from influenza, but it has been noticed to occur some time after apparent recovery. The onset usually is sudden and manifested by pain (th

¹ Ein Beitrag zur Casuistik der Hemorrhagien bei Influenza. Wiener medicinische Presse, 1900, vol. xli. p. 1770.

² Ueber eine Familiäre Streptococcenerkrankung nach Influenza. Correspondenzblatt Schweizer Aerzte, 1901, vol. xxx. p. 233.

³ Thèse de Paris, 1901.

may be so severe as to require morphine), tenderness along the course of the inflamed artery, paræsthesia (that may even precede the pain), lowering of the temperature of the affected part, and mottling of the skin. Arterial obliteration is indicated by cessation of pulsation below the site of obstruction. This may be temporary and followed by restoration of the circulation without damage to the part; or it may be permanent and followed by gangrene. The gangrene usually is of the dry variety, but it may be moist if there be associated phlebitis. In the event of gangrene there may occur sphacelation of the gangrenous area and recovery, or there may occur general infection and death. Of fifteen patients, six died. As regards treatment, it is recommended to encase the affected limb in wadding and splints, and to apply methyl salicylate or an ointment of salicylic acid, turpentine, and belladonna. The pain, if severe, should be relieved by morphine, and in the event of certain complications surgical intervention is called for.

Diemer,¹ writing of influenza polyneuritis, states that usually it develops during convalescence (as does diphtheritic neuritis), within from ten days to one month from the beginning of the infection. The initial manifestations may be either sensory or motor; the latter occur constantly without any special predilection for particular muscles. While all the muscles may become affected, the muscles of the upper or the lower extremity of one side or of the trunk, or of the neck, are most frequently affected. Frequently, also, the muscles of the face and the muscles supplied by the vagus are affected. Less frequently the oculomotor, the abducens, the optic, and the auditory nerves are affected, and exceptionally the nerves of the palate, the pharynx, and the diaphragm. Paralysis that involve the extremities diminish in intensity as the trunk is approached. Reactions of degeneration are constant. Ataxia is common, and the gait may resemble that of a tabetic; flaccid paralysis is constant. Disturbances of sensation are more or less pronounced; they are usually less intense than are those that occur in other polyneuritides, notably the alcoholic. Trophic and vasomotor disturbances are not conspicuous. The skin and tendon reflexes are diminished or absent. The course of the affection may be acute or subacute. General and localized forms may be distinguished. Complete recovery usually occurs; in some cases, however, more or less atrophy of certain groups of muscles persists. The treatment, as a rule, should be symptomatic; but especial attention should be directed to hygienic and dietetic measures, that recurrences may not occur.

PNEUMONIA. Schultz² directs attention to the fact that during recent years increasing support has been given the view that croupous pneu-

¹ La polynévrite grippale. *Gaz. hebdom. de méd. et de chir.*, 1901, vol. xlviii. p. 37.

² Contribution à l'étude de la pneumonie fibrineuse. *Archives des sciences biologiques de St. Petersburg*, 1901, vol. viii. p. 1.

monia is due to a primary pneumococcic infection of the blood and secondary infection of a lung that has become reduced in resistance rather than to a direct infection of the lung through the respiratory passages. In an attempt to confirm this view through experimental investigation he injected pneumococci into the jugular veins of low animals, and after their death he examined their lungs microscopically even when they presented no naked-eye deviations from the normal. He states that the lesions of pneumonia were evident in all the lungs and that these lesions corresponded to those of red hepatization in the human subject. The bloodvessels of an entire lobe or of some of the lobules were dilated and contained many leucocytes (mostly mononuclear) and many pneumococci. The walls of the alveoli were infiltrated with young cells, and the alveolar epithelium had desquamated. In the lumen of the alveoli considerable quantities of fibrin formed early; in the later stages this was mixed with erythrocytes, so that in some instances the lumen of the alveoli was completely filled. It is concluded that pneumonia may be induced in rabbits merely by injecting pneumococci into the blood-stream—that is, without previous alterations in the lung. Regarding the question how infection of the blood occurs in man—that is, infection of the blood previously to infection of the lung—an opinion is withheld.

TYPHOID FEVER. Recently a number of studies of what may be termed the nervous manifestations of typhoid fever have been published. Thus Debove,¹ directing attention to the fact that most of the symptoms of typhoid fever are nervous in nature, and are to be attributed to the action of the typhoid toxin on the nervous system, reports the case of a man, aged twenty years, who, in September, 1900, passed through an attack of typhoid fever with intestinal hemorrhages. At the end of about two months the patient was considered well enough to be discharged from the hospital, but that same day he became ill with lumbar pain, fever, and delirium, said to have resulted from mental excitement. The lumbar pain increased in severity until about the middle of December, when after a short walk it became so severe that the patient was unable to move his lower extremities. With rest in bed the pain abated somewhat, but it recurred with severity as soon as any attempt at movement was made. Examination revealed motor, sensory, trophic, and vasomotor disturbances. The lumbar pain was much increased by pressure over the sacrum. Spontaneous pain or disturbances of sensation were not present in the lower extremities, but on attempting to walk there occurred severe pain extending from the lumbar region through the legs. The legs were distinctly paralyzed and atrophied. The temperature on the first day ranged between 38° C. and 38.5° C.

¹ Sur un cas de paraplégie post-typhique. *Le Bulletin Médical*, 1901, vol. xv. p. 141.

on the seventh day it was 40.4° C., and on the twelfth day it reached normal. At this time the lumbar pains and the other manifestations gradually subsided. At the time of the report, when at rest the patient had no complaints; when he attempted to walk, however, slight pains in the lumbar region occurred. He was able, nevertheless, to ascend stairs without especial difficulty. Sensation was normal, but in the lower extremities there was distinct atrophy. The case is considered an instance of post-typhoid paralysis due to circumscribed inflammation of the lower portion of the spinal meninges.

Foulerton and Thomson¹ state that the nervous symptoms of typhoid fever may be due either to the direct action of the specific bacillus or to the action of secondary invaders. It is difficult to determine clinically the range of activity of each of these, though it is stated that in the beginning of the disease the typhoid bacillus alone is probably the active agent; later, other bacteria may play a rôle, as we know they are active in producing the intestinal lesions. The symptoms referable to the typhoid bacillus may be produced by either the bacillus acting directly or by its toxin. The toxæmia may give rise to manifestations of severe irritation of the brain, which sometimes can be distinguished from the meningitis due to the typhoid bacillus or other organisms only by the onset of paralysis or optic neuritis. Two cases illustrating the two forms of cerebral disturbance are reported. The first case was that of a girl, aged nine years, who suffered from severe toxæmia. She was noisy and restless, passed into a low, muttering delirium, and died. No alterations were found in the brain, and cultures from the brain gave negative results. The second case was that of a boy, aged twelve years, who was thought to be suffering from tuberculous meningitis until the Gruber-Widal reaction was obtained. The necropsy revealed marked congestion of the meninges, and bacteriological investigation revealed the typhoid bacillus. This is therefore a case of true typhoid meningitis, of which it is said that eighteen cases have been reported. The writers undertook the study of the action of the typhoid toxin on the ganglion cells of the central nervous system by injecting living cultures of the typhoid bacillus and the typhoid toxin into rabbits, and studying the brain lesion. Aside from occasional slight swelling and indistinctness of the details of the cells, no noteworthy alterations were detected; from which it is concluded that typhoid infection or typhoid toxæmia does not produce noteworthy discoverable alterations in the ganglion cells of the nervous system.

A year or more ago, at the German Hospital, I performed a necropsy on a subject dead of fibrinopurulent meningitis complicating typhoid fever. From the exudate the typhoid bacillus was obtained in large

¹ The Causation of Nervous Symptoms in Typhoid Fever. *Lancet*, 1900, vol. i. p. 1121.

numbers and in pure culture. The case will be reported shortly by the assistant pathologist to the hospital, Dr. Edward Kemp Moore.

Salomon¹ points out that he has observed that in a number of cases of typhoid fever the optic disk presents an appearance suggesting cerebral tumor—not a distinct papillitis, but a veiling of the disk, with indistinctness of its edges, injection of the capillaries, dilatation and tortuosity of the veins. Performing lumbar puncture, he found the spinal fluid under high pressure—from 180 to 250 mm. of water. The fluid was sterile, contained about the normal amount of albumin and of leucocytes, and did not cause agglutination of the typhoid bacillus, although in each of the three cases investigation of the blood-serum did cause such agglutination—in one case even when diluted 1 to 500. It is believed that an acute serous meningitis is very common in typhoid fever, and that to this is due one of the characteristic features of the disease—the slow pulse. Spinal puncture was followed by subsidence of the headache.

Stadelman² points out that in his previous communication on lumbar puncture he referred to the symptoms of brain pressure not only in typhoid fever, but also in pneumonia, scarlatina, etc., and he attributed them to an angioneurotic hydrocephalus the result of the action of the typhoid toxin. He has not observed the good effects of lumbar puncture reported by Salomon.

Kühn³ reviews briefly the previously reported cases of typhoid spondylitis, and reports a personal observation of his own. The case was an instance of severe typhoid infection in which, thirty days after the temperature had become normal, vague pains developed in the lumbar region. Gradually a circumscribed swelling and marked tenderness on pressure developed in the region of the lumbar vertebræ, and these were associated with excessive pain on motion of the trunk. In the course of about a week a distinct lumbar kyphosis developed, but symptoms of compression of the spinal cord did not appear. Gradually the pain and tenderness lessened and the kyphosis disappeared, so that at the end of about three months the patient could be discharged from the hospital. It is stated that, according to Quincke, who first described typhoid spondylitis, its principal characteristics are tenderness of certain spinous processes and manifest swelling over the affected part. To these Kühn adds the kyphosis, which, however, has been observed in but few cases, and is to be attributed probably to destructive processes in the body of the vertebræ. Treatment should consist in absolute rest in bed and the local application of cold. In the event of serious symptoms antiphlogistic remedies may be called for—even in some cases orthopedic appliances.

¹ Berliner klin. Wochenschrift, 1900, No. 6.

² Ibid., 1900, No. 8.

³ Ueber Spondylitis typhosa. Münchener medicinische Wochenschrift, 1901, vol. xlviii. p. 926.

Mannini¹ directs attention to what he believes to be the rather common occurrence of peritonitis in typhoid fever in the absence of perforation of the intestine. He states that in typhoid fever the intestinal epithelium has lost its protecting power; that the lymph vessels of the intestine are inflamed, and that this inflammation extends not only to the serous covering of the intestine, but even to the mesenteric lymph glands; that the typhoid bacillus is present in the spleen, the liver, and the kidney; and that as it finds its way to these organs so also does it find its way to the peritoneum. It is believed that the reason that peritonitis does not occur more frequently in consequence of such infection of the peritoneum by the typhoid bacillus is because the peritoneum possesses marked absorptive powers and is able to destroy bacteria such as the staphylococci, the bacterium coli commune, and the typhoid bacillus. This bactericidal property is associated with the presence of leucocytes and the peritoneal serum. It is believed that there occurs in the course of typhoid fever and during convalescence an acute peritonitis that is not a perforative peritonitis; that this is due to propagation and diffusion through the intestinal wall of the specific infective agent; that such propagation and diffusion occur in every case of typhoid fever, but that peritonitis results only when the resorptive and bactericidal properties of the peritoneum have been overcome. Such peritonitis is said to give rise to symptoms similar to those of perforating peritonitis, from which it is to be distinguished by the absence of gas in the peritoneum. It gives a better prognosis, and its treatment is medicinal.

Osler² has drawn up the following schedule of specific directions to be followed in cases of typhoid fever in which perforation is suspected; though intended especially for use in hospital practice, they are of general adaptability and of great value: 1. Instructions should be specific and definite to the night superintendent and head nurses to notify the house physician of any complaint of abdominal pain by the patient, of hiccough or vomiting, of a special rise of pulse or respiration, of sweating or of signs of collapse. 2. House physicians should note the character of the pain, as to (a) onset—whether only an aggravation of slight abdominal pain, such as is common both with constipation and with diarrhoea, or whether it was sudden, intense pain which caused the patient to call out, and which, though relieved by stupefies and ordinary measures, soon recurred in paroxysms and grew worse; and (b) the locality—whether diffuse or localized in the hypogastric or right iliac regions; radiation, as to penis. It is to be borne in mind that abdominal pain of a severe character may be associated

¹ Sulla peritonite da propagazione dall'intestino nel corso della febbre tifoidea. *Riforma med.*, 1900, p. 210. *Centralblatt für innere Medizin*, 1901, vol. xxii, p. 101.

² On Perforation and Perforative Peritonitis in Typhoid Fever. *Philadelphia Medical Journal*, 1901, vol. vii, p. 116.

with an acute pleurisy, with distended bladder, with cholecystitis, and with a packed rectum, or may follow an enema. 3. State of the abdomen—the conditions to be noted in writing at once as to the following particulars: (a) whether flat, scaphoid, or distended, and, if distended, whether the distention is uniform or chiefly hypogastric; (b) respiratory movements, whether present, if uniform, and seen below and above the navel; (c) palpation, as to tension and peristalsis, locality and extent, and degree of pressure necessary to elicit; muscular rigidity and spasm, whether present or not, and in which special localities; and noting particularly its absence or presence in the hypogastric region and the right iliac fossa; (d) percussion; character of notes at front and in flanks; liver flatness, extent in middle, nipple, and mid-axillary lines; note specially every third hour; remember, too, that obliteration may occur in a flat as well as in a distended abdomen; (e) auscultatory percussion may be helpful; (f) auscultation; obliteration of signs of peristalsis; presence of friction; (g) examination of the rectum; whether tenderness or fulness between the rectum and bladder; (h) stools; character, frequency, presence of blood or sloughs. General condition of the patient: (a) facies, whether change in expression; risus, slight or marked; pallor; sweating, etc.; (b) pulse, change in rhythm, rate, and force; (c) temperature, whether a drop or no drop, whether after a tub or not; (d) respiration, sudden increase not infrequently; whether shallow or sighing; (e) sweating, if subject to, during attack; if onset with pain; whether local or diffuse; (f) vomiting, whether with onset of pain or not; character of vomiting; (g) hiccough. 5. Blood-count, leucocytosis, whether stationary or rising, may be marked and early. In a majority of cases well followed there is a rise. The constant leucopœnia in typhoid fever has to be taken into account. Also a count of the red blood-corpuscles and hæmoglobin, as a decided drop might indicate hemorrhage.

Considerable attention has been directed recently to the occurrence of typhoid bacilli in the urine of typhoid fever patients, to the occurrence of typhoid cystitis, and to the consequent necessity to disinfect the urine of all typhoid fever patients—Gwyn,¹ Curschmann,² Neufeld,³ Schumburg,⁴ Smith,⁵ etc. With a view to prevent the occurrence of typhoid cystitis, to rid the urine of typhoid bacilli, and to limit the spread of typhoid fever, the internal administration of urotropin (7

¹ The Disinfection of Infected Typhoid Urines. Philadelphia Medical Journal, 1901, vol. 1, p. 80.

² Ueber Cystitis typhosa. Münchener medicinische Wochenschrift, 1900, No. 42.

³ Deutsche medicinische Wochenschrift, December 20, 1900.

⁴ Zur Disinfection des Harns bei Typhusbakteriurie durch Urotropin. Deutsche medicinische Wochenschrift, 1901, No. 9.

⁵ Ueber die echte typhöse Cystitis und ihre Behandlung. Monatsbericht für Urologie, 1901, vol. vi., Part 5.

15 grains three times daily) has been much recommended. In general this is a very valuable remedy, and it is usually well borne. It is well, however, to remember that several cases of hæmaturia following its use have been reported—Brown,¹ Griffith,² Gordon,³ etc.

A number of cases of hemorrhagic typhoid fever have been reported recently by Stahl,⁴ Musser and Sailer,⁵ Hamburger,⁶ Nicholls and Learmonth,⁷ Dickinson,⁸ Eshner and Weisenberg,⁹ etc. Hamburger gives a good review of the literature of the subject. An unusual case of hæmoglobinuria complicating typhoid fever and terminating in recovery of the patient is reported by Musser and Kelly.¹⁰

DIPHTHERIA. McCollom¹¹ urges the use of large doses of antitoxin in the treatment of diphtheria, stating that, as we cannot estimate the amount of toxin generated by the diphtheria bacilli in the false membrane, we should administer the antitoxin in dose sufficient to produce the effect desired—that is, shrivelling of the false membrane, lessening of the amount and fetor of the nasal discharge, and improvement in the general condition of the patient. He says that when one sees a patient with membrane covering the tonsils and uvula, profuse sanious discharge from the nose, spots of ecchymosis on the body and extremities, cold, clammy hands and feet, a feeble pulse, and the nauseous odor of diphtheria, and finds that after the administration of 10,000 units of antitoxin in two doses the condition of the patient improves slightly; that after 10,000 units more have been given there is marked abatement in the severity of the symptoms; that when an additional 10,000 units have been given the patient is apparently out of danger, and eventually recovers—one must believe in the curative power of antitoxin. When one sees patients in whom the intubation tube has been repeatedly clogged, when the hopeless condition of the patient changes for the better after the administration of 50,000 units, one cannot help but be convinced of the importance of giving large doses of antitoxin in the very severe and apparently hopeless cases. In the majority of instances these large doses are not required, particularly if the patients are seen

¹ Hæmaturia following the Administration of Urotropin. *British Medical Journal*, 1901, vol. i. p. 1472.

² *Ibid.*, p. 1617.

³ Hæmaturia following the Use of Urotropin. *American Medicine*, 1901, vol. ii. p. 848.

⁴ Gangrenous Dermatitis Complicating Typhoid Fever. *Transactions of the College of Physicians of Philadelphia*, 1900, vol. xxi. p. 33.

⁵ *International Medical Magazine*, 1899, vol. viii. p. 827.

⁶ *Johns Hopkins Hospital Reports*, 1900, vol. viii. p. 309.

⁷ The Hemorrhagic Diathesis in Typhoid Fever, etc. *Lancet*, 1901, vol. i. p. 305.

⁸ A Case of Hemorrhagic Typhoid Fever. *Lancet*, 1901, vol. ii. p. 24.

⁹ Hemorrhagic Typhoid Fever. *AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, 1901, vol. cxi. p. 281.

¹⁰ Hæmoglobinuria Complicating Typhoid Fever. *Philadelphia Medical Journal*, 1901, vol. vii. p. 119.

¹¹ A Plea for Large Doses of Antitoxin in the Treatment of Diphtheria. *Boston Medical and Surgical Journal*, 1900, vol. cxliii. p. 627.

early in the attack, 4000 to 6000 units being enough to produce characteristic effect on the membrane. Dr. Osler¹ suggests that those in charge of institutions who still have any lingering doubts (concerning the efficacy of diphtheria antitoxin) should, in the interests of the little patients, and in a spirit of humility, visit the South Department of the Boston City Hospital, and learn a few salutary lessons from its director, Dr. McCollom.

REPORT OF A CASE OF FIBRINOUS BRONCHITIS, WITH REVIEW OF ALL CASES IN THE LITERATURE.²

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CASES of fibrinous bronchitis, or plastic bronchitis, or bronchopseudomembranosa, or bronchial croup, are as interesting as they are rare. The following is the history of a case observed in the Maternity Ward of the Johns Hopkins Hospital in September last.

Maggie S., colored, aged twenty-two years; II.-para. Rathe emaciated looking; very dull mentally.

Family History. No tuberculosis; father suffers from rheumatism; otherwise negative.

Past History. Labors normal. Menstrual history normal. Two years ago, in the fall, she had an attack similar to the attack observed in the hospital, the symptoms being cough, pain, respiratory distress, profuse expectoration, containing, evidently from patient's description, branching casts. Following this attack she had "pneumonia," being confined to bed one month. Every year in the fall since that time she has had similar attacks. These attacks had no relationship to her previous pregnancies, which occurred two and three years previous to her entrance to hospital, respectively. (See Note I.) She has lost in weight throughout the last two years. She entered the hospital in the ninth lunar month of her pregnancy, on July 27th. At the time of her entrance she had a temperature of 101.5°, which, in two or three days, had dropped to normal. From this time until the time of her delivery there is no note on her history of any abnormal condition, and for the last twelve days of this period she was under personal observation; nothing abnormal was noted. She was delivered on September 12. She came to the delivery-room with a normal temperature, no cough, no respiratory distress, or expectoration.

Present Illness. Her puerperium was normal except for a slight cough and a slight evening temperature—100° to 101°. She had no signs of a light bronchitis, lengthened expiration, and crepitant rales at the bases in the back.

On the tenth day she was allowed up in her chair, but the discomfort

¹ The Principles and Practice of Medicine, 1901, fourth edition, p. 155.

² Parts of this paper were read at the Johns Hopkins Medical Society on June 2, 1901, and the specimens demonstrated.

from her cough increased so much that she was put to bed again, her evening temperature having touched 102°.

On the thirteenth day of her puerperium I was in the ward delivering another case, and my attention was called by the nurse to the patient's extreme dyspnoea.

Physical Examination (during first attack, observed on September 25th). Patient is sitting up in bed in intense dyspnoea. All the accessory muscles of respiration are called into play. The alæ nasæ dilate with each respiratory movement. She coughs incessantly, and brings up large masses of expectoration. She is complaining of an intense sharp pain radiating from the lower left side through the back, increased on coughing. Respirations are 35 per minute; pulse, 100; temperature, 102°. Loose tracheal râles are heard in the throat. On palpation there is normal vocal fremitus throughout. On percussion there is high tympany in both lower axillary regions, higher and approaching dull on the right. On auscultation over the fronts respiration is prolonged and accompanied by fine moist râles. The breathing is more intense in the right lower axilla, and approaches tubular breathing in its quality. The inspirations are here accompanied by râles distinctly crepitant in character. There is impaired resonance in the right lower back and harsher and louder breath sounds, and in one region covered by the bell of the stethoscope the breathing is distinctly tubular and accompanied by crepitant râles. The attack of intense dyspnoea lasted about one hour. The expectoration contained the large casts, from which I have prepared sections, and some smaller incomplete casts. Three days after the first attack Dr. Fletcher made a careful physical examination, with the following result:

Right Lung. Percussion note and breath sounds are particularly clear throughout the entire front. The note is slightly impaired over the lower subscapular and axillary region. Over the lower interscapular and throughout the subscapular region the breath sounds are enfeebled, and there are a few moist râles on expiration. The voice sounds are slightly impaired.

Left Lung. On percussion the note is clear throughout the upper front and distinctly impaired throughout the lower half of the interscapular region. On auscultation a definite pleuritic friction rub is audible over the second, third, and fourth interspaces in the parasternal line, and is audible out to the anterior maxillary line. The breath sounds are not tubular. In the third interspace there is a cardiac respiratory rhythm to the friction rub. Between the mid-axillary and anterior scapular lines, and extending from the apex of the axilla to the costal margin, there is a definite area of impaired resonance, over which the breath sounds are almost inaudible and the voice sounds enfeebled. In the lower intrascapular region there are fairly numerous medium moist râles, some of which approach a crepitant quality.

Heart. Point of maximum impulse is best seen and felt in the fifth interspace 10 cm. from the median line. There is normal cardiac dullness. Both sounds are audible at the apex. The first is accompanied by a very soft systolic bruit. In the third interspace a pleuropericardial friction rub is audible as noted above.

Leucocytes at this date were 5000, and the differential count was normal.

Further course of present illness: She had another paroxysmal

attack, not so severe in character, on October 6th (eleven days later), in which she coughed up the large cast which is represented in the drawing, and several smaller less complete casts. (See Fig. 1.) The sputum expectorated during this attack on October 6th showed the following characteristics: Amount, 100 c.c.; separates into three layers on standing: (1) a granular detritus; (2) milky fluid; (3) branching cast.

First Layer. Cells; alveolar epithelium, leucocytes which were not eosinophiles.

FIG. 1.



Second Layer. Bacteria, streptococci and diplococci; no tubercle bacilli, no Charcot-Leyden crystals, or Curschmann's spirals.

Third Layer. Branching casts which contained cells, the nature of which was not determined. No tubercle bacilli, no Charcot-Leyden crystals.

In the intervals between the attacks there was some slight respiratory distress and an evening temperature ranging between 100° and 101° . Only on a few occasions did the temperature reach 101° , so that a uterine culture was not taken, but puerperal infection could be definitely excluded by the absence of local signs. She had a slight cough, and occasionally expectorated small casts, which caused her very little dis-

comfort. On one occasion a to-and-fro vibrating sound was heard on expiration in the right axilla. This corresponds in character to the "ventilgeräusch" of the Germans. The urine was negative throughout. The patient continued to have a slight cough and dyspnoea and slight evening temperature, and left the hospital in spite of our protests, October 10th. She remained in Baltimore for three weeks after her discharge from the hospital, and had frequent attacks of violent coughing and expectorated "tree-like things" (description of a friend). She then went to her home in Virginia, where she died two weeks later. No data as to the cause of death are obtainable.

FIG. 2.



Cast expectorated; three-fourths natural size. Drawn from fresh specimen.

Casts—Macroscopical Structure. The casts are of various sizes, the largest being 10 cm. long and showing branchings down to the seventh or eighth degree. (See Fig. 2.) They number about twelve. Their color is white, their consistency that of fibrin. Many show little intumescencia over the surface of the larger branches due to little bubbles of contained air. The morphology of the casts is best seen by looking at the illustration showing a portion of the cast embedded, from which the sections have been cut. (See Fig. 2.) It shows an outer stratified layer or skin, which encloses, in what would otherwise be a very large

lumen, various separate whirls and cylinders. One immediately gets the impression from looking at this cross section that the exudation starts in the finer bronchi, and is then gradually pushed up into the larger bronchi, where additional exudation is apposed. That is, supposing the largest branch of a large cast to represent a cast of the bronchus of the third degree, the substance forming that cast represents not only exudation formed in that bronchus itself, but also that formed in all the bronchi below the level of this bronchus of the third degree. Various portions of the cast were hardened in Müller's fluid or by the formalin alcohol method, and were stained by the following methods:

1. Weigert's fibrin stain (celloidin sections).
2. Hæmatoxylin and eosin (celloidin sections).
3. Eosin and methylene blue (paraffin sections) (see Wright and Mallory).
4. Ziel-Nielson's method for tubercle bacilli (paraffin sections).
5. Unna's method for mucin (paraffin sections).

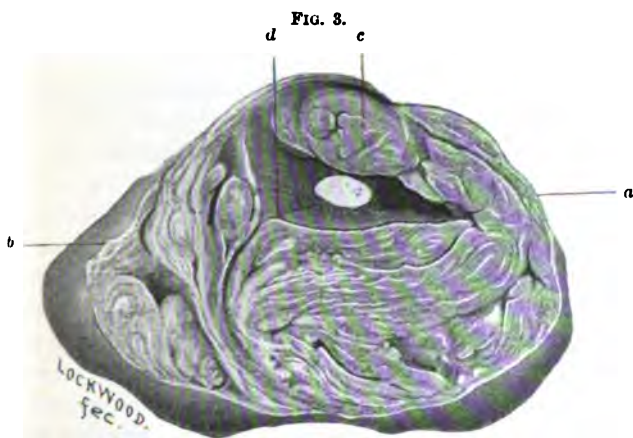
Results. 1. A surprisingly small portion of the sections take Weigert's fibrin stain. The greater number of fibrin fibrils are distributed in the outer layer or "skin" of the cast, or in the outer layers of the smaller whirls. The stain brings out organisms, streptococci and staphylococci, generally adhering to the outer side of the cast, though a very few are occasionally found in the mucus in the inside of the cast.

2. *Hæmatoxylin and Eosin.* This stain shows a ground substance consisting of two apparently distinct materials, a fibrillar substance taking a deep eosin stain, which is concentrically arranged about the lumina of the separate whirls. This encloses a second substance which is transparent and takes a light tinge of hæmatoxylin. Undoubtedly this latter substance, from its optical properties and from its staining reaction, is mucin. The substance taking the eosin stain so deeply is not altogether fibrin, but in part shows itself to be such, for it contains the fibrillæ which take the fibrin stain. What the nature of the portions that do not take the fibrin stain is it is difficult to say from the data at hand. Cells are very numerous, and are for the most part mononuclear leucocytes, though some polynuclears are seen. They are more numerous in the mucin ground substance than in the eosinophilic material. There are a few alveolar cells, many of which contain blood pigment; a very occasional red blood-cell is seen. Beside these cellular elements there are certain bodies which we supposed at first were cellular degenerations, but which in other stains took on a more specific character, and are, perhaps, some peculiar form of organism. These will be spoken of under 4. There were also a small number of cells showing eosinophilic granules. None of these are polymorphonuclear leucocytes, but are either degenerating epithelial cells or large mononuclear leucocytes.

3. *Sections stained in eosin and methylene blue.* These show the ground substance to be reticulated and taking altogether a deep eosin stain. The methylene blue stains only the nuclei of the cells. There are no mastzellen. This stain brings out well the few eosinophilic cells.

4. *The tubercle stain* showed no tubercle bacilli, but brought out those peculiar bodies which are mentioned above. They are irregularly round, from 7 to 15 μ in diameter. (See Fig. 3.) They retain the fuchsin, keeping a deep red color. They have apparently a double

contoured outer shell, from which in certain places the deeply staining protoplasm seems to have shrunk away. Vacuoles of various sizes are seen in their protoplasm. The protoplasm stains diffusely, but shows a few more deeply staining granules. The shell seems to have a tendency to roll up on itself. These bodies were first seen in the sections stained for tubercle just four days before the reading of this paper at the Johns Hopkins Medical Society on June 2d, so that it is too early to give accurate descriptions of them or to judge as to their nature. The specificity of their staining and morphology suggests that they are some form of organism. After finding them here we looked for them in Weigert sections, and found that they retained Weigert's stain, showing the same characteristics with this stain as with the tubercle stain. They also take a specific stain in the eosin methylene blue method. In this they show well the double contoured, highly refractive outer shell, but



Drawn under a magnification of 16, showing the portion of the cast at the level of its primary division into three branchings. From the specimen embedded in celloidin, thus giving an idea of the cross section and the third dimension.

a and b. Outer skin. c. Separate whirl representing exudate from a smaller branch. d. Largest branching.

none of the details of the protoplasm are brought out. In the hæmatoxylin and eosin stain these bodies look like degenerated nuclei, retaining the hæmatoxylin.

5. Unna's method with polychromethylene blue for mucin shows a substance taking a blue stain and a more opaque substance taking a beautiful red.

Cultures were made from the inside of the main branch of the largest cast after searing its surface, and streptococcus pyogenes and staphylococcus aureus obtained in pure culture. No diphtheria bacilli or pneumococci were cultivated.

Collections of all the cases in the literature have been made by Valleix (1843), Peacock (1854), Lebert (1869), Biermer (1867), Riegel (1875), West (1889). Lebert's article is very complete. It is entitled

"Ueber das Vorkommen fibrinöser entzündungsproducte in Bronchien und Lungen-alveolen—Ueber fibrinöse oder Pseudomembranöse Bronchitis und Pneumonie, Bronchitis Fibrinosa, Bronchitis Pseudomembranacea, Pneumonia fibrinosa."

He excludes from consideration all cases secondary to diphtheritic inflammation of the pharynx and larynx. These are cases caused, as we now know, by the Klebs-Löffler bacillus. They are especially discussed by Peter, Millard, and Trousseau. Lepine, who reviews this whole subject, gives only one authentic case, which he cites from Trousseau. One is led to expect that actual cases are rare, for the subject receives always at the hands of French writers general consideration. The most often seen following tracheotomy. Lebert comes back to the term "fibrinous bronchitis" as opposed to Biermer's "pseudomembranous bronchitis." His group of cases of imperfect observation thirty-two in all—are interesting mainly from an historical standpoint. Galen evidently observed a case and considered the expectorated material as casts of the vessels of the lung. Donatus (1586) describes the casts as "*verruca frustum parvum per tussum excreatum e pulmonone.*" Nicol Tulpius as "*surculi venæ arteriosæ expectorati.*" Bussiere (1750) made an autopsy on a case in all probability associated with tuberculosis, and found a cast *in situ*, thus proving that they were not vessels of the lung, but bronchial secretion. In the early part of the eighteenth century the casts were commonly called bronchial polypi: thus Sanctorius (1727) and Ruyschius (1721). The most interesting case in this group is and unique in the whole history of the disease is that of Hayn (1818) who describes finding at autopsy in an infant, aged three weeks, extensive casts throughout the bronchi of both lungs. Lebert's second group of cases—five in all—are not cases of fibrinous bronchitis. They are fatal cases of bronchopneumonia in which at autopsy bits of membrane are found in the bronchi, never forming any definite branching casts, but being scattered here and there throughout the pneumonic area. Lebert's third group are cases of acute fibrinous bronchitis including seventeen cases. He gives a careful analysis of these cases, four of which were fatal, and complete histories of seven cases, including four fatal ones. Three of these four were fatal in an attack, and died in typhoidal collapse. The cases of Lebert's fourth group, associated with pneumonia, are, indeed, remarkable, and very suggestive. He calls attention to the fact well established through the researches of Lobstein, Remak, and Rokitansky that in lobar pneumonia 1 per cent. according to Remak), and are frequently found at autopsy in the smaller bronchi. He then reports five cases out of the literature in which definite branching casts of the bronchi were found in pneumonia. Four of these five cases were fatal, and in three out of these four

were never expectorated but were found at autopsy. All were fatal early in the disease. His fifth group of cases, including twenty-seven, are cases of idiopathic chronic fibrinous bronchitis. He gives a careful analysis of these, which it will be unnecessary to repeat, as the analysis of the cases given below covers the ground. There are seven cases in his sixth group of chronic symptomatic fibrinous bronchitis. Four of these are associated with the last stages of pulmonary tuberculosis; one with fatal abscess of the lung and mitral disease; one with syphilitic ulceration and stricture of the larynx, and one with empyema, this last case being the only one that was not followed to a fatal termination and autopsied.

I have tabulated all the cases that have been reported since the publication of Lebert's paper in 1869 in French, German, English, and American journals and monographs. Allow me to present an analysis of these cases. The cases reviewed fall naturally into the nine groups given below:

I. Chronic bronchitis with expectoration of branching casts of the bronchial tree: twenty-seven cases.

II. Acute bronchitis with expectoration of branching casts of the bronchial tree: fifteen cases.

III. Cases in which branching casts were not expectorated, but were found in the bronchi at autopsy: six cases.

IV. Cases in which the casts expectorated showed no dichotomous branching: eleven cases.

V. Expectoration of branching casts in organic heart disease: ten cases.

VI. Expectoration of branching casts in pulmonary tuberculosis: fourteen cases.

VII. Expectoration of small casts, often non-branching, in association with asthma: five cases.

VIII. Formation of casts in the bronchi in association with pulmonary oedema following thoracentesis: four cases.

IX. Cases whose classification is doubtful because of incomplete reports: six cases.

ANALYSIS OF CASES IN GROUP I. *Etiology.* Sex: Males, 15; females, 11; (?) 1.

Age: First decade, 1; second decade, 3; third decade, 5; fourth decade, 9; fifth decade, 4; sixth decade, 2; seventh decade, 2; (?) 1. This shows progressive increase up to middle life, and thence a decline.

Occupation: There are five cases in which occupation is given as possibly having some bearing: Street, "captain of the hold" in a ship; Stirling, dock laborer; Schittenhelm, grave-digger; Roque, blacksmith; Hall, painter.

Infectious Diseases. There is a history of previous pneumonia in Worthington's case, and also in Singer's, Sax's, and Model's (Case

VII.) cases. In Caussaude's case there is onset after the third attack with signs of massive pneumonia. In Roque's case influenza seemed to be etiological.

Chemical Irritants. In Schmidt's case the onset was after inhalation of smoke at a fire.

Family History. There is a family history of tuberculosis in Schi tenhelm's and Beshorner's cases.

Personal History. An almost invariable past history is chronic bronchitis; duration varies much in different cases. In Brannan's case there was a history of angioneurotic oedema, and the author thinks that possibly the condition is the result of angio-oedema of the bronchi. In Mader's case there was a history of pemphigus previous to the onset of the attack, which condition extended to the mucous membranes of the mouth, throat, and larynx. The author is inclined to think that all cases represent a pemphigus of the bronchi.

Symptomatology. The onset is usually with an exacerbation of the chronic catarrh; some fever may accompany it. In Caussaude's case the onset was with a massive pneumonia at the left base. In Roque's case there was a long period which preceded the fibrinous expectoration during which the patient had cough and night-sweats, and gradually became emaciated. A diagnosis of neurasthenia following influenza was made.

Duration—Interval Between Attacks and Duration of Attacks. The one most common characteristic in all the cases outside of the fibrinous expectoration is its tendency to recur in distinct attacks separated by intervals of freedom of longer or shorter periods. It is difficult to give the average duration of the disease, for, of course, many of the cases are lost sight of. Kisch's case is the longest in the history of the disease, lasting over a period of twenty-five years.

Dyspnœa. Paroxysmal attacks of dyspnœa generally immediately precede the fibrinous expectoration; but, strange to say, there are several cases in which the author distinctly states that there was very little dyspnœa: Singer, Model (Case VII.), Schmidt, Pousselt and Zenker (two cases). Evidently the respiratory distress is due entirely to bronchial obstruction. Between the attacks in which the larger complete branching casts are expectorated smaller ones or incomplete portions of casts are brought up, causing, as a rule, very slight dyspnœa. In some cases the expectoration of fragments of casts occurs over long periods before the complete casts appear: thus, in Regarde's case for a period of six years smaller non-branching cylindrical structures compared to macaroni were expectorated, causing considerable dyspnœa.

Cough. Paroxysms of coughing accompany the paroxysms of dyspnœa, and are proportionally intense, but in those cases in which the expectoration of casts is not associated with dyspnœa there may be

very little or no cough. Several authors mention that the simple "rauspern" was all that was necessary to cause the expectoration of casts.

Fever. In six cases the paroxysmal attacks of coughing and dyspnoea were associated with slight fever—in Zenker's (Cases I. and II.), Hall's Bernoulli's, Causssaupe's, and Street's cases. The fever range was high. Fever may be said to rarely accompany the attacks. Possibly this might point to a non-inflammatory etiology of the disease in its chronic idiopathic form.

Hæmoptysis. In only two cases was hæmoptysis associated with the expectoration of casts—in Sokolowski's first case and Street's case. In the latter there is suspicion of pulmonary tuberculosis from the history, and only one hæmoptysis took place while under observation—a period of six months. In Sokolowski's case, however, the hæmoptysis was copious and usually accompanied the expectoration of casts, which took place daily for a period of nine months. In Schittenhelm's case the hæmoptysis began at the onset of a prior tuberculosis which complicated the fibrinous bronchitis, resulting fatally. Stirling's case on two occasions had hæmoptysis amounting to one ounce, but the author does not say whether there was any association with expectoration of the casts.

In Model's (Case VII.), Mader's, Regarde's, and Worthington's cases the casts were occasionally blood-tinged. Hæmoptysis may be said, then, to rarely accompany the expectoration of casts.

Physical Signs. It is impossible to make a diagnosis from the physical signs. Theoretically if the casts were *in situ* in the bronchi—that is, before the expectoration—the physical signs would be those of what the Germans call "bronchial stenose"—that is, impairment or absence of breath and voice sounds in an area of the lung that is obstructed, with no change in the percussion note. Impairment of resonance is often noted, however, in an area apparently involved, and is usually explained on the ground of collapse of the lung as a result of persistent bronchial obstruction. All types of râles are heard, from crepitant to the large, dry, sibilant râles. The most characteristic adventitious sound is the so-called "ventilgeräusch" of the Germans, "bruit de drapeau" of the French. This is a very coarse, dry, clicking sound, apparently from its character, caused by the flapping to and fro of loosened portions of the cast with inspiration and expiration. Model, Beshorner, and Causssaupe have noted it especially in their case before the expectoration of the casts. Localization of the sign is usually in the lower portions of one or both lungs. Chronic emphysema with its characteristic signs is often associated.

General Symptoms. In several cases a considerable degree of loss of weight and strength has been noted (Baumgarten, Championnière,

Street, Waldenburg, Sokolowski, and Roque). Baumgarten attributes the emaciation in his case to the withdrawal of alcohol. Sokolowski is attributable to the frequent hemorrhages from the lungs. Undoubtedly however, the emaciation is not an uncommon symptom, due in many cases to the very frequent and exhausting attacks of coughing and dyspnoea. Night-sweats without any discoverable tuberculosis have been associated in a few cases. (Roque and Street.)

Subjective Symptoms. A feeling of oppression and tightness in the chest is the rule, with complete relief on expectoration of casts. The patient can often tell from which portion of the chest the casts come and a peculiar subjective sensation in one region. (Schmidt and Causseaud.) Pain is not a common symptom, though it is marked in some cases.

Complications, Sequelæ, and Results. Complication with some form or other of skin disease have been noted frequently. Thus in Madey's case pemphigus of the skin and mucous membranes preceded the attack of fibrinous bronchitis. In Waldenburg's case an impetigo capitis was noted six months after the symptoms had disappeared. Street's case was complicated by herpes zoster over the shoulder, and later by the eruption of an impetiginous character over the forehead. During the prevalence of the eruption the patient was free from attacks of fibrinous bronchitis.

In three cases the patient was under observation at the time of death. Schittenhelm's case died as the result of florid tuberculosis. The occurrence of fibrinous bronchitis in the course of tuberculosis is common, and will be seen later. This is the only case, however, of tuberculosis supervening on an attack of fibrinous bronchitis. Sokolowski's case was autopsied. He died as a result of emaciation and anæmia. Madey's case died six months after the last attack of fibrinous bronchitis, the cause being laryngeal stenosis due to cicatrization of a laryngeal ulcer and hypostatic pneumonia. In only one case (Sokolowski's) was the fatal termination the direct result of the disease.

Macroscopical Characters of Casts. In Schittenhelm's case the casts were 10 to 15 cm. long. In Pousselt's case the picture of a cast 10 cm. long is given; in Beshorner's case, 10 cm. long; Stirling's case, 3 to 4 to 6 inches long; Kisch's case, 3 to 8 cm. long; Bernoulli's case, one cast 10 cm. long with ten branches. Championnière gives an excellent picture of a cast 10 cm. long; Waldenburg's case 4 to 5 inches long. All authors who give accurate descriptions note the lumen sometimes in the larger, sometimes in the smaller branches. Championnière noted the separate whirls inside of an outer laminar layer. Dr. Osler examined the casts in Worthington's case and states that "each cast consists of a variable number of laminæ arranged concentrically, those in the centre being much folded and involuted." Most of the branches are cylindrical, some of the larger ones are f

Irregularities on the surface of the cast are often noted, and are supposed to be due to bubbles of contained air.

Microscopical Characters of Casts. In eight out of the twenty-seven cases no detail is given as to the microscopical structure of the casts. The usual description is of a fibrillar stratified ground substance containing in its meshes leucocytes. Pousselt, Schittenhelm, and Model are the only authors who state definitely what the characters of these leucocytes are, and they found them to be for the most part mononuclear leucocytes. Sokolowski, Kisch, and Stirling found red corpuscles. Schmidt finds peculiar bodies in the casts which he considers analogous to the rows of red blood-corpuscles, such as are seen in thrombosed capillaries. He also finds corpora lecitinoidea. Alveolar epithelial cells are commonly found, but no authors mention cylindrical epithelial cells, and in my case, after a careful search through many sections, I did not find one. No authors reporting cases in this group mention eosinophiles. In Model's (Case VII.) and Waldenburg's case there were very numerous fat-droplets throughout the casts, and in Model's case the milky appearance of the sputum led the author to suspect that his patient was fooling him. His conclusions with reference to his case are that it represents a lymphorrhagia or chylorrhagia, for (1) a chemical analysis of the sputum and casts gives the same percentage of fat as is found in chylous exudates; (2) the cells in the casts were mostly lymphocytes; (3) there was no dyspnoea or fever associated with the expectoration; (4) the remissions over long periods are exactly similar to those seen in lymphorrhagia or chyluria. He concludes generally that many cases of fibrinous bronchitis may represent a chylorrhagia or lymphorrhagia, for (1) it is in many cases secondary to disturbances of circulation; (2) the absence of epithelium in the casts and the intact mucous membrane found at autopsy point to this explanation.

Charcot-Leyden crystals were found in both of Zenker's cases. Five authors state definitely that they found none. Other authors do not mention these structures. We shall hear more of this in connection with the cases related to asthma.

Chemical Composition of Casts. Most authors simply state that the main mass of the casts were composed of laminae of fibrin, being influenced wholly by the past views with reference to this and the laminated structure and general consistency of the casts. Championnière (1876) was the first to suggest that the casts were not composed of fibrin but of mucin. He says that microscopically it has not the reticulated appearance of fibrin "mais bien l'aspect uniforme et presque hyalin des matieres muqueuses." It resists the action of acetic acid. He noted little irregular whirls enclosed by an outer lamina, which whirls he considered to be mucous moulds of the bronchial glands. Caussaude (1889)

came to the conclusion that the casts were composed of syntonin from the following data :

- a. Are insoluble in lime-water ; do not swell in water at a temperature of 45° ; are soluble in acetic acid ; are therefore not mucin.
- b. Insoluble in sulphate of sodium and chloride of sodium and deposit in very irregular whirls and circles, and the fact that it does not take the Weigert stain. It is, therefore, not fibrin.
- c. Put in the incubator and immersed in a 1 : 1000 solution of hydrochloric acid at the end of twenty-four hours some solution had taken place.

The filtrate when neutralized gave a precipitate which redissolved on the addition of lime-water. Are, therefore, syntonin.

Stirling stated that they consisted of coagulated albumin, soluble in alkalis. Beshorner (1893) states that the casts are not fibrin, for lime-water dissolves them ; they do not swell up in acetic acid, and, excepting a few fibrils, they do not take the fibrin stain. The fibrillæ that take the stain look like the fibrillæ in the central thread of Curschmann's spirals. The author concludes that the exudate is like that in enteritis pseudomembranacea. Pousselt describes portions that take the fibrin stain enclosing mucus which does not take the stain, and is surprised that sections at different levels show portions morphologically similar, some of which take the fibrin stain and some of which do not. Schittenhelm concludes that chemically they are fibrin, for they digest in artificial gastric juice and decompose in hydrogen dioxide. The fibrin stain shows portions which take the stain and which enclose mucus in which are embedded cells.

Study of Bronchi at Autopsy. Schittenhelm's case was fatal as a result of florid tuberculosis. He could determine no especial localization of the tubercles about the bronchi. He found the alveoli and the bronchi of the right lower lobe filled with desquamated epithelial cells and fibrin. The bronchial mucous membrane showed nothing except dilated mucous glands which contained desquamated cells and mucus. The peribronchial tissue showed dilated capillaries infiltrated with leucocytes. Some of the bronchi of the right lower lobes contained plugs but no definite branching casts. The fibrin stain gives fibrin fibrils throughout the tissue of the lungs, especially about the tuberculous areas (but the author does not lay stress on this, as it is found in uncomplicated tuberculosis). Streptococci are seen free in the lumen and in the glands of the bronchi, but none are found in the tissues. No pneumococci, no diphtheria bacilli. He concludes that the disease is the result of a desquamative catarrh of the alveoli of the lobe of the lung and an acute exudation into the bronchi, bronchioli, and alveoli.

GROUP II. The second group of cases, the acute cases with exudation of branching casts, number fifteen.

Etiology. Sex: Males, 11; females, 3; (?) 1.

Age: First decade, three; second decade, six; third decade, one; fourth decade, four; seventh decade, one. There is no striking preponderance in youth.

Occupation: Occupation was deemed etiological in the following cases: Glasgow's case, a painter; Ott's case, a knife-sharpener; Sokolowski's case, a physician who was doing bacteriological work, and the author thinks it was a laboratory infection, as he cultivated streptococci and staphylococci from the sputum.

Acute Diseases. (a) *Pneumonia.* Koch's case, a man, aged eighteen years, had suffered three attacks of pneumonia, the last of which was characterized by great dyspnoea, a great deal of cough and expectoration, the latter containing numerous non-branching "gerinnsel." Temperature fell on the seventh day by lysis, and six days later the patient was seized with a violent pain and a paroxysm of intense dyspnoea and coughing, which was relieved by the expectoration of three or four branching casts. In Ott's case the expectoration of branching casts preceded the pneumonia; there was no expectoration of casts during the disease. He cultivated the staphylococcus pyogenes aureus and Fränkel's pneumococcus from the casts.

(b) *Typhoid Fever.* In Moller's case there was expectoration of casts coming on twenty-three days after the onset of so-called "typhoid." The diagnosis was perhaps doubtful.

In Souque's case, on the thirtieth day of a typhoid complicated by cutaneous abscesses which kept the temperature slightly elevated, there was a paroxysm of intense dyspnoea which was sufficient in duration to make tracheotomy necessary. No relief was experienced from this operation, and the tube was removed. From the tracheal wound a branching cast 15 cm. long was coughed up. There is no further history.

In this connection I might mention a case which I have not classed in any group, but which is both interesting and unique. This is the one reported by Eisenlohr. A typical case of typhoid with considerable bronchitis at the onset and expectoration, without respiratory distress or effort, of exquisitely branching casts on the second, third, fourth, and sixth days after the onset. The casts dissolved in lime-water and contained red blood-corpuscles, leucocytes, and alveolar and cylindrical epithelium.

(c) *Scarlet Fever.* (Aderson.) Scarlet fever followed by bronchopneumonia preceded the fibrinous bronchitis by some months.

(d) *Measles.* (Yäger.) Onset two days after convalescence from measles.

Basedow's Disease. Fritsche reports a case in the course of Basedow's disease.

Symptomatology of the condition as concluded from twelve out of the fifteen cases is somewhat as follows. (We exclude Koch's case following pneumonia; Souques', in typhoid, and Vintras' case.)

Onset. Preliminary acute bronchitis in eight cases varying in duration; in Herzog's case it had existed four weeks before the expectoration of casts. The characteristic expectoration usually appears about the end of the first week.

Chills. In Bettelheim's case chill at onset, followed three days later by the expectoration of casts. In Kretschy's case the patient had a chill every evening up to the fifth day of the attack. He died on the eleventh day. The author thinks it represents an acute infection.

Glasgow's case: A chill just before the expectoration of casts.

Fever. The fever range is high, in Kretschy's case averaging 40° C. in the evening. This high fever preceded the expectoration of casts by five days. In Fritsche's case the temperature was 40° C. in the first attack.

Distinct Attacks. The disease shows the same periodicity that is exhibited in the chronic form, consisting of a number of distinct attacks characterized by fever, dyspnoea, cough, and expectoration of casts. In the interval between the attacks the patient is comparatively comfortable, though still febrile. In Bettelheim's and Herzog's cases there was only one such attack, but in the latter case it was preceded by a fever and catarrhal symptoms in the bronchi, lasting four weeks. In Kretschy's case there were only two attacks, occurring on the fifth and seventh days of the disease. In Glasgow's case there were two attacks with a long interval between the attacks, the second attack being marked by freedom from dyspnoea and great ease in expectoration. It is quite common in all cases to have fragments of casts expectorated for long periods before and after the characteristic expectoration. In Fritsche's case there were two attacks with an interval of four days. The paroxysms generally occur in the morning.

Pain. The pain in the thorax is usually very intense during attacks. A feeling of oppression in the chest exists between the attacks.

Hæmoptysis was found in only two cases—Bettelheim's and Herzog's.

Number and Macroscopical Structure of Casts. The casts are remarkably few in number. In Bettelheim's case only two complete casts were expectorated. In Kretschy's case only four (duration, eleven days, resulting fatally). Macroscopically there is no difference from those seen in the chronic form.

Microscopical structure does not differ from those seen in the chronic form. Koch found Charcot-Leyden crystals in the casts. Aderson, Ott, and Vintras state definitely that these structures were not present. Herzog, from staining reactions and chemical tests, concluded that the casts were fibrin. Koch found that most of the cells were mononuclear

leucocytes. In Vintras' case the casts dissolved in lime-water. Souques thinks they were fibrin from their morphological appearance, but states that they did not give the fibrin reaction.

Result. Naturally some of the cases in this group might very well be the first attack of a condition which was to become chronic. Bettelheim's and Glasgow's cases suggest this, for after the subsidence of the acute attacks they had slight attacks for a month or so. In several of the chronic cases the first attack is very like in symptomatology to the acute form, and undoubtedly there are transitions between the two which might well be classed as subacute.

In the fifteen cases here considered four were fatal and three autopsied.

Autopsies. 1. Kretschy's case. Young man. Cast found *in situ* in right middle and upper lobes. The mucous membrane of the bronchi of one lung showed loss of epithelium and infiltration with white cells. The other lung showed œdema.

2. Yäger's case. Child, aged five years, died, the expectoration of casts diminishing with increasing dyspnoea and aphonia. Section showed a fresh adhesive pleuritis and pericarditis—bronchopneumonia of the left lung—a weak, incompletely adherent deposit in the larynx, which became thicker and more solid as bifurcation was approached, and extended into all the branches of the bronchi of the left lung and a little way into the right. The case followed an attack of measles. In its autopsy finding and clinical course it is almost identical with Hilton Fagge's case, which we have quoted above.

3. Vintras' case is unique in the history of this disease. An emaciated soldier, aged sixty-eight years, had suffered from cough for three months before admission. He had copious hæmoptysis a week before admission. Four days before admission an attack of intense cyanosis and dyspnoea relieved by expectoration of a branching cast. Seven days later another attack, and fifteen casts were coughed up. Seven days later the patient died in another attack.

At autopsy a rapidly growing epithelioma was found in the œsophagus opposite the bifurcation of the trachea, which latter would hardly admit a goose-quill. A cast was found projecting from the right bronchus into the trachea and obstructing the already strictured lumen of the latter. One small cast was also detected in the tubes of the right middle lobe.

GROUP III. The next group of cases includes those cases in which casts were found at autopsy, but were not expectorated during life. They number six. Clinically they do not belong to the group of fibrinous bronchitis. The probability is that many such cases go undiagnosed because no autopsy is made. Hutchinson's case is probably a case of diphtheria of the larynx in which extension of the membranes into the bronchi followed tracheotomy.

The two cases reported in Virchow's *Archiv* (1879) followed rupture of caseous lymph glands into the bronchi, both in very young children. Escherich's case was a fatal case of double pneumonia. Mazotti's case, a woman, aged seventeen years, died at the end of two weeks with high fever and dyspnoea. At autopsy the lesions of typhoid were found and a fibrinous inflammation of the bronchi. (This makes in all four cases showing an association with typhoid.)

Cutler's case is the only idiopathic case in the group. A woman, aged sixty-five years, had had acute bronchitis for three days; intense dyspnoea and cyanosis supervened, resulting in death in three hours. Section showed "pharynx and larynx healthy. A croupous membrane lay reflected on itself over the entrance of the two primary bronchi. When laid in place this membrane reached more than half-way up the trachea. Downward it extended into the minutest divisions of the bronchi, in many of them forming an almost solid plug. The alveoli contained no solid matter, and except in a few places where there was oedema and collapse they contained air." Evidently in these cases death occurred from asphyxia, because the casts were not expectorated. In this connection the rather bad prognosis of the disease in the two extremes of life might be mentioned, possibly having some relation to the fact that expectoration is difficult at these periods. A study of the cases in this group and the fatal cases of the second group shows the following facts: Out of twenty-one cases ten were fatal.

In the second group, Möller's case, aged thirteen years, not autopsied; Kretschy's case, a boy, autopsied; cast in bronchial tree. Yäger's case (measles), aged five years, autopsied; cast in bronchial tree. Virtras' case (epithelioma), aged sixty-eight years, autopsied; cast in bronchial tree.

In the third group, Cutler's case, aged sixty-five years, autopsied; cast in bronchial tree. Mazotti's case (typhoid), aged eighteen years, not autopsied; associated with typhoid. Escherich's case (pneumonia), aged twenty-seven years, autopsied; casts in bronchi. Virchow's *Archiv* (rupture of bronchial lymph gland), two cases, aged four months and four years respectively, autopsied; casts in bronchi. Hutchinson's case (diphtheria), child, autopsied; casts in bronchi and trachea.

GROUP IV. The fourth group of cases includes all those in which, although no definite arborescent moulds of the bronchial tree were expectorated, fibrinous structures evidently emanating from the bronchi were: eleven cases.

The symptomatology in some of the cases is very much like the symptomatology of the cases in the first and second groups. The difference is, perhaps, only a matter of degree, in this group the exudation being less in amount, perhaps more limited in area, and the expectoration taking place before a definite branching cast is formed.

In going over these cases one is impressed with the fact that the authors in many instances have reported in the same article cases belonging to the first and second groups. Thus have Chvostek, Kretschy, Model, Sokolowski, and Chauffaud. The natural inference is that these authors, having observed one case in which the expectoration contained these wonderful arborescent casts, kept their eyes open for other similar cases. It is well to separate these cases from those in the other groups to bring out the frequency of the cases in which arborescent casts are expectorated, or, rather, the infrequency. It might be mentioned that fibrin is frequently present in the expectoration in ordinary bronchitis. I have a specimen of a plug expectorated in a chronic bronchitis showing quite a marked content in fibrin (Weigert's stain). There are eleven cases in the group.

GROUP V. The fifth group of cases includes all those associated with organic heart disease—ten cases. There is no good reason why the cases in this group should not be included in the two groups of idiopathic cases. Heart lesions predispose to bronchitis through congestion. If this bronchitis assumes a special type there is no reason to suppose that the heart lesion is the etiological factor. We certainly consider the bronchitis of heart disease as an idiopathic bronchitis, though recognizing that a predisposing factor lies in the organic heart lesion. In Bernouilli's, Degen's, Lawrence's, Hint's, and Habel's cases the condition was chronic. It is worthy of note that all these cases were afebrile, and in three of them dyspnoea was absent during the attacks. This might indicate that the process was purely mechanical, and not inflammatory, the exudate taking place quickly and as a result of congestion. Seven of the cases were under observation at the time of death. In Degen's case, in a boy, aged sixteen years, the duration of the disease had been eight years; the autopsy findings were adherent pericarditis externum with exudation, aortic and mitral insufficiency; brown induration of the lungs. The right main bronchus and its branches contained a cast *in situ*, also the bronchi of the left lower lobe. Rohr's case: a woman, aged twenty-one years, under observation during the last month and a half of life, during which time she had loss of compensation and three attacks of fibrinous bronchitis. At autopsy: mitral insufficiency, infarct of lung, no casts, a tubercular kidney.

Beshorner's case; a male, aged forty-nine years. No clinical history. At autopsy a pleuritic exudation, adhesive pericarditis, nephritis, and peribronchitic infiltration of the lungs. It is worthy of note that in three out of the five cases that were autopsied adherent pericarditis was found. In Habel's, Hint's, and Schittenhelm's cases (all of which were under observation at the time of death), when compensation was lost, there were no longer any attacks of fibrinous bronchitis. Schittenhelm's case resembled an acute fibrinous bronchitis, the other two were chronic.

Structure of Casts. The four authors who investigated carefully found that the casts were not in the main composed of fibrin. Grandy and Beshorner found that the casts did not take the fibrin stain at all. Habel found a few fibrillæ staining for fibrin. Hint concludes that it was mucin not fibrin on rather insufficient data. No authors mention Charcot-Leyden crystals or eosinophiles.

Study of Bronchi at Autopsy. Grandy made a very careful study of the condition of the bronchi and of a cast *in situ*. He found dilatation of the vessels of the bronchi, clumps of leucocytes especially about the bases of the mucous glands, which latter were much dilated and filled with mucus, and it could be seen that this mucus merged into the casts. There were very great numbers of goblet cells present. The character of the staining reaction of the mucus was the same as that of the outer layer of the cast (blue with Van Gieson, red with thionin); but in the inner part the reaction had changed somewhat, showing that the mucus had altered in character.

Heart Conditions in this Group. Mitral insufficiency in five cases; mitral insufficiency and stenosis in one case; mitral stenosis in one case; mitral insufficiency and adherent pericardium in one case; adherent pericarditis in two cases.

GROUP VI. The sixth group includes those cases associated with tuberculosis of the lungs. There are fourteen in all, ten of which were under observation at the time of death, and were autopsied.

The expectoration of casts generally occurs late in the disease. In Homolle's case two casts were expectorated, one eight months before death and one eleven days before death. In Fraentzel's case casts were expectorated for the last seventeen days of life. In Pramberger's case a cast was expectorated fifty-five days before death and one eleven days before death. Jaccoud's case just one day before death expectorated two casts. Model's case (Case IV.) expectorated casts for two months before death. Model's case (Case V.) for ten days previous to death. Sokolowski's case, thirty days before death expectorated one cast. Casts were found at autopsy *in situ* in three cases—Homolle's (Case V.), and Klein's cases.

Model found a caseous infiltration of the walls of the right upper bronchus in which a cast was found *in situ*. Magniaux found peribronchial miliary tubercles in both apices, with denudation of epithelium and infiltration with leucocytes.

Structure of Casts. The casts are very few in number, and are the typical large branching structures found in the idiopathic form. All authors in this group state on rather insufficient evidence that the casts are composed of fibrin. Klein is the only author who goes carefully into the composition, and he finds that it does not take the fibrin stain and that it dissolves in chloroform. No authors mention staining sections of the casts for tubercle bacilli, so that if these peculiar bodies

which we have described in our cases were present they were overlooked. Tubercle bacilli were always found in the sputum.

All possible tuberculous conditions of the lung are found in this group of cases. Magniaux cultivated the diplobacillus of Friedländer from the blood, organs, and casts.

GROUP VII. Occasionally in the literature on bronchitis fibrinosa there is a reference to the relation of this disease to asthma. The suggestion of a relationship is brought about by the fact that there is a group of cases in which small fibrinous structures are expectorated along with the common contents of asthmatic sputum, Charcot-Leyden crystals, Curschmann's spirals, and eosinophiles. Are these cases to be classed as asthma or as bronchitis fibrinosa?

Schmidt has made elaborate studies into the sputum in asthma, and he found more or less large fibrinous structures in six out of eight cases of asthma which he investigated, using Weigert's stain to demonstrate the fibrin. He gives two cases especially in which the "gerinnsel" were quite large—2 to 3 cm. long. He describes them as "fädige und flockige gerinnsel," which were composed for the most part of fibrin, and also contained some mucus in spiral arrangement, many eosinophilic cells, and numerous crystals. One of these cases was associated with hypertrophic rhinitis, and suffered typical asthmatic attacks. His conclusion is: "Berücksichtigt man aber, das wiederholt auch bei fibrinöser Bronchitis Spiralen gefunden worden sind, so dürfte es in der that schwer sein, Fälle wie diesen, welcher dem Sputum nach eher zur fibrinöser Bronchitis, dem klinischen Bilde nach zum Asthma gerechnet werden muss, zu classificieren." The author also found small fibrinous structures in two cases of ordinary bronchitis. He concludes that the exudation of fibrin is quite common in inflammatory conditions of the bronchioles. (My preparation under the microscope is a plug from a case of ordinary bronchitis in emphysema. It shows fibrin in fair amount. Demonstrated at the Johns Hopkins Medical Society, June 2d.) Pousselt makes an elaborate analysis of many cases of bronchitis fibrinosa from the point of view of the relation of this disease to asthma. He concludes that the cases reported as fibrinous bronchitis can be classified into two distinct groups:

(a) "One in which the casts consist almost exclusively of fibrin containing very few cells and little or no mucus, and in which the expectoration takes place without any asthmatic attacks or the least subjective inconvenience. A relationship to any other pathological condition is not demonstrable. The etiology is obscure. There are very few cases of this essential primary fibrinous bronchitis. The course is chronic, with subacute, rarely acute attacks, in which casts are expectorated."

(b) "Second group. Very much more numerous than the cases of the first group. There are cases in which the fibrinous bronchitis is complicated by an asthma in which the casts are rich in cells and crys-

tals, and spirals are often found. The casts are also rich in mucus. The course is much more severe and acute." I will not tire you with the gross misstatements and contortions of fact upon which this classification of Pousselt's is based. They are numerous, occupying about one hundred pages. His other conclusions are that bronchitis fibrinosa is often complicated by asthma, but that the two processes are entirely distinct—the former not being in any case an extension of the asthmatic exudation into the larger bronchi. However this may be, the fact remains that there is a well-marked group of cases in which the so-called "asthma-eueger," Charcot-Leyden crystals, Curschmann's spirals, and eosinophilic cells are expectorated along with small fibrinous structures representing small casts of the finer bronchi, but generally not branching. The amount of dyspnoea in the attacks in these cases is much greater than the size and number and form of the casts would indicate if one looked to *bronchial* obstruction as the etiological factor in causing the dyspnoea. One is, therefore, tempted to classify these cases as asthma in which the exudative process has extended beyond the bronchioles in some areas of the lung, causing the formation of casts, generally not branching in some of the smaller bronchi. "Leyden has stated that one might consider asthma as a fibrinous bronchiolitis which occasionally can extend to the large bronchi, so that large 'gerinnsel' are expectorated. Nevertheless, one cannot identify asthma with fibrinous bronchitis, for there are many cases of the latter affection which have nothing to do with asthma. The cases which are related to asthma are characterized by the fact that the casts are filled with Charcot-Leyden crystals. Fränkel agrees with Leyden as to the relationship of the two diseases, but does not think asthma should be designated a fibrinous bronchiolitis, for the exudate is not for the most part fibrin." (From Pousselt.)

In going over the cases since 1869 I find that in five out of the seven cases in which Charcot-Leyden crystals have been found in the casts the latter have been small, generally possessing very few or no branchings. I am, therefore, tempted to place these in a group by themselves, showing a transition between fibrinous bronchitis and fibrinous bronchiolitis (asthma). They certainly represent a group distinct from the first and second groups, in which the casts are very large and show exquisite and numerous dichotomous branchings.

In Vierordt's case Curschmann's spirals appeared after the fibrinous structures had disappeared, thus suggesting the alternate occurrence of asthma and fibrinous bronchitis.

I am fortunate in being able to present, through the kindness of Dr. Osler, a case which I think represents this group well, and will bring in marked contrast the case spoken of earlier in the paper.

Kate M., aged forty-five years, white; first admission to hospital March 6 to 15, 1900. Complaint: Cough for several years, and lately some smothering sensation on lying down.

Family History. Father died of "asthma." Mother died of dropsy. Two sisters died of consumption.

Personal History. Has had eight children. Had a severe attack of rheumatism eight years ago. Is frequently a sufferer from tonsillitis. During the last sixteen years Dr. Mackenzie has removed about twenty polyps from the nose.

Present Illness. Has suffered from cough for periods throughout the last twenty years. The cough comes on in paroxysms at night, and is associated with attacks of shortness of breath. She says she can bring on a paroxysm by spraying her nose. She brings up considerable expectoration during these attacks.

Physical Examination shows typical signs of mitral insufficiency, with good compensation. Herpes about the lips and nose; tonsillitis while in the hospital; no note on the sputum; trace of albumin in the urine; hæmoglobin, 68.7 per cent; no temperature.

Second admission, April 15th; June 2, 1901. Complains that she has never been free from cough since she left the hospital. For the last two months this cough and "smothering feeling," as she calls it, have been worse.

Present Illness. Has been kept in bed on account of her heart lesion ever since entrance to the hospital. During this time she has had frequent attacks of dyspnoea and coughing and considerable amount of expectoration. No fever has been present. The attacks usually occur in the early morning. The respiratory distress is extreme, and vomiting is often associated with the expectoration. In the interval between these paroxysmal attacks she has, as a rule, been free from dyspnoea, though at times she has suffered considerably from it, having to remain in a reclining position in bed.

Physical Examination shows the signs of a chronic bronchitis; a well-marked mitral insufficiency; no signs of loss of compensation; hæmoglobin, 47 per cent.; no albumin in urine.

Sputum. Sputum contains Curschmann's spirals in abundance, eosinophilic cells, no tubercle bacilli. There are a fair number of very small branching casts, none over 3 cm. long, the majority averaging not over 1 cm. in length and only showing one or two incomplete branchings. Curschmann's spirals are seen at the end of the finer branches very frequently, and suggest the relationship spoken of above between bronchiolitis fibrinosa (asthma) and bronchitis fibrinosa. The calibre of the largest cast is not over 2 mm., and no lumen is seen on cross section. The casts consist for the most part of fibrin (as shown by Weigert's fibrin stain), containing leucocytes, generally polynuclear eosinophiles. No Charcot-Leyden crystals are seen. Sections cut longitudinally through the branches which end in Curschmann's spirals show that the fibrin from the casts is continuous with the fibrin in the central thread of the spiral. Streptococci and staphylococci are numerous in the casts.

GROUP VIII. The eighth group of cases includes those associated with acute oedema of the lung after thoracentesis. There are four such described by Ortner. In two of the cases fibrinous casts were expectorated (Hampeln and Ortner's second case), associated with a great amount of albuminous expectoration. In Ortner's case only one cast 3 cm. long was expectorated. The patient, a woman, died two months

later with increasing loss of compensation; adherent tuberculous pericarditis, double tuberculous pleurisy, œdema of the lungs, congestion of the liver, and ascites were found. At autopsy there were no casts in the bronchi. Ortner and Scriba describe cases which resulted fatally in which casts were not expectorated, but were found *in situ* at autopsy. In Ortner's case, in the right lung; in Scriba's, in the left, corresponding to the side on which thoracentesis was performed. In Ortner's case the autopsy findings were adherent pericarditis, mediastinitis, congestion of the liver, and ascites. In Scriba's case tuberculous pleurisy and pericarditis were found at autopsy. In this connection it is interesting to recall that in three out of the five cases of fibrinous bronchitis associated with heart disease which were autopsied, adherent pericarditis was found also in one case in the second group adherent pericarditis was found.

GROUP IX. The ninth group includes a few cases which I have been unable to classify through dearth of description. (See References.)

In going over the literature I came across an interesting and unique case reported under the title of "Bronchite Pseudomembraneuse Aspergillaire Primitive," by Devillers et Renon, in *La Presse Médicale* for 1899. It affected a woman, aged forty years, who coughed up for a considerable time greenish membranes emanating from the bronchi entirely composed of mycelium of *aspergillus fumigatus*. Her profession consisted in selecting seeds for cultivation. Her method of testing them was to feel their consistency with her teeth. It is supposed that she thus acquired this peculiar infection. Attacks of dyspnoea and cyanosis preceded the expectoration.

Expectoration of blood-casts of the bronchi following hæmoptysis is rather rare, and in consulting the literature I came across only three cases the references of which are given.

NOTE I.—*Note with Reference to the Relation of Pregnancy and Menstruation to Fibrinous Bronchitis.*

No such relation was made out in the case of Maggie S. Schnitzler (1864) observed a case. A woman after her sixth labor, instead of having her menstrual flow at the eighth week (as was her custom), had an attack of fibrinous bronchitis. For several months this phenomenon recurred. After her seventh and eighth labor the author made the same observation. This same case was again observed by Brik seven or ten years later, when the woman was fifty-nine years old, in an attack of fibrinous bronchitis. Evidently at this age neither pregnancy nor menstruation could have had an etiological influence.

Pousselt quotes Stofelln, who observed a case in which attacks of fibrinous bronchitis replaced menstruation.

NOTE II.—*The Bacteriology of Fibrinous Bronchitis.*

In the cases considered in this paper the following bacteriological facts have been elicited:

GROUP II. Souques demonstrated streptococci, pneumococci, and staphylococci in the casts. Ott cultivated the staphylococcus aureus and the pneumococcus from the casts. Herzog demonstrated staphylococci and bacilli in the casts. Koch found bacteria which retained the stain by Gram's method always enclosed in mucus. He therefore does not consider them etiological.

GROUP IV. Sokolowski cultivated the staphylococcus albus and aureus from the sputum—a fact having no bearing whatsoever.

GROUP IX. Sokolowski demonstrated staphylococcus and streptococcus and a tetragenous conus in the casts. Neither diphtheria bacilli nor tubercle bacilli have ever been demonstrated in the casts.

Landireux and Triboulet (reference given under "Foreign References not Read") cultivated pneumococcus from the casts. It is an accepted fact that staphylococcus, streptococcus, and pneumococcus are etiological in the production of simple purulent bronchitis, so that their presence in the casts does not wholly explain the etiology of this rare disease. One observer has gone so far as to claim that in a case of his antistreptococcal serum was curative. Hoffmann (Nothnagel's *Handbuch*, xiii. Bd., iii. Th., 1 Abth.) recognizes two organisms as etiological: the pneumococcus and the diphtheria bacillus. He admits that the pneumococcus can be demonstrated in simple purulent bronchitis, but ventures the conjecture that cases in which it is alone or principally active develop fibrinous bronchitis; whereas, the purulent form appears where streptococci or other cocci overgrow it. This is only a conjecture.

Pinhini isolated a bacillus in three cases which, inoculated into the trachea of rabbits, practically reproduced the disease. Inoculation into other parts of the body was with negative result. His three cases occurred in three bricklayers who were working in the neighborhood of a sewer, and are supposed thus to have acquired the infection. I have not read the original article, "Contributio allo studio della Bronchite fibrin, acuta prim," in the *Riv. clin. arch. ital. di clin. med.*, 1889, p. 105.

COLLECTIONS OF CASES.

For all references previous to 1869, see Lebert's article. References follow the text, being arranged in groups.

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GROUP II. 15 Cases. *Acute Fibrinous Bronchitis: Branching Casts.*

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 1873. Bettelheim. Casuistische Mitth. über B. Fibrinosa, Wien. (Schmidt's Jahrbücher, vol. clxlii.)
 1873. Kretschy. Wiener med. Wochenschrift, Nos. 14, 15, 16.
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 1879. Glasgow. Transactions of American Medical Association for 1879.
 1883. Aderson. Hosp. Tidende, 3 R. i. 49-50. (Schmidt's Jahrb., vol. cciv.)
 1882. Yäger. Württemberger Aerztliche Correspondenz-Blatt, No. xv.
 1884. Müller. Hosp. Tidende, ii. 12. (Schmidt's Jahrb., vol. cciv.)
 1892. Koch, R. St. Petersburger med. Wochenschr., No. ix.
 1893. Fritsche. Read in Schmidt's Jahrbücher, Bd. cxxxvii. S. 219.
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 1900. Souques. La Independance Médicale, Jan. 17.
 Case in the course of typhoid fever.

1876. Eisenlohr. Berliner klin. Wochenschrift, No. xxxi.

GROUP III. 6 Cases. *Cases in which casts were found in bronchi at autopsy, but were not expectorated.*

1874. Hutchinson. Trans. of Path. Soc. of Phila. (Probably diphtheritic.)
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 1887. Cutler. Boston Medical and Surgical Journal, p. 443.

GROUP IV. 11 Cases. *Cases in which the casts expectorated showed no dichotomous branching.*

1875. Chvostek. Wiener med. Presse.
 1882. Kingsley. St. Louis Courier of Medicine, March.
 1882. Madigan. Chicago Medical Review, p. 184.
 1883. Kretschy. Deutsch. med. Wochenschrift, No. viii.
 1881. Barron. The Lancet.
 1889. Gibson. Practitioner, vol. xliii.
 1890. Model. "Ueber Bronchitis Fibrinosa." Dissertation, Freiburg.
 1893-94. (?) The Hospital, London, p. 240.
 1896. Sokolowski. Deutsch. Arch. f. klin. Med., p. 482.
 1898. Lepine. Rev. de Méd., 1898, vol. xviii. p. 835.
 1899. Chauffarde. Rev. Internationale de Méd. et de Chir.

GROUP V. 10 Cases. *Expectoration of Branching Casts in the Course of Organic Heart Disease.*

1877. Bernouilli. Deutsch. Arch. f. klin. Med., Bd. xx.
 1878. Degen. Württemberger med. Correspondenz-Blatt, Bd. xlviii.
 1886. Stack. Berliner klin. Wochenschrift, S. 221.
 1892. Rohr. Correspondenz-Blatt f. Schweizer Aerzte, vol. xxii. p. 767.
 1893. Lawrence. Lancet, vol. i. p. 247.

1898. Beshorner. Volkmann's Sammlung f. inn. Med., No. III.
 1897. Grandy. Centralblatt f. Allg. Path. u. Path. Anat., vol. viii. p. 513.
 1898. Habel. Centralblatt f. innere Med., 1898, No. 1.
 1898. Hinta. Wiener med. Wochenschrift, vol. xlviii.
 1900. Schittenhelm. Deutsch. Arch. f. klin. Med., p. 338.

GROUP VI. 14 Cases. *Expectoration of Branching Casts in the Course of Pulmonary Tuberculosis.*

1870. Tuckwell. Trans. of Path. Soc. of London (doubtful).
 1874. Flint, Austin. Med. Record, vol. ix. p. 41.
 1875. Homolle. Bul. de la Soc. Anat., t. x. p. 542.
 1878. Fränzel. Charité Annalen, v. Jahrgang, p. 295.
 1881. Pramberger. Mittheil. d. Verein d. Aerzte in Steienmark, 1880, Graz, 1881, vol. xvii, pp. 3-68.
 1884. Müller. Hosp. Tidende, vol. II. p. 12. (Read in Schmidt's Jahrb., vol. cciv.)
 1885. Jaccoud. Quoted from Letellier's Thesis, q. v.
 1890. Model. Ueber Bronchitis Fibrinosa. Dissertation, Freiburg (4 cases).
 1895. Magniaux. Thèse de Paris.
 1896. Klein. Wiener klin. Wochenschrift.
 1898. Sokolowski. Deutsch. Arch. f. klin. Med., lvi. p. 476.

GROUP VII. 5 Cases. *Cases showing a distinct relation to Asthma. Casts small.*

1864. Friedreich. Virchow's Archiv, Bd. xxx. S. 381.
 1861. Pramberger. (See Group VI.)
 1893. Vierordt. Berliner klin. Wochenschrift, No. 29.
 1892. Wolf. Ueber Bronchitis Fibrinosa. Dissertation, Würzburg.

GROUP VIII. 4 Cases. *Fibrinous Exudation into the Bronchi associated with Pulmonary Edema following Thoracentesis.*

1885. Scriba. Deutsch. Arch. f. klin. Med., Bd. xxxvi. (Cited in Ortner.)
 1892. Hampeln. St. Petersburger med. Wochenschrift, S. 336. (Cited in Ortner).
 1899. Ortner. Wiener klin. Wochenschrift, No. 44. (2 cases.)

GROUP IX. 6 Cases. *Cases unclassified through dearth of data.*

1876. Cohen. Transactions of Path. Soc. of Phila. (Laryngeal Tuberculosis.) Vol. v. p. 126.
 1875. Siridez. In Championnière's Thesis, 1876.
 1893. Escherich. Deutsch. med. Wochenschrift, No. viii.
 1884. Müller. Second Case. Read in Schmidt's Jahrbuch., Bd. 204.
 1898-99. Limont. Northumberland and Durham Med. Society.
 1900. Lowenthal. Liverpool Medical and Chirurgical Journal.

MISCELLANEOUS REFERENCES.

"*Bronchite Pseudomembraneuse Aspergillaire Primitive.*"

1899. Devillers et Renon. La Presse Médicale.

Croupous Bronchitis in Animals.

1896. Friedberger u. Fröhner. "Pathology and Therapeutics of Domestic Animals."
Expectoration of Blood-casts of the Bronchi following Hæmoptysis.
 1873. Peacock. Transactions of Path. Soc. of London, vol. xxiv.
 1890. West. British Medical Journal, vol. i. p. 282.
 1893. Richards. Lancet, May, 1893.

Relations of Pregnancy and Menstruation to Fibrinous Bronchitis.

1864. Schnitzler. Wiener med. Halle, p. 450.
 1868. Stoffeln. Oppolzer's Vorlesungen über Spec. Path. u. Ther., Bd. i.

SOME FOREIGN REFERENCES, NOT READ.

1896. Mills. Clinique Brux. "A propos d'un cas de Bronchite Pseudomembraneuse. Recherches Bacteriologiques."
 1896. Landrieux et Triboulet. Rev. gén. de Clin. et de Ther., vol. x. pp. 97-100. "Bronchite pseudomembraneuse aigue à pneumocoques."
 1897. Koren, F. Worsk. Mag. f. Megeverdiensk. Kristiania, 4 R. xii.
 1900. Prik. Riforma Med., Palermo, 1900, vol. i.
 1900. Demel. Gros. d. r. Acad. di Med. di Torino.
 1900. Vyšší. Casopis lékařsk., Praha, vol. xxxix.

REVIEWS.

A TEXT-BOOK OF PHYSIOLOGIC CHEMISTRY FOR STUDENTS OF MEDICINE AND PHYSICIANS. By CHARLES E. SIMON, M.D., of Baltimore, Md. Philadelphia and New York: Lea Brothers & Co., 1901.

THIS text-book, while not so encyclopædic in scope as some of the standard German works on physiologic chemistry, contains all that the student of medicine and the physician for whom the book is written need know. The author has adopted the wise plan of avoiding theoretic discussion, and limits himself to a clear, concise, and well-ordered exposition of the facts of chemical physiology.

A careful review of the different chapters fails to reveal a single statement that is not fully in accord with the most recent research. The author is conservative, and does not often indulge in apodictic statements; where he does so, he is in full agreement with the best recognized authorities.

The book is fully up to date—the more recent discoveries in the field of the oxidizing tissue-ferments, of coagulation by nucleo-histone, the purin bases, etc., are fully and adequately described and characterized. At the same time the classical facts of the science are presented in a precise and comprehensive manner. It also possesses two negative virtues. In the first place, the author wisely refrains from wandering into the clinical field; this temptation very few writers on this subject have been able to resist; the author has enhanced the value of his work by limiting himself strictly to the chemical and physiological aspects of the subject without attempting to teach pathological chemistry or chemical diagnosis; it is possible Dr. Simon realizes that he has treated these two subjects exhaustively in a separate publication, and is anxious to avoid repetitions.

In the second place, happily, all literature references are omitted. Whoever has been forced to seek his way through the protean array of names, titles, and page numbers that disfigure the average text-book of this character will heave a sigh of relief to find the path so clear and unencumbered. Literature references are of value to the original investigator and not to the student and the practitioner. It is really a matter of indifference to the beginner whether A or B or C has discovered this, that, or the other fact. Such is fame! Even the investigator, if he is an investigator, will not be content with those references that are given by any one author in a text-book; he will want the *complete* literature, and will consult an index of his specialty. Wherever we see too many names and too many quotations we place the burden of proof on the author to show that his book is not a mere compilation.

The book is a pioneer; it is the first large text-book of physiologic chemistry that has been written in America; it is timely, for we are on the threshold of a new era in scientific medicine; physiological chemistry

istry, we believe, is destined to throw much light into the obscure recesses that are around us and in front of us.

We welcome Dr. Simon's book most cordially; it fills a want and deserves a place on the shelves of every thoughtful and progressive physician.

A. C. C.

A LABORATORY HAND-BOOK OF URINE ANALYSIS AND PHYSIOLOGICAL CHEMISTRY. By G. CHARLES L. WOLF, B.A., M.D., Instructor of Physiological Chemistry, Cornell University Medical College, New York. Illustrated. Philadelphia: W. B. Saunders & Co., 1901.

This unpretentious little volume fulfils the purpose for which it is written. The first part of the book deals with some of the simpler exercises in physiological chemistry. In the second part, a brief sketch of the most common methods of urinary and gastric analysis are described.

Particular attention is given to clinical questions. There are a number of tables of urinary diagnosis at the end of the book that are conveniently arranged for reference, and present much that is described in the text in a diagrammatic form.

In the preface the author modestly disclaims completeness for his volume; the book certainly contains all that the average medical student at the average American medical college can assimilate within the short time usually allotted to the laboratory study of physiological chemistry—so that in the hands of a competent laboratory instructor the little book should prove a useful guide.

A. C. C.

IRREGULARITIES OF THE TEETH AND THEIR TREATMENT. By EUGENE S. TALBOT, M.D., D.D.S., Professor of Dental and Oral Surgery, Northwestern University Woman's Medical School, Chicago, etc. Fourth edition, with 580 illustrations. Pp. 537 and index. Philadelphia: The F. A. Davis Company, 1901.

A VAST amount of research is represented in the work before us, much of it valuable and interesting, and much that is open to criticism, both as to its scientific accuracy and as to the deductions drawn. One gathers the impression from a critical study of the work that the author in his endeavor to defend his main contention of the constitutional origin of dental irregularities has belittled and practically ignored the evidence on the other side of the question, for unlimited and unanswerable data are at hand to demonstrate that many cases of dental irregularity do arise solely from local causes.

Many of the statements are involved and lack clearness, while others are self-contradictory; thus, at page 15 we find "the peculiarity in the size and shape of the jawbone may be inherited, but the manner of the eruption of the teeth is not transmitted, hence irregularities of the dental arch *per se* are not inherited. The muscles of the cheeks have nothing to do with the production of the V- or saddle-arch. The only

tissues involved are the jawbone on the one hand, the teeth and alveolar process on the other." The idea that irregularities of the dental arch *per se* could or could not be inherited presupposes a common understanding of the term "inherited." Teeth regular or irregular are not *per se* inherited in any strict sense, nor are any other organs for that matter. It is the developmental impress which is the physical hereditary factor, a fact that the author fails to realize, or at least one which he ignores in many instances where the subject of heredity is being dealt with as an etiological factor. The dictum of the author with reference to the causal relation supposed to exist between mouth breathing and contracted dental arches cannot by any means be accepted as fitting in all cases. It is with regard to this question especially, but by no means exclusively, that his statement is rather the contention of the advocate than the opinion of the judge. He asserts (page 16) "that the vault of the mouth is not contracted by mouth breathing." "That mouth breathing (due to hypertrophy of the nasal bones and mucous membrane, deformation of the nasal bones, adenoids, or any pathological condition producing nasal stenosis) does not cause contracted jaws, but all these conditions are due to neuroses of development. On the Continent of Europe the practical acceptance of these views has been shown in the recent translation of Max Bauchwitz of my work on the subject." This is jumping at a conclusion with a vengeance—that Max Bauchwitz saw fit to translate some of the writings of Talbot into German is scarcely evidence that the views of the latter have found practical acceptance on the Continent of Europe. On the contrary, there is on the Continent of Europe a wide spread belief among many thoughtful practitioners of medicine, surgery, and dentistry that mouth breathing due to nasal stenosis does cause a contraction or lateral flattening of the upper dental arch as well as faulty occlusion of the upper and lower arches.

Grevers, of Amsterdam, has shown very clearly the developmental part played by the tongue in giving form and proportion to the upper arch, and the further fact that the tongue cannot properly exert its moulding effect upon the arch when the mouth is continually kept open for breathing purposes.

Cryer (*Studies of the Facial Region*) has further shown the developmental effect of the percussive force of mandibular occlusion which is lost in the mouth-breather. These are factors which cannot be ignored, nor can they be included in Talbot's etiological classification as neuroses of development.

The author is not as precise in the use of his terms as one could wish, and, indeed, has the right to expect, in a really scientific exposition on any subject. His fondness for the theory of degeneracy has resulted in an undue importation into this work of matter related to degeneracy having only a remote and often questionable bearing upon irregularities of the teeth. Indeed, two thirds of the matter could have been omitted or condensed with benefit. In pursuit of his favorite theme of degeneracy he makes frequent allusions to "arrest of development" as an explanation of certain defects. There are unquestionably many cases of arrest of development, but a retardation of development is not to be confounded therewith, which the author does in numerous instances.

In the preface the reader is directed to pursue numerous other works of the author in order to secure a comprehensive grasp of the whole subject, notwithstanding the fact that the author himself has exten-

ively drawn upon his other writings in the compilation of this one. Thirty-one of the thirty-two chapters are taken up with matters exclusive of the practical treatment of cases. The final chapter on treatment is an exhibit of the author's methods to the exclusion of all others, while throughout the work the use of the personal pronoun in the first person singular is rather oppressively apparent. Notwithstanding its flavor of egotism, dogmatism, intolerant critical attitude toward opposing views, many instances of bad English and misspelling of words, the book is one which anyone interested in irregularities of the teeth, their etiology and treatment, should read for the information it contains, but before it can be accepted as a standard authority on the subject it will need more thorough revision and correction. E. C. K.

ANATOMY, DESCRIPTIVE AND SURGICAL. By HENRY GRAY, F.R.S. Edited by T. PICKERING PICK, F.R.C.S., Consulting Surgeon to St. George's Hospital, etc., and ROBERT HOWDEN, M.A., M.B., C.M., Professor of Anatomy in the University of Durham, etc. A revised American, from the fifteenth English edition, with 780 illustrations, many of which are new. Philadelphia and New York: Lea Brothers & Co., 1901.

In this new edition of *Gray* a marked change has been made from previous issues. It is not simply the reissuing of an old book with a few alterations and additions. A new editor, Robert Howden, has assisted the former editor, T. Pickering Pick, and the result is that the entire volume has been thoroughly revised, brought up to date, and its illustrations much improved.

This has been done without materially increasing its size, only eight pages having been added. This we believe to have been wise. As it now stands, complete in one volume, it still fills its place as the students' text-book as well as a satisfactory book of reference for the practitioner. The publishers state that 231 new engravings have been inserted. These are scattered through the book, many replacing old cuts. Owing to a rearrangement of material space has been saved in some places. Thus, the development of the peritoneum is now included in the section on embryology, and not in that on the organs of digestion. The surgical anatomy of the organs of digestion has been entirely rewritten and brought up more in accord with recent practice. There are a couple of most excellent new cuts, showing a posterior view of the kidneys, colon, and great vessels in relation to each other and to the bones. The cuts on the teeth *in situ* are decidedly superior to those in the previous editions. In the section on the Skeleton thirty-four additional cuts have been inserted besides those which replace old ones. The text in the chapter on Articulations has not been changed much, but many of the illustrations have been entirely redrawn. The muscles of the perineum are included under the section on Muscles, there being besides a separate chapter devoted to the surgical anatomy of the perineum. Instead of considering the heart in a separate section on the thorax, it is included in the section entitled the Blood and Vascular System. This section also describes the arteries and the veins.

The description of the lymphatic system bears evidence of careful revision; this applies not only to the purely anatomical part, but also

to that on its surgical application. This is of particular value in reference to the active interest which is now being taken in glandular infection in cancer and other diseases. The section on the Spinal Cord and Brain has been practically written over again and many of the illustrations redrawn.

The same careful revision has been carried through the chapters on the Cranial and Spinal Nerves.

The section on the Organs of Special Sense likewise bears evidence of having been carefully scrutinized and, where desirable, altered. Some new material has been added to the chapter on hernia. The section on General Anatomy and Embryology has been placed at the end of the volume instead of at the commencement, as in the old edition. It has been entirely rewritten from a more modern stand-point. Instead of beginning with a description of the blood, it starts with the cell, its character and properties. Seventy-six additional illustrations have been inserted in this part of the volume, many being reproduced from the excellent atlas of His. In this section is included the development of the various organs, and taken altogether it forms a most valuable chapter both for study and reference. The previous voluminous index has been made still more complete by the addition of two pages. This makes the book particularly satisfactory in hunting up disputed points. There is no question that the original author of the work, Henry Gray, possessed in a remarkable degree the faculty of presenting anatomical facts in a clear, concise, and attractive manner, and it is a great pleasure and satisfaction to see that notwithstanding the numerous revisions to which the work has been subjected the genius of Gray is still preserved. More attention is given to the applied feature of anatomy both in regard to its topographical and surgical and medical aspects than was the case in previous editions. This, of course, is due to the advances made in anatomy in these directions in recent years, and makes the book more suitable for use at the present time than previous editions would be. In this edition half-tone illustrations have been used to a greater extent than formerly. The manner of illustrating text-books is a subject with which publishers are now grappling. What the ultimate outcome will be is hard to say. The combination of half-tone and colors as used in this work may be a solution of the problem. At all events a case such as that of the popliteal artery, on page 580, in which the arteries are red, the veins blue, and the nerves yellow, leaves little to be desired. Concerning the press-work nothing need be said; it is faultless.

G. G. D.

A MANUAL OF MEDICINE. Edited by W. H. ALLCHIN, M.D. Vol. I. General Diseases (*continued*)—Diseases Caused by Parasites; Diseases Determined by Poisons Introduced into the Body; Primary Perversions of General Nutrition; Diseases of the Blood. Vol. III. Diseases of the Nervous System. The Macmillan Co., 1901.

As an additional reference work on Pathology and Practice the present collection of monographs by English authors may be confidently recommended. While it does not pretend to rival in completeness detail the larger systems of medicine, it possesses the advantage of being

confined within limits which justify the title of a manual. The prevailing views on the etiology and pathology of the morbid conditions which at present are engaging the attention of investigators receive their due share of consideration. Thus, in the introductory chapter on Diseases of the Blood, a fairly comprehensive exposition of the actual state of hæmatology will be found, although the portion devoted to clinical laboratory work might with advantage have been more fully elaborated.

The third volume is devoted to Diseases of the Nervous System. It is enriched by numerous differential tables and an elaborate table of spinal localizations, besides numerous explanatory cuts and diagrams scattered through the text. Of practical value is the short article on the method of examining patients suffering with speech defects, especially for one who is not in the habit of seeing many nervous disorders, as is also the chapter on Focal Diagnosis, embracing only twenty pages.

In the article on cerebral vascular lesions the differential points between the various forms of hemorrhage are stated with apparent clearness if somewhat dogmatically, but one's faith in the author's accuracy is shaken by an unfortunate contradiction in the symptomatology. Thus, on page 70, it is stated that "ventricular hemorrhage is indicated by rigidity giving place to flaccidity of the limbs on both sides," while on the opposite page we read that in the same condition "there may coexist rigidity of the limbs on one or both sides." It is to be hoped that the opportunity to correct or explain such discrepancies will be afforded by the appearance of a new edition.

A cursory survey of the most common conditions usually seen by the general practitioner leaves one with the pleasant impression that the authors are not afflicted with the pessimistic views so often displayed by special writers on the subject of treatment. Where the disease is incurable some consideration is at least devoted to palliative measures. The freedom with which the bromides and, above all, the coal tar antipyrhetics are recommended in a great variety of conditions requiring sedative and analgesic medication is to be deprecated. The treatment advised in epilepsy follows the beaten tracks, no mention being made of the recent endeavors to control the disease by dietetic management, thus avoiding the danger of mental deterioration incident to bromide medication. Indeed, the author states that "mental deterioration takes place at least as rapidly in those who are treated by other drugs," thus apparently taking its ultimate occurrence for granted. In fact, throughout the work treatment other than by drugs receives too little attention. The fact that the experience and methods of English physicians are reflected in the book will not detract from its interest to American readers. The style of the work is uniformly excellent, and presents a pleasing variety born of its mixed parentage.

R. M. G.

SURGICAL APPLIED ANATOMY. By FREDERICK TREVES, F.R.C.S. New Edition. Revised by the author, with the assistance of ARTHUR KEITH, M.D., F.R.C.S. In one 12mo. volume of 576 pages, with 80 engravings. Philadelphia and New York: Lea Brothers & Co.

Treves' *Applied Anatomy* has long been prized by medical students for its lucidity, brevity, and, withal, its comprehensiveness. We know of no book between whose covers is compressed so much valuable infor-

mation in such an attractive style. Interwoven with the dry structural details are rich similes, novel cases, many curiosities of surgery, and much reasoning as to why we are contextured thus. This does not burden an anatomical memory for us, but it does establish a process whereby many facts are recollected with but little effort. But not only to the undergraduate has this unique little work proven of great value, but also to the graduate whose familiarity with anatomical data diminishes proportionately with his remoteness from the dissecting-room. Many details are absolutely useless to the practitioner; these are omitted, and only those points directly bearing on practice considered.

In the present edition much new matter has been incorporated; old material has been thoroughly revised and in some portions entirely rewritten. Some of the old illustrations have been eliminated and several new ones added. We note with regret the absence of the relational anatomy of arteries with reference to their ligation. Beginning with the surface markings, a part is studied from without inward. The structures severed in the various operations, together with their dangerous neighbors, are pointed out. Fractures and dislocations are considered in comparatively great detail; indeed, in parts of the book anatomical descriptions are sacrificed to the discussion of surgery. In the text the references are few, and in those that are used, sometimes the journal alone, and often the journal and the year only, are mentioned. There are a few evidences of hurried composition in places and numerous typographical errors.

Treves remarks the little trouble from hemorrhage attending brain operations, a phenomenon not noted in this country. In the same paragraph the measurements of the orbit are given in the English, and those of the eyeball in the French, system. The seton is still recommended for corneal ulceration.

F. T. S.

ATLAS AND EPITOME OF SPECIAL PATHOLOGIC HISTOLOGY. By DOCTOR DR. HERMANN DÜRCK, Assistant in the Pathologic Institute; Professor to the Municipal Hospital L. I. in Munich. Authorized translation from the German. Edited by LUDVIG HEKTOEN, M.D., Professor of Pathology in Rush Medical College, Chicago. Liver; Urinary Organs; Sex Organs; Nervous System; Skin; Muscles; Bones. With 123 colored illustrations and 60 lithographic plates. Philadelphia and London: W. B. Saunders & Co., 1901.

In this the second volume of his *Special Pathologic Histology*, Dürck completes his description of organic lesions, taking up the morphological histology of the liver, urinary organs, sexual organs, nervous system, skin, muscles, and bone. Like its companion volume of the well-known Saunders' *Medical Atlas* series, it partakes more of the nature of an atlas, although the accompanying text is more than explanatory of the illustrations, containing, as it does, short descriptions of the normal histology of the organs, besides more detailed accounts of their abnormal macroscopic and microscopic appearance. The colored illustrations, of which there are 123, are of uniform excellence, and should be of great assistance not only to the student, but also to the trained pathologist as a matter of reference. For the sake of completeness we could wish that

some space and a few plates had been devoted to the neoplasms in their relation to the various organs considered, but this subject has been reserved for the third volume on general pathologic histology, only those tumors occurring solely in connection with definite organs (hypernephroma, adenomyoma uteri, etc.) being described in the present volume. The system diseases of the central nervous system and the diseases of the organs of special sense have also been omitted, but as the latter are usually the subject of separate text-books the loss does not mar the value of the work.

H. H. C.

LA LÉPROSE. Par le DR. DOM SAUTON, Avec planches hors texte et 60 figures en noir et en couleurs. 8vo., pp. 496. Paris: C. Naud, Editeur, 1901.

THE author of this elaborate monograph has devoted ten years to the special study of leprosy, and during that period has visited nearly every region of the earth in which cases of the disease are grouped. He is, evidently, thoroughly equipped for its study, whether in the laboratory or at the bedside. The evidences of close observation, keen reasoning, patient experimentation, and historic research are abundantly present in this comprehensive work. It is divided into fourteen chapters, any one of which might have been issued separately as a monograph, a statement which does not necessarily imply a lack of unity in the work as a whole. It is evident, therefore, that the reviewer must confine his attention to such portions of the book as, in his opinion, possess the greatest general interest. Even with such limitation there is an *embarras de richesses*.

The chapter on the History of the Disease might be read with interest and profit by every physician, whether specially interested in dermatology or not. The first traces of leprosy are shown to exist in Egyptian sculptures of the earliest Pharaonic dynasties, which represent the mutilations of its advanced stages. Moses, who, as our author well remarks, spoke the language of his day and had no intention of writing a treatise on pathology, confounded scabies, psoriasis, tinea, and syphilis under the head of leprosy. A somewhat similar confusion of these affections is found in the works of Hippocrates. The first genuine description of leprosy, under the name of elephantiasis, is found in Galen, *De re medica*, in the first century of our era. The disease was probably imported into America by the Spaniards. Be this as it may, there is documentary evidence of its existence in this country toward the end of the fifteenth century.

It is commonly taken for granted that leprosy was vastly more prevalent in Europe during the middle ages than at the present time, this opinion being largely based upon the numbers of mediæval asylums for lepers. Thus, for example, according to Mathieu Paris, there were 19,000 such asylums in Christendom in the year 1244, and of these, 2000 were in France. When, however, we recall the fact that the diagnosis of leprosy at the period referred to was faulty to such a degree that, in the opinion of Lancereaux, three-fourths of the cases of so-called leprosy were, in reality, cases of syphilis, it becomes apparent that this traditional opinion may well be questioned. Sauton goes much further than this, for he says that to-day "leprosy" would, perhaps, be more

common than it was in the thirteenth century if we included under it scabies and various other dermatoses and syphilis—a mistake which is still made in the extreme Orient.

In the course of this historical chapter Sauton makes a quotation from the *Philonium of Valescus de Tarenta*, and, in so doing, assigns the date of publication of this work to the year 1400. This manifest absurdity might be regarded as a misprint but for the fact that it occurs in two places.

In Chapter III., which treats of the geographical distribution of leprosy, the most interesting section, and the longest, is that relating to the Sandwich Islands, of which Molokai is selected for the sequestration of those affected with the disease. On this island there are two leper villages, called Kalawao and Kalaupapa, the former of which contains about 400 lepers and the latter 700. Kalawao is renowned as the site of the heroic self-sacrifice of Father Damien, whose work is continued by a number of men and women, most of whom belong to religious orders, and none of whom is named by Sauton except Father Wendelin. Notwithstanding the fact that this island is set apart for lepers, whose deportation thither is compulsory, there are, according to Sauton, scattered among the islands of the Hawaiian Archipelago as many lepers as reside in Molokai itself, i. e., between 1000 and 1100. The dwellers in the leper colonies of Molokai might well adopt the celebrated line of Dante,¹ for hope, whether of recovery or return, they must abandon. Sauton tells us that he saw several persons at Molokai who for years had exhibited no symptoms of leprosy.

The chapter on Etiology is divided into four parts, of which the first is devoted to the discussion of the etiological influence of climatic and telluric conditions, alimentation, defective hygiene, sex, race, etc. The conclusion is that any cause, or set of causes, which impairs the resistance of the organism to disease in general renders it an easier prey to the bacillus of Hansen. Perhaps, also, the virulence of this organism is augmented by some of these causes, and especially by overcrowding and defective hygiene in general. An interesting observation, in this connection, is that certain dermatoses, such as scabies, eczema, and prurigo, may open the way to infection through the skin.

The second part of this chapter contains a discussion of the question of heredity in all its aspects. In this connection the reviewer cannot refrain from expressing the opinion that, from a practical stand-point, there is considerable hair-splitting in the definition of heredity. This statement refers particularly to the distinction that is drawn between "conceptional heredity," i. e., heredity through an infected ovum or spermatozoon, and "heredo-contagion," i. e., infection acquired *in utero* after the ovum has undergone a certain degree of development. It seems manifestly impossible to determine whether a child born with the signs of leprosy, or exhibiting them shortly after birth, has been developed from an infected ovum or has been infected *in utero*. It is established that the bacillus of Hansen may be present in the seminal fluid and in the ovum, but it is a mere matter of opinion whether or not an infected ovum is capable of development. Sauton holds the view that conceptional heredity or true heredity, as he calls it, involves sterility, abortion, or non-viability. To the reviewer it seems an ultra-refinement of terms, if not an absurdity, to designate as true heredity ("l'hérédité vraie") a condition which prevents inheritance. The third and fourth

¹ "Lasciate ogni speranza voi ch'entrate."

parts of this chapter deal with the subject of contagion, and in the latter there is a detailed account of fifty-three examples of the communication of the disease.

The chapter on bacteriology occupies seventy pages, and is profusely and beautifully illustrated. The physical properties of the bacillus of Hansen and its color reactions are described in detail. The question whether it can be cultivated is left undecided until the statements of Carasquilla are more thoroughly investigated. This observer, in a communication to the National Academy of Medicine of Bogota, claims success in his attempts at cultivating the organism, and his methods are detailed by Sauton. Whatever different opinions may exist concerning the culture experiments, all authorities seem to agree in the belief that all attempts to inoculate animals, including man, with leprosy have failed. It is true that a Hawaiian criminal who was inoculated by Arning developed leprosy some two years later, but the experiment is inconclusive, inasmuch as the man belonged to a leprous family and lived in an infected neighborhood. Certain points of resemblance between the bacillus of Hansen and the bacillus tuberculosis, as well as the prompt and decided reaction of lepers to the hypodermic injection of tuberculin, conduct Sauton to the conclusion that these two organisms have probably issued from a common stock. The most remarkable fact concerning the bacteriology of leprosy is the occasional disappearance of the bacillus of Hansen from tissues and organs in which it had previously abounded. It is now established by the authority of the International Congress of Berlin of 1897 that the diagnosis of leprosy must occasionally rest solely upon clinical evidence.

Sauton regards the customary clinical division of leprosy into tubercular and anæsthetic varieties as convenient for description, but artificial. It is too diagrammatic. Nevertheless, it is impossible for the writer to pursue his way among the labyrinth of symptoms or to act as a guide to others without some such clew. Our author, in common with other authorities, finds a schematic division indispensable, and adopts the following: The first period is that of infection, characterized by the primary accidents or symptoms. These consist of fever, progressive anemia, headache and vertigo, digestive disorders, coryza, and epistaxis, and various sensory disorders. Among these various symptoms the only one upon which a diagnosis may be based is rhinitis. In the nasal discharge the bacillus of Hansen may frequently be detected.

The secondary period comprises the multiiform tegumentary and mucous lesions, and also sensory disorders, among which the well-known "dissociation symptom," once supposed to be pathognomonic of syringomyelia, may be present.

The tertiary period is characterized by the predominance of tropho-neurotic lesions, which may manifest themselves in the skin, the muscles, and the bones. The extraordinary resemblance of leprosy to certain diseases lately described under the names of syringomyelia, Morvan's disease, anhum, Raynaud's disease, etc., is referred to in Chapter VII., in which the statement is made that the majority of cases exhibited as types of syringomyelia and Morvan's disease have, on closer study, been proved to be cases of leprosy. It is in great part owing to the work of Zambaco Pacha that the leprous character of many of these cases has been recognized.

The foregoing remarks suffice to indicate the nature and scope of this

monograph, which should be in all our medical libraries. It remains to add that the principal object of Dr. Sauton's labors was not to write a classical treatise upon leprosy, but to found in France a sanatorium for lepers. With this end in view he has purchased a tract of land near Neufchâteau (Vosges), upon which, as he tells us, his charitable design would have been executed but for a veritable insurrection among the inhabitants of the neighborhood.

The work is dedicated to the memory of Pasteur, "cet illustre pionnier de la science et de la charité." F. P. H.

THE READY-REFERENCE HANDBOOK OF SKIN DISEASES. By GEORGE THOMAS JACKSON, M.D., Chief of Clinic and Instructor in Dermatology, College of Physicians and Surgeons, New York. New (4th) edition, thoroughly revised. In one 12mo. volume of 617 pages, with 82 engravings and 3 colored plates. Philadelphia and New York: Lea Brothers & Co., 1900.

THE thorough revision to which Dr. Jackson's well-known *Handbook of Skin Diseases* has been subjected brings it fully up to date. The most important recent additions to our knowledge of cutaneous diseases are appropriately mentioned, and the new remedies and methods of treatment are succinctly described. Considerable new matter has been added in the shape of sections on Acne Keratosa, Acne Urticata, Caratée, Craw, Endothelioma, Erythrodermie Pityriasique en Plaques Disséminées, Fordyce's Disease of the Lips, Granuloma Necrotica, Impetigo Bockhardt, Lichen Annularis, Lichen Pilaris, Pityriasis Lichenoides Chronica, and Verruga Peruana. These maladies belong among the less common dermatoses, except Fordyce's Disease of the Lips, which occurs in such a large proportion of persons, if it is not present in some degree in all, that it is rather doubtful if it should be regarded as a diseased condition at all.

The successful application of light and the Röntgen ray to the treatment of lupus vulgaris is briefly mentioned, but the very remarkable results reported in the past year or two following the use of the same measures in epithelioma of the skin are, through some oversight, not even referred to.

The section on Psorospermiosis Follicularis Cutis might very well have been entirely omitted and the space given to some more important subject, since it is now admitted that the affection thus named is not psorospermiosis at all, but belongs to the keratoses—that so far as we know there is no such thing as a cutaneous psorospermiosis.

We regret that some mention has not been made of the deep suppurating forms of trichophytosis which follow infection from the domestic animals. A considerable number of cases of ringworm, in which the deeper parts of the skin were invaded by the trichophyton, causing more or less extensive suppuration, are on record in the dermatologic literature of the past few years, and it is time that some account of this important variety of the disease found its way into the text-books.

Of the new illustrations special mention should be made of the beautiful plates representing the eruption of variola; these leave little to be desired in the way of pictorial illustration.

Upon the whole, this new edition of Dr. Jackson's book well maintains the reputation acquired by its predecessors as a reliable guide in dermatology.

M. B. H.

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Observations on Carcinoma of the Stomach.—BOAS (*Arch. f. Verdauungskrankheiten*, 1901, vii., 413) communicates a number of interesting observations made on 141 cases of carcinoma of the stomach occurring within the last five years. He treats especially of the manner of onset, course, and complications of the disease, as well as of the condition of the intestine, together with some observations concerning the urine.

In thirty-nine, or more than a quarter of all his cases, the symptoms began abruptly. In the words of the author :

"1. The onset of carcinoma of the stomach is by no means always gradual ; it is often wholly acute, with or without slight prodromata scarcely recognizable to the patient or the physician.

"2. Sometimes the onset manifests itself by a single sudden hemorrhage from the mouth or anus occurring during apparently perfect health."

If several hemorrhages follow in rapid succession the differential diagnosis between simple ulcer and *ulcus carcinomatosum* or carcinoma may be very difficult. If the patients are of advanced age one should, under all circumstances, think of carcinoma. Boas observes that about one-third of the cases retain their appetite, or even show an increased desire for food. The condition of the appetite in carcinoma of the stomach may be most variable. With regard to the tongue, the author states : " According to my experience, there can be no question of any recognizable connection between the serious pathological process in the stomach and the tongue." Not infrequently the tongue may be perfectly clean. The decided majority of his cases showed constipation.

Enlargement of the supraclavicular glands on the left side is an extremely rare symptom. It was not present in a single one of the 141 cases. Three instances of secondary carcinoma of the umbilicus are reported. While this

is, as a rule, a late symptom, it may occur early in the course of the disease so as to be the first diagnostic sign.

Albuminuria is a rare symptom; it occurred in but three of Boas' cases. In only one instance was glycosuria found. In five cases there was a complicating pulmonary tuberculosis. The diagnosis of ulcer carcinomatousum is often impossible. If the history points to a previous gastric ulcer, one would naturally suspect its relation to the existing malady, but the proof can only be obtained by autopsy.

Diagnostic Value of Blood Examinations in Gastric Ulcer and Carcinoma.—RENCKI (*Arch. f. Verdauungskrankheiten*, 1901, vii., pp. 234, 235) studied the blood in fifteen cases of carcinoma and fourteen of ulcer of the stomach. The red and white corpuscles were counted, the hæmoglobin estimated, and differential counts made of the leucocytes. No characteristic differences could be made out between the anæmia in carcinoma and ulcer, and the conclusion was reached that the examination of the blood is of relatively little value from a diagnostic point of view.

As a result of further studies in normal and various pathological conditions, in eleven cases of carcinoma ventriculi and in ten of gastric ulcer and benignant pyloric stenosis, the author arrives at the following conclusions:

1. During the digestion of albumin there appears, in the majority of cases, an increase in the number of white blood-corpuscles—a digestive leucocytosis.
2. The climax occurs generally in the third and fourth hours, the average increase in the colorless corpuscles amounting to 3543.
3. Its appearance is connected with the normal function of the pylorus and the intestine.
4. The absence of the digestive leucocytosis is dependent upon pathological conditions of the digestive tract.
5. The presence of an increased number of white corpuscles during digestion is of no value as a diagnostic symptom in cases where there is a question of doubt between new-growth and gastric ulcer, for it may appear in both conditions and is independent of the nature of the disease.
6. The absence of digestive leucocytosis in healthy individuals may depend upon a diminution in the contractile power of the pylorus, which results from its insufficiency.

The Westphal-Piltz Pupil Phenomenon.—SCHANZ (*Berlin. klin. Wochenschrift*, 1901, xxvii., 1065) states that Westphal, in 1899, called attention to a phenomenon present only in cases where the pupillary reflexes were abolished, with moderately or widely dilated pupils, and also in instances where the light reflex was absent, with retention of the convergence reaction. This phenomenon, which he failed to find in normal pupils except in the instance of hysteria, consisted in the fact that when the patient contracts the orbicularis muscle forcibly while the eyelids are held open, the pupil becomes appreciably contracted, while the eyeball turns upward and outward. Westphal believes that in forcible closing of the eyelids the pupillary branch of the oculomotorius and the sphincter iris muscle yet come into play even in pupils where the light reflex is abolished. The contraction of the p

is then an associated movement, and results from the same mechanism as that determining the upward and outward rotation of the eye. At about the same time Piltz made a similar observation, and called attention to the fact that the symptom, while present in a large number of patients with progressive paralysis, tabes, catatonia, epilepsy, dementia præcox, etc., was considerably commoner in progressive paralysis than in tabes, so much so as to be of some value in diagnosis. Galassi, who has observed this symptom previously, believed that in incomplete peripheral paralysis of the oculomotorius, with loss of all pupillary movements, this reaction might yet be brought about by energetic closure of the lids. The symptoms might, he thought, be of value in distinguishing a peripheral from a central paralysis. Antal, in 1900, studied this phenomenon and came to the conclusion that it was present as a prodromal symptom when the light reflex was still retained, as well as a late symptom in pupils with absence of light, accommodation, and convergence reflexes. Parisatti, however, in 1887, called attention to the fact that the mere increase of pressure in an eye might cause this symptom. Schanz has paid especial attention to the phenomenon, and appears to have accumulated proof of this latter view. The phenomenon was clearly demonstrable in one case after the use of atropine and in another instance of complete ophthalmoplegia interna of long duration.

In view of these cases the author believes that the possible action of the paralyzed nerve branch is out of the question, and concludes that the phenomenon is purely mechanical, due to an engorgement of the vessels of the iris from pressure. This phenomenon is probably masked in the normal eye by the light reflex. It is natural, therefore, that it should be observed only in pupils which are not wholly normal, at best when moderately dilated. In the absence of light reflex it may be missed owing to the narrowness of the pupil; this may well account for the fact that Piltz found the symptom less frequently in tabetics than in paralytics. The completeness of the reaction may depend upon various movements: the power of the orbicularis, the position of the eyeball, the hardness of the sclera, etc. The author has observed the phenomenon, also, in cocaineized eyes. He concludes: "According to my opinion, this phenomenon is purely mechanical, and the only conclusion which can be drawn from it is that the innervation of the pupil is not wholly normal, for otherwise the light reflex would hide this mechanical narrowing. All further conclusions which rest upon the idea that it is an associated movement similar to the rolling upward of the eyeball on closing of the eyelids appear to me erroneous."

Primary Cancer of the Bile-ducts.—DEVIC and GALLAVARDIN (*Revue de Médecine*, July, August, and October, 1901, pp. 557, 658, and 839) report a case of primary carcinoma of the common bile-duct and give an exhaustive analysis of a total of 55 cases of primary cancer involving various portions of the bile passages, which they have collected from the literature. Primary cancer of the gall-bladder and of the ampulla of Vater are not included. Only those cases in which the hepatic duct and its branches, the cystic duct and the common duct (exclusive of the ampulla) are involved are included in the series.

From the fact that they have been able to find only 55 cases, they conclude

that the condition is rare. In 46 cases in which the sex was mentioned there were 30 males and 16 females. This is entirely contrary to what is the case in cancer of the gall-bladder, where from 75 to 80 per cent. are women. Gallstones are less frequently mentioned as a probable cause than in cancer of the gall-bladder. The largest number of cases occur between fifty and sixty-six years of age. In 53 of the cases the distribution of growth was as follows: The common duct was involved in 22; the junction of the hepatic, cystic, and common ducts in 15; and the hepatic duct and its branches in 16. In about one-fifth of the cases metastases occurred, though being practically confined to the liver.

The condition of the gall-bladder as regards dilatation was of interest. Of 18 of the cases of cancer of the common duct in which reference to the gall-bladder was made, it was dilated in 17 and normal in 1; of 14 of the cases where the growth was at the junction of the common, hepatic, and cystic ducts, the gall-bladder was dilated in 7, normal in 3, and contracted in 4. In only 7 of the 16 cases of cancer of the hepatic duct and its branches was reference made to the condition of the gall-bladder. It was dilated in 2, normal in 1, and contracted in 4.

In the majority of the cases the liver was enlarged. Of 37 cases in which its condition was noted it was hypertrophied in 25, and either atrophied or normal in 12. Primary cancer of the bile-ducts is characterized by a chronic jaundice which is insidious in its onset and of gradually increasing intensity. Digestive disturbances occur. Pain is not a prominent symptom. Progressive emaciation and a decided cachexia precede death, which occurs usually in from two to six months. The examination of the abdomen practically never reveals any tumor. The spleen may be increased in volume. Ascites occurs in about half of the cases.

The writers divide the cases clinically into four groups, as follows:

1. The usual type, with hypertrophied liver and enlarged gall-bladder, comprising almost all of the cases of cancer of the common duct, and some of the cases with involvement of the hepatic duct.
2. The atypical form with or without hypertrophy of the liver, and atrophy of the gall-bladder, found chiefly with cancer of the hepatic duct.
3. A form simulating hypertrophic biliary cirrhosis, owing to the enlargement of the liver and spleen.
4. A form without jaundice. This is extremely rare, only one case having been reported.

Diagnosis is not easy and it has to be differentiated from hypertrophic biliary cirrhosis, obstruction of the common duct by a calculus, and from obstruction of the common duct by an adjacent cancerous gland or a cancer of the ampulla of Vater or cancer in the head of the pancreas.

The treatment is chiefly surgical, and then merely palliative in the form of a cholecystostomy or cholecystenterostomy.

Cyto-diagnosis and Tuberculous Meningitis. — MARCOU-MUTZMANN (*Archives Générales de Médecine*, September, 1901, p. 345) reports a case of tuberculous meningitis, in which he studied the character of the cells in the cerebro-spinal fluid, the result leading him to cast doubt on the value of "cyto-diagnosis" in the differentiation of various forms of meningitis.

Widal, Sicard, and Ravaut were the first to draw attention to the possibility of making a diagnosis of the form of meningitis from the morphology of the cells contained in the cerebro-spinal fluid. Subsequently it was believed possible to determine the cause of the development of pleural, peritoneal, and joint fluids from the character of the prevailing cell present in these exudates.

Milian has recently formulated the results obtained by the study of the cells in the various fluids as follows :

1. If the prevailing cell in the cerebro-spinal, joint, or hydrocele fluids be the mononuclear lymphocyte, the inflammatory process is tuberculous in origin.

2. If, on the other hand, the polymorphonuclear leucocyte be the prevailing cell, the condition is due to some other organism than the tubercle bacillus.

3. In the case of pleural and peritoneal exudates, as well as of hydrocele fluid, if the predominant cell be an endothelial cell, the fluid is the result of some mechanical disturbance, such as cardiac or renal disease.

Marcou-Mutzner's case was a man forty years of age, who had been gradually losing weight for several months. For two months previous to admission to the Charité Hospital, he had suffered from a right-sided sciatica, for which he sought treatment. There were signs of local consolidation at the apex of the right lung. During the patient's stay in the hospital he developed definite signs of meningitis, including Kernig's sign. Lumbar puncture was performed, and the cerebro-spinal fluid, which was fairly clear, was centrifugalized, and the sediment stained and examined microscopically. The prevailing cell was found to be the polymorphonuclear leucocyte. This occasioned surprise, as the case had previously been regarded as one of tuberculous meningitis. As a result of the examination of the spinal fluid, however, the diagnosis was changed to that of acute meningitis of non-tuberculous origin.

The patient died three days later, and an autopsy was performed. He was found to have typical pulmonary and meningeal tuberculosis.

The writer cites another case reported by Rendu, in which the patient was admitted to the hospital comatose and aphasic. The examination of the cerebro-spinal fluid showed that the lymphocyte predominated. A diagnosis of tuberculous meningitis was made. The autopsy eventually showed a fracture of the base of the skull.

These cases cause Marcou-Mutzner to doubt the value of the study of the cells in the cerebro-spinal fluid in meningitis, and he concludes that it is possible to have an excess of the polymorphonuclear leucocytes in the spinal fluid in tuberculous meningitis, and also to have an excess of the lymphocytes without the latter disease being present.

Results Obtained by Antityphoid Inoculation in the Case of an Epidemic of Typhoid Fever which Occurred in the Richmond Asylum, Dublin.—WRIGHT (*The Lancet*, October 26, 1901, p. 1107) gives the results obtained from the inoculation with antityphoid vaccine of the inmates of the Richmond Asylum during the prevalence of an epidemic of typhoid fever in that institution. The first case of the epidemic came under obser-

vation on August 7, 1900, and from that date on to the end of December cases of typhoid fever occurred among the patients and the nursing staff. No reference is made to the probable source of the infection. Wright applied to for a supply of antityphoid vaccine. The first batch of inmates was inoculated on September 6th, 0.75 cubic centimetre of vaccine being used at each inoculation. By November 30th, after a series of seven sittings, a total of 511 persons had been inoculated. Excluding patients above fifty-five years of age as insusceptible, Mr. Cullinan, who made the inoculations, estimated that the total number of susceptible persons exposed to infection on the date on which the inoculations were begun was 655. Of these, 541 were insane inmates, the balance of 114 being nurses. Of the 541 inmates, 511 were ultimately inoculated. None of the 114 nurses received the protective serum. According to Mr. Cullinan's report, 29 and 7 cases of typhoid fever, respectively, occurred in the uninoculated and inoculated groups after the commencement of the treatment on September 6th. Wright claims that 2 of the 7 cases must have contracted the infection before the inoculations were made, thus leaving only 5 cases among the inoculated. He does not consider that the uninoculated nurses were more exposed to infection than the inoculated inmates. A further comparison is made by considering the number of cases that developed in the two groups after the completion of the inoculations. Thus in the uninoculated persons 5 cases developed, whereas in 504 inoculated inmates (excluding the outside limit of 7 cases that previously developed the disease) no cases occurred. Wright thinks that these statistics prove the undoubted protective power of the vaccine, and says they agree well with the results which have reached him from other sources.

On the Presence of Micro-organisms in the Circulation during Pneumonia.—PROCHASKA (*Deutsch. Arch. f. klin. Med.*, Band lxx. p. 559), continuing his early observations in this field (*Centralbl. f. Gen. Med.*, 1900, No. 1)—in the clinic of Prof. Eichhorst—has examined bacteriologically the blood of forty consecutive cases of pneumonia. The blood, nearly 10 c.c. amount, was collected by a sterilized syringe from one of the arm veins after an elastic constriction and thorough cleaning of the skin. Control experiments with normal individuals proved the reliability of this method; in no instance were contaminations obtained.

Forty cases were examined, of which seven were fatal. In thirty-eight instances the pneumococcus was obtained. Twice there developed organisms, concerning the nature of which the author is uncertain. These were streptococci, with a tendency to arrangement in pairs; they may have been an especially virulent variety of pneumococcus. One of these cases ended fatally; in the second slow resolution was followed by a secondary indurative process.

The cultures were made at varying periods of the disease, sometimes very early as the second day. In two cases positive results were obtained on the day following the crisis; in one of these there developed later a muscular abscess. In one case, followed by an empyema, pneumococci were found in the circulation on the second day after the crisis. In another instance with slow resolution, organisms were found three days after the febrile crisis.

Prochaska's observations are not sufficient to justify comment on the suggestion of Paesler that the number of organisms in the circulation and the degree of leucocytosis may bear an inverse relation one to another.

The author believes that his positive results are due in part to the large quantities of blood used, and in part to the use of bouillon as a culture medium.

Adding to these cases those mentioned in his previous publication, Prochaska is able to report fifty cases of pneumonia in which septicæmia has been regularly demonstrated; and only twelve of these cases resulted fatally. The number of organisms in the circulation varied greatly in different instances, having apparently no relation to the severity of the case. These results appear to justify the conclusion that pneumococci are always present in the blood during pneumonia. The demonstration of this fact affords a ready explanation of the frequent metastatic complications of pneumonia due to *diplococcus lanceolatus*, occurring, as they not infrequently do, in mild as well as in severe cases.

SURGERY.

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Three Cases of Pulmonary Abscess, with Operation.—SMITH (*Yale Medical Journal*, February, 1901) states that the indications for the operation of pneumotomy are: tuberculous cavities in exceptional cases, where the disease is confined to the lung; bronchiectasis, hydatid cyst, gangrene, abscess, foreign bodies, and hemorrhage. The results of operations for tubercular and bronchiectatic cavities are not especially encouraging. With the former, tubercular disease is left after the operation has been performed and new cavities will probably occur. With the latter, temporary relief or improvement is all that can usually be expected, for there will be other smaller cavities that will burrow and enlarge. Hydatid cyst of the lung is best treated by pulmonary incision, and the results are very encouraging. Reclus reports eleven operations with but two deaths. Gangrene should be treated by incision, if the disease is not diffused and yet is causing symptoms of septicæmia. Statistics prove that over 50 per cent. of these hitherto fatal cases can be saved by early and efficient operation. Abscess of the lung, which is not being drained through the bronchi, or if being drained is wearing the patient out by prolonged fits of coughing, or where the evidences of septic absorption are pronounced, should be opened and thoroughly drained, unless some strong contraindication exists. Injuries to and foreign bodies

in the lung may call for operation, especially when inflammatory action and the formation of abscess have occurred. Hemorrhage of the lung from traumatism, which cannot be controlled by ordinary methods, may possibly be checked by incision and ligation—an operation that has been but rarely done. Gangrene is diagnosed by the history, the physical signs, severe constitutional symptoms, together with the fetid breath and characteristic sputum, which is greenish-brown and contains elastic fibres arranged in an alveolar manner, and having an unequalled odor. The diagnosis of abscess of the lung is usually not difficult. There are the physical signs of dullness, increased vocal resonance and fremitus, and frequently the sound of fluid passing through a cavity filled with fluid. The sputum is very offensive and is usually of a brownish color. The microscope shows septic micrococci, but not of lung tissue, with crystals of fat and hæmatoidin. In acute cases the constitutional symptoms are usually severe, septic absorption commencing early. This is probably accounted for by the inefficient walling off of the abscesses in the lung tissue. The exploring needle will sometimes find pus but may fail even in cases where it is present. It may serve the purpose of establishing whether the pleural surfaces are adherent, for if so the needle will oscillate but slightly when introduced, but if not adherent it will oscillate synchronously with respiration. Among the dangers and contraindications of the operation are that there must always be some danger from an anæsthetic given at a time when the patient is seriously ill. Chloroform is probably the best anæsthetic in most cases, as it provokes less irritation of the respiratory organs than ether. Many surgeons advise against operation when the pleural surfaces are found free, but the author believes that there is better hope of success in operating at once than in waiting for adhesions to follow a primary operation, for in no one of the author's cases was a patient likely to have survived unaided that length of time. Septic infection in all cases must be scrupulously guarded against. There is but little danger from hemorrhage, and if there be any it can usually be easily controlled. The best incision is one following the curve of the ribs from 6 to 12 cm. in length over the seat of the disease. This incision should be made over the centre of a rib, if one is to be resected; if not, the opening is to be made in the intercostal space midway between the ribs. The intercostal muscles are divided and the costal pleura examined. If a rib is resected from 3 to 4 cm. should be removed by the curved bone forceps or the Gigli saw, after separating the periosteum by means of a periosteal elevator. It should next be determined if the pleural surfaces are adherent. The edges of the wound are held apart by retractors and an aspirating needle or trocar and canula should be introduced. If pus is found and the pleura are adherent a blunt instrument or the finger should be carried into the lung in the track of the aspirating needle. If they are not adherent one must decide between the dangers of an immediate operation and possible infection of the pleural cavity, and the dangers of postponing further operative interference until adhesions are formed or the abscess ruptures. The author has had good results with irrigation in those cases where there was an offensive discharge. If the pus is not offensive and there is good drainage irrigation is not necessary. The future of this operation should be one of progress. There should be more thorough understanding of its applications, its limits

tions, and a readiness on the part of practitioners to give their patients the benefit of it when necessity arises.

The Surgery of the Spleen.—WARREN (*Annals of Surgery*, May, 1901) states that splenectomy is no new operation, and that the number of operations performed in modern times is by no means small. Hagen has collected 360 cases, with a mortality of 38.3 per cent. After carefully revising his list and eliminating what would be considered incurable cases and cases in which the diagnosis has not been sufficiently established, Hagen was able to reduce this mortality to 12.2 per cent. (1) Malarial spleen. Hagen has collected 88 cases of malarial hypertrophy of the spleen, exclusive of wandering spleen. Of these cases, 24 previous to the year 1890 gave a mortality of 62.5 per cent., while 64 cases operated after the year 1890 gave a mortality of 23.4 per cent. When one considers the very large size that the organ often attains in this disease, and the unfavorable constitutional condition of the patient, such results are at least encouraging. (2) Splenic anæmia or splenic pseudoleukæmia. Sippy reports 7 cases in which splenectomy was performed, with 5 recoveries. Osler has reported 15 cases, 1 of which was operated upon, with a fatal result. Warren reports 1 successful case. (3) Splenic leukæmia. Splenectomy in this disease is almost invariably followed by a fatal result. Hagen reports 42 cases, with only 4 recoveries. Death was almost, without exception, due to secondary hemorrhage from the surface of the wound, owing to the condition of the walls of the large vessels. Richardson has had one successful case. (4) Chronic enlargement of the spleen in infancy is generally secondary to some other disease, and yields rapidly to treatment appropriate to the causal condition. (5) Banti's disease, or hypertrophy, with cirrhosis of the liver. Hagen reports 16 cases in which splenectomy was performed, with only 3 deaths. (6) Wandering spleen. The number of cases operated upon up to the last decade has been comparatively few, but during this decade 11 reported cases, with 4 deaths, giving a mortality of 36.3 per cent. On the other hand, of 43 cases of wandering spleen operated upon during the last decade there were only 3 deaths. It would seem, therefore, that as a prophylactic measure extirpation of the wandering spleen was a justifiable procedure, and that we should certainly be within the limits of propriety in advising an operation where the displacement had reached a degree to produce marked abdominal symptoms. Splenoplexy does not seem to meet with approval, as the result is uncertain. (7) Abscess of the spleen. Only certain forms of abscess of the spleen are suitable for splenectomy. Such cases are those in which the spleen itself is surrounded with pus, or cases in which the spleen, containing an abscess, is not too tightly bound down to the abdominal wall by adhesions. Where there is danger of infecting the general peritoneal cavity by an attempt to extirpate the spleen it would be better to simply open the abscess and drain. Hagen has collected 7 cases of abscess, all of which recovered after splenectomy. In 1 of these cases the abscess followed appendicitis. The same author has also reported 3 cases of splenectomy for tuberculosis of the spleen, with 2 recoveries. (8) Rupture of the spleen. Pitts reports 3 successful cases of splenectomy for rupture. Sevor reports a successful case. Since 1890, 34 cases of splenectomy for rupture of the spleen have been reported, with a mortality of 41.2 per

cent. (9) Sarcoma of the spleen. Hagen, up to the year 1890, has reported 5 cases of splenectomy for sarcoma, of which 2 died and the remaining recovered. From 1891 to 1900, 4 cases have been reported, with only 1 death. After reviewing at length the technique and sequelæ of splenectomy Warren concludes with the report of 5 cases: (1) Splenic anæmia, splenectomy; recovery. (2) Sarcoma of the spleen, splenectomy; death. (3) Splenic hæmæmia, splenectomy; recovery. (4) Rupture of an infected spleen, splenectomy; death. (5) Splenopexy, recovery.

Circumcision as a Preventive of Syphilis and Other Disorders.—FRANKLAND (*Lancet*, London, December 29, 1900) states that if it were possible to secure the efficient circumcision of every male in infancy not only would many of the disorders incidental to the genito-urinary organs in childhood, adolescence, and adult life be prevented, but also the frequency of syphilis would be materially diminished. It is necessary to adduce good evidence, not only that the results justify the operation, but also that the operation is justifiable. Therefore, it is necessary to show that the operation is free from risk, that the deprivation of the structure which it is proposed to remove will not inflict any physical disability on the individual, and that the benefits which are likely to accrue are substantial and commensurate with the sacrifice. Everyone has agreed that the operation of circumcision in a healthy child by a competent surgeon is attended with such infinitesimal risk that for all practical purposes it may be considered safe, and that from a sanitary point of view the removal of the prepuce is a decided advantage and does not in any way interfere with the physical welfare of the individual, but instead is a distinctive advantage. It is the prevalent opinion that syphilis is less common among the Jews, but actual statistics are few. Hutchinson analyzed 330 cases of venereal diseases among hospital patients, 58 of whom were Jews, 47 of these had gonorrhœa and 11 had syphilis. The author states that his own experience completely bears out the idea that syphilis is much less prevalent among those who are circumcised. Statistics collected by Fournier, Clerc, and Hill show that in 898 cases of initial lesion 659, or 73.3 per cent., appeared on the prepuce or just behind the corona. Probably the veriest enthusiast would hardly claim that the whole of this 73.3 per cent. of chancres could be prevented by circumcision. One may conclude, however, that the universal practice of circumcision should reduce the case incidence of genital syphilis in the male by something between 25 per cent. and 73 per cent., or, taking the mean of these, by 50 per cent. In syphilis it is not only the guilty who suffer, but the innocent suffer with the guilty, and the sins of the fathers are in very truth visited upon the children. There is direct evidence to show that the practice of circumcision will materially diminish this massacre of the innocent. Hutchinson found that out of 97 female hospital patients that came under observation in one year for venereal disease, 92 were Christians and 5 were Jews; of the former 6 were married, and at least two-thirds of these apparently contracted syphilis from their husbands. The same observer found that out of 252 children under five years of age who came under his care in the course of a year there were 179 Christians giving 27 cases of genital syphilis, and 73 Jews giving only 3 cases of congenital syphilis.

These statistics clearly show an enormous reduction in the case incidence of syphilis among the women and children of the circumcised Jews, and in view of what has already been said the only deduction which can reasonably be drawn from their study is that if the practice of circumcision were to become as general among Christians as it is among Jews a proportionately great reduction of the case incidence of syphilis would take place among their women and children, and this amply proves that the end justifies the means.

PEDIATRICS.

UNDER THE CHARGE OF

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Intra-uterine Rickets.—F. C. ABBOTT, of London (*British Medical Journal*, September 7, 1901, p. 597), describes under this title the physical appearance and post-mortem findings in a female child first seen by him a few weeks before death, at the age of fifteen months. The child was one of twins, the last children of healthy parents. The mother altogether had had seven living children, as well as eight miscarriages; these included three pairs of twins, one of each of the two previous pairs had, however, been a "false conception" and born dead. The living children were all well. The last pair of twins, one of whom was the patient, were, in the mother's opinion, three weeks premature. She was well when carrying them, and knew of nothing abnormal in her diet during the pregnancy. The mother was certain that the baby's legs were at birth quite the same as at the time of observation.

At the time of death the child showed marked rachitic changes in the trunk and abnormal curvatures of the limbs. Many of these curves were the ones commonly associated with rickets and usually explained as being due to muscular tractions, or to methods of progression and support of the body weight. It is interesting and suggestive, however, in the author's opinion, that many of these curves could be produced entirely by intra-uterine pressure, such as the longitudinal chest groove for the arms to rest in, and the curve of the femora against the trunk. This latter was exactly the same curve as was noted in Makin's case (a baby fourteen days old). Some of the curves, on the other hand, were quite unusual, and could only be explained by the intra-uterine folding of the child. These were the curve of the lower end of the forearm with its convexity forward and inward, and the curve of the right leg, with its convexity forward and inward, and a curve of the right leg, with its concavity forward and outward, across which concavity the left leg lay. These unusual curves were only satisfactorily explained by replacing the child in its foetal posture.

The author considers the case the most conclusive yet published from the point of view of the curves being due to intra-uterine pressure. Similar cases have been reported by Barlow and by Makin.

In discussing this case ASHBY referred to the case of an infant brought to the hospital when fourteen days old with five fractures of the long bones. There was also marked craniotabes and typical deformed rickets characteristic. When seen nine months after, the infant had two teeth and there was no sign of rickets. Excluding cases of achondroplasia, there was a fair number of cases recorded of so-called intra-uterine rickets; but, as far as he knew, very few had been examined pathologically shortly after birth. He had some doubts as to whether such cases were true rickets. He thought possibly they were really cases of osteoporosis, the result of malnutrition during intra-uterine life. They resembled the cases of osteoporosis found in pups fed on food in which there was a deficient amount of lime-salts.

Symptoms of Typhoid Fever in Infancy and Childhood.—J. P. CROZIER GRIFFITH (*Journal of the American Medical Association*, 1901, vol. xxxv, No. 7) very succinctly sums up the symptomatology of typhoid fever in young patients in the following sentences:

Most cases of this disease, when affecting the very young, are of the ambulatory type. The child is hardly indisposed, although anorexia and headache are sometimes noted. In a minority of patients vomiting may usher in the disease and fever may be high at the outset.

The mortality of typhoid in childhood is not far from 3 per cent., and, roughly speaking, the younger the child the better the prognosis. Thus the mortality is less in the first than in the second quinquennium. As in adults the roseola is commonly but not universally present. In some cases the rash is very abundant, covering the whole integument.

Enlargement of the spleen is doubtless constantly present, although it is always discoverable. Epistaxis is often present, although exact statistics are wanting. The course of the disease is distinctly shorter in the young than in the adult, and the average duration is about seventeen days.

Diarrhœa is much less frequently present in the child, while, on the other hand, vomiting, rare in the adult, is of common occurrence, not only as an initial phenomenon, but throughout the disease, and it has even been seen as a terminal symptom in cases ending fatally. Tympanites and hemorrhages are of rare occurrence in the child, and the same may be said of perforation. The nervous phenomena are not marked in childhood.

Finally, as a point of great practical significance, it should be stated that the onset of typhoid in the child may simulate meningitis to a more characteristic extent than in the adult.

Observations upon Milk Coagulation and Digestion.—FRANKLIN WHITE (*Journal of the Boston Society of Medical Science*, 1901) has made some important observations as to the value of various methods of rendering the proteids of cow's milk more readily digestible. The scope of his inquiry covered the following questions: First, whether or not cereal decoctions render the milk curd more fine and soft than a simple dilution with water; second, whether one cereal is better than another for this purpose; third,

whether or not the property of yielding a fine curd is wholly due to the starch in solution, and, if so, what per cent. of starch is desirable; fourth, what is the value of certain modifications of the method; and fifth, what is the effect of lime-water and albumin-water upon the casein curd.

The conclusions of his study are summarized as follows:

1. Dilution of milk with cereal decoctions of proper strength renders the casein curd much more fine, soft, and digestible than simple dilution with water. There is no difference in the action of various cereals, such as barley, oats, rice, and wheat.
2. The above property is due mainly, if not wholly, to the starch in solution. The most desirable amount of starch in the milk mixture for practical use is approximately $\frac{1}{4}$ per cent.
3. Diastase, by converting the starch into dextrine and maltose, promptly lessens and removes the action of cereal waters upon casein. Its addition, therefore, is not a practical measure when the action upon the curd is desired.
4. Albumin-water has no practical value as a diluent of milk.
5. Lime-water added to milk has no more effect than water upon the character of the curd produced in the animal stomach.

The Pathogeny of Night-terrors.—REY (*Revue mensuelle des Maladies de l'Enfance*, May, 1901, p. 217) reaffirms the conclusions of his earlier studies on this subject (*Jahrbuch für Kinderheilkunde*, N. F., Bd. xlv.). The views of various authorities as to the etiology of night-terrors in children are by no means in accord. Some consider these attacks as a symptom of another disease, as of an intestinal affection, for example; others believe that they constitute a disease sui generis, an idiopathic affection of the brain; while yet a third class hold that there are two forms, one primitive, another secondary. Rey maintains that the phenomenon is developed and is always determined by an obstacle to respiration and hæmotosis, which is either direct or of reflex origin. Both forms are simply the effect of a slow and prolonged intoxication by carbonic acid. In the attack all the symptoms of a slow poisoning by carbonic acid are reproduced—suffocation, loss of consciousness, with hallucinations, followed by amnesia after recovery, and enfeeblement of muscular motility. The slow onset of this intoxication explains why the attacks never commence until three or four hours after the child has gone to sleep. Another state which results from the same cause, but exhibits symptoms of less intensity, is the nightmare of adults. The child does not raise himself quickly, but in a maladroit fashion, and cries without being able to articulate intelligible words; the tongue is rigid, and its movements are made with difficulty; when taken up in the mother's arms the child's limbs seem heavy and are moved very slowly against the body. All these signs are the result of a paresis of the muscles. As soon as he can speak he seems oblivious of what is passing on about him; he does not speak of anything but his hallucinations, and after a certain time which suffices to supply the want of oxygen and to purify the blood of its surcharge of carbonic acid, he asks simply to be put back in bed to continue his interrupted slumber.

According to the author's observation the susceptibility to night-terrors is not increased by the nervousity of the child; but the tendency seems to be

in almost direct proportion to the intensity of obstruction to nasal spiration. Children with adenoid vegetations are almost always restless during sleep. They turn and twist constantly during the night, and very often suffer from night-terrors. The same condition occurs when nasal respiration is interfered with by other obstacles, such as a tumor, a foreign body, or even a simple rhinitis. A confirmatory circumstance is that adenoid vegetations are particularly frequent at an age at which night-terrors are most frequent—that is, from three to ten years. From ten years to fifteen years both diminish in frequency. Moreover, both nocturnal pavor and adenoid growths are more frequently encountered in girls than in boys.

Carbonic-acid intoxication also produces the fright and hallucinations observed with children during the fitful slumber of an attack of pneumonia, pleurisy, angina, or bronchitis. In children who are subject to night-terrors even the slightest obstruction to nasal breathing suffices to precipitate an attack, a coryza or the mere presence of crusts obstructing the already narrowed passages being sufficient to cause it.

In the cases produced by intestinal disturbance, an overloaded stomach or intestinal worms, it is difficult at first sight to explain a carbonic-acid intoxication. But here it suffices to remember that even according to Silbermann, who considers the affection as a disease *sui generis*, symptomatic night-terrors would be a reflex neurosis of the termination of the pulmonic vagus starting from the gastric terminals, a reflex which produces dyspnoea and sensation of suffocation. Both forms of the affection, therefore, result from the same cause, and there is no ground for distinguishing an idiopathic and a symptomatic form, since both are symptomatic of a carbonic-acid intoxication, direct in one case, indirect by reflex action in the other.

THERAPEUTICS.

UNDER THE CHARGE OF

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Hydrophobia and the Pasteur Methods.—DR. C. W. DULLES speaks of the early trumpeting of the Pasteur method advocates and the development of institutes for the treatment of the disease. He makes a strong and logical arraignment of the early figures, showing that they were founded on the most flimsy sort of grounds, and from an analysis of recent statistics shows that the mortality from hydrophobia in France, with its institutes, was thirty times as great as that in Germany, without such treatment methods, the ratio of affected dogs being, of course, included in this comparison.

France the number of deaths is stated to be greater after the employment of the Pasteur methods than before, the number of affected dogs being considered. He criticises the report of Salmon, of the United States Government, for adopting as facts faulty statistics, and basing upon them conclusions which will only mislead and cause unnecessary death. The author advocates a dog-muzzling law, the use of silver nitrate as a caustic, and the employment of narcotics and restraint in the attack.—*Medical Record*, 1901, vol. lx, p. 41.

Massage in Sciatica.—DR. R. JUDICA advocates the abandonment of all ointments, pills, embrocations, opiates, etc., in the treatment of this condition, and recommends almost unqualifiedly the use of persistent massage. This may follow one of three methods, or all three may be employed. He mentions: 1. Schreider's method, which aims to render the limb temporarily bloodless, at the same time stretching the sciatic nerve by both active and passive means. These procedures are considered not advisable. 2. Simple massage by slow, passive movements, which, as the pain may permit, become more extensive and complete. A rest of a few moments is allowed between each movement. 3. Negro's method, which is strongly recommended. In this the patient is made to lie upon his stomach and face, the gluteal muscles being distinctly relaxed. The thumb is placed on the sciatic notch, and, with the other thumb superimposed, strong pressure is brought to bear from fifteen to twenty seconds on the nerve. Some slightly lateral and rotatory movement of the thumb is permitted during this time, but it should not be raised. This treatment is repeated after five minutes. If this is carried out on alternate days for six or seven times good results are to be expected. The author has had excellent success even in long-standing and chronic cases.—*Gazzetti degli Ospedale*, 1901, vol. xxii., p. 860.

Neuritis Treated by Currents of High Frequency.—DRS. A. DENOYÈS and H. LAGRIFFOUL report on the treatment of an arsenical and a post-influenzal neuritis by D'Arsonval's high-frequency currents. The results were very satisfactory.—*Archives d'Electricité médicales*, 1901, No. 7, p. 281.

Treatment of Chronic Hydrocephalus.—DRS. WILLIAM EWART and W. LEE DICKINSON advocate the process of "artificial pneumocephalus" for the treatment of this condition as suggestive of what might be done for it. The method is worthy of recording. They tap the ventricles with a fine trocar; the intracranial pressure is relieved by the escape of a certain amount of fluid, and they substitute for it aseptic air in such a manner as to avoid the symptoms so commonly noted after complete removal of fluid. Their provisional conclusions are of interest: 1. With due precautions the fluid of chronic hydrocephalus may be completely evacuated from the yet unclosed skull of infants, and aseptic air may be allowed to take its place. This operation may be repeated without detriment and with scarcely more risk than belongs to the usual method of paracentesis. 2. In unfavorable cases of moderate effusion a single operation may suffice. Continual oozing from the puncture for a few days after the removal of the tube is not unfavorable. 3. In cases of considerable effusion an obvious

indication is to relieve the brain from the weight and from the pressure of the fluid. The evacuation is facilitated by the introduction of aseptic air. By a timely repetition of the operation a hydrocephalic infant might be enabled to carry the weight of the head, and, if the treatment were begun sufficiently early, permanent damage to the brain tissue might be avoided and a normal development might ensue. 4. In large heads while hydrocephalus persists a considerable splashing sound is readily obtained. There is obvious risk in eliciting this sound by forcible succussion, and for the same reason any abrupt movement of the head should be avoided.—*British Medical Journal*, 1901, No. 2123, p. 602.

Intramuscular Injections of Calomel in Gonorrhoeal Arthritis.—DR. M. THOMAS, adopting the method advocated by Grosse, has treated a number of patients suffering from gonorrhoeal arthritis by intramuscular injections of calomel. Doses of half a grain were injected at intervals of from two to four days. The results are reported as specific, but one patient had a return of his arthritis and another developed stomatitis.—*Le Mois Thérapeutique*, 1901, vol. ii., p. 59.

Value of Phosphorus Therapy in Rhachitis.—DR. A. MONTI says that the recent discussions concerning the use of phosphorus in rhachitis contain no new points of view. Both Hahnemann and Trousseau recommended, and later rejected it. He is of the opinion that this drug is of little or no value in such condition, and maintains that the early experimental studies were not free from error, and that the results obtained in the hands of clinicians are unsatisfactory and open to doubt.—*Wiener klinische Wochenschrift*, 1901, vol. xiv., p. 180.

Sidonal as an Aid to Uric-acid Excretion.—DR. M. G. BARDET reports on the use of this drug, re-introduced into therapeutics in 1899. It is piperazine quinate, first prepared in 1868 by Lanthemann, and employed as a therapeutic agent by Rabuteau in 1872. It decomposes in the body into quinone and then into benzoic acid, and is supposed to form hippuric acid by combining with uric acid—a chemical impossibility, according to Rabuteau. Clinically, certain facts seem to point to a certain amount of efficiency. Bardet had under observation three patients afflicted with mild gout, to whom daily doses of from forty-five to seventy-five grains were given. After a month of medication the patients had improved, and weekly examinations of the urine seemed to show a diminution in the amount of uric acid eliminated. The results were not startling enough to encourage him to proceed further.—*Le Mois Thérapeutique*, 1901, vol. ii., p. 59.

Means of Arresting Acute Endocarditis.—DR. RICHARD CATON advocates three procedures for the treatment of this condition—physiological rest, stimulation of trophic centres, and the administration of sodium iodide. The rest should be absolute, the patient remaining quietly in bed for a period of at least six weeks. The head should be low; sleep is encouraged, pain prevented, and all mental excitement avoided; the diet should be light and stimulating. Salicylates, the author holds, are of more value for

the joint condition than they are for the endocardium or myocardium. They should be employed, however. A second step in the treatment is by means of small local blisters. Their effect is due entirely to the stimulation of the vasomotor and trophic nerves. He applies in succession a number of small blisters, an inch in diameter, to the wall of the chest between the clavicle and the nipple. Finally, to aid in the absorption of effused material, sodium iodide is advocated.—*British Medical Journal*, 1901, No. 2128, p. 1049.

Schneiderlin's Anæsthesia.—DR. B. KORFF reports on this author's recent contribution to the subject of a new method in anæsthesia whose object was to obtain a non-dangerous continuous anæsthetic that did not worry the patient with excessive manipulations and cumbersome apparatus, and was free from the unpleasant after-effects of vomiting, coughing, etc. Schneiderlin believed that in a mixture of hyoscine hydrobromate, one two-hundredth of a grain, and morphine sulphate, one-seventh of a grain, he had obtained this ideal combination. He advises this dosage twice hypodermically at an interval of from one to two hours. Korff has modified this method as follows: He injects one one-hundred and sixtieth of a grain of hyoscine and one-sixth of a grain of morphine twice at an interval of two hours, and then applies a few drops of chloroform in a mask until complete anæsthesia is reached. Little chloroform is therefore needed, and the anæsthesia is profound and satisfactory.—*Münchener medicinische Wochenschrift*, 1901, vol. xlviii., p. 1169.

Use and Therapeutic Indications of Iodopyrine.—DR. WILHELM BAUM has been using this drug in the medical clinic at Halle, and recommends it highly as an alternative or adjuvant to potassium iodide when this latter is badly borne by the patient or does not seem to be effective in the usual doses. With reference to its mode of medication, the author finds it ineffectual as an ointment, but internally, in doses of three or four drachms a day or more, it is effectual. It may also be employed in hypodermic medication in doses of one and one-fourth drachms of a 25 per cent. solution. The gluteal regions are preferable for injection.—*Therapeutische Monatshefte*, 1901, vol. xv., p. 641.

Heart Disease and its Treatment.—DR. GRAHAM STEELE considers in detail various forms of cardiac lesions, and, under the head of treatment, says: It is the muscle of the heart that can almost alone be influenced by treatment. Certain pathological events strike directly at the heart muscle, and for such treatment is of little avail. These are the toxic degenerations—chief of which is that of rheumatic infection—and mechanical conditions which cripple the supply of nourishment supplying the cardiac muscle, such being narrowing of the coronary arteries or thrombosis of the coronary branches. Alcohol degenerations, he holds, offer the best field for successful treatment. Digitalis, he says, is by far the best drug of the materia medica, but it should be among the last resources of the physician rather than the first. Familiarity breeds contempt in the heart muscle as well as in society. Simple rest in bed induces that much-desired result—profuse diuresis, with

disappearance of dropsy, evidently by diminishing the work of the organ and enabling it to recover, and so restore the balance of the circulation often much better than drugs. The influence of diet is not to be ignored. Exercise deserves a place among therapeutic agents; massage also. If the use of digitalis and drugs of this class can be delayed, by all means it should be done. Tonics and strychnine can always be given with impunity. Dropsy and an engorged liver may surely be taken as indications for digitalis and like drugs. What is necessary to remember is that one cannot go on indefinitely with digitalis. It should always be given in courses of varying duration and as widely separated as possible. A prolonged rest in the early stages of heart disease will often long postpone the evil day, but care is requisite, when the patient gets up, that the change from rest to activity be very gradual; otherwise not only may benefit be lost, but injury be inflicted. Massage, with overfeeding, may be applied during the stay in bed in some cases. The author believes that an immense amount of harm is done in heart cases by a largely carbohydrate diet. It would seem that carbohydrates promote catarrhal inflammation, and in heart disease—for this reason, among others—they should be withheld or rather greatly diminished. The congested liver of heart disease and the crippled state of the organ, whose functions are probably augmented by a largely carbohydrate diet, should not be forgotten. After a certain age, apart from heart or other disease, the customs of society should be abandoned, and a simplification of diet adopted as well as a diminution of carbohydrates made. Thus the patient is allowed toast without stint at breakfast, the bread cut thin and toasted crisp and buttered cold, weak tea or coffee sweetened with saccharin, and an egg or two. Fruit may follow. At midday fish, fowl, or red meat, with well-boiled green vegetables, form the meal in most cases, but a custard pudding or cheese and butter may be added. Not a crumb of bread or biscuit is, however, permissible, and not more than half a small tumblerful of water is to be drunk, with, in many cases, half a glass of spirits added. In the evening a similar meal is given. Between the midday and evening meals a small cup of Chinese tea, sweetened with saccharin, may be given, but no bread or biscuit is permissible. On such a diet the cardiac sufferer is most likely to thrive. If he insists on a big drink—not likely when his carbohydrates are cut short—he should have it at a time far removed from his meal-times. If the patient craves for water, it should not be given with his food. For the treatment of the gastric catarrhs promoted by the venous stasis of heart disease, there is nothing like twelve or twenty-four hours' abstinence. In milder cases peptonized milk or koumiss should constitute the only food during the attack.

With reference to drug treatment: digitalis is the great agent to be employed. The drug acts slowly; and a diuresis, the great mark of its beneficial action, is never induced by it until the lapse of a certain time. If a diuresis sets in at once on its exhibition, such diuresis is spontaneous, in all probability. Occasionally a heart that has failed to respond to digitalis will respond to strophanthus, and occasionally caffeine citrate or diuretin will succeed in establishing a diuresis after both digitalis and strophanthus have failed, by their action on the kidney rather than the heart. The relief of dyspnoea and the promotion of euthanasia by the hypodermic administration of

morphine and atropine is recommended by the author. Paroxysmal dyspnoea is the symptom that specially indicates the use of morphine, and such dyspnoea is often found to be associated with considerable arterial tension. At first nitrites and alcohol give relief, but later recourse must be had to morphine and atropine. As conditions that militate against the use of morphine, much accumulation of bronchial secretion is a stronger contraindication than kidney disease. Indeed, some cases of heart failure in Bright's disease derive great benefit from its use, and stand fairly large doses; but the first dose should always be small; one must feel one's way to the dose that brings relief without risk. One thing is essential for the practitioner who treats heart disease: it is that he should never know when he is beaten while his patient is alive. When his patient is dead he should at least have the consolation of having fought a good fight on his behalf.—*Quarterly Medical Journal*, 1901, vol. ix., p. 347.

GYNECOLOGY.

UNDER THE CHARGE OF

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Surgical Treatment of Cancer of the Ovary.—ESTER and PUESCH (*Revue de Gyn. et de Chir. Abdom.*, 1900, No. 6) from a study of 372 cases draw the following conclusions: 1. The immediate mortality of abdominal section for this condition is not so great as to furnish an argument against surgical intervention, being about 21.3 per cent. 2. Though recurrence of the disease occurs in 59 per cent., it is not greater than after extirpation of the cancerous uterus. The number of cases in which a permanent cure is obtained certainly justifies an operation, without which a fatal termination is inevitable. 3. While operation is entirely justifiable, it is not proper to operate in all cases. The cancerous ovary should only be removed when on opening the abdomen it is found that neighboring organs are involved, or metastatic foci are discovered, or even when the poor general condition of the patient leads to the inference that the disease has become disseminated.

Curettement and Drainage of the Uterus for Salpingitis.—BEAUSSENAT and BLUM (*Revue de Gyn. et de Chir. Abdom.*, 1900, No. 6) bring up the old question of the value of intra-uterine treatment in certain affections of the tubes, which they affirm was originally proposed by Walton in 1887. Twenty cases are reported in detail in which endometritis was associated with salpingitis. They found that catarrhal salpingitis, either single or double, was cured by curettement and prolonged drainage; thickened, distorted tubes were reduced in size, and favorable results were noted, even in

the case of old pus-tubes. Cases with acute symptoms are, of course, excluded. The *rationale* of the treatment, according to the writers, is that in consequence of the dilatation, the uterus becomes softer and the uterine opening of the tube less contracted; by curettement the hypertrophied endometrium which is the cause of tubal disease, is removed; and by prolonged drainage the uterine canal is kept patent, so as to allow exit of the secretions from the uterus and tube.

[It is rather interesting to observe a revival of the theory of tubal drainage, which has been so strongly opposed on anatomical grounds. Without questioning the value of curettement in these cases, we have never been convinced that the operation actually reopens an occluded tube.—ED.]

Anatomy of the Round Ligament.—SELLHEIM (*Beiträge zur Geb. und Gyn.*, Band iv., Heft 2), from careful dissections in the cadaver, notes several points of practical interest in connection with Alexander's operation. He was always able to find the ligament at the external ring. Both the mass of fat (mentioned by Imlach) and the nerve are uncertain guides. Within the canal, especially at the middle third, the cord is attached to surrounding tissues by fibres of muscular and connective tissue.

The writer regards an open canal of Nuck as a rare condition. He never found it in any of his subjects. The ligaments are longer in nulliparæ and thicker in multiparæ. In the latter a process of peritoneum is often carried into the canal; less frequently the cord may be cleft. Both of these conditions result from the process of involution following childbirth.

To expose the ring he recommends an incision, two inches long, beginning about half an inch inside of the pubic spine, and insists on blunt dissection after incising the skin and superficial fascia. No attempt should be made to pick out the tissue at the external ring, for fear of dividing some of the fibres of the ligament. It is not necessary to open the canal.

Retroversion without Symptoms.—E. SCHROEDER (*Zeitschrift für Geburtshilfe und Gyn.*, Band xliii., Heft 3) examined 411 patients, with the view of determining how many with retrodisplacement presented no symptoms referable to the condition. His conclusions are that the normal position of the uterus is ante flexion. In 25 per cent. of his cases retrodisplacement was present without giving rise to any painful symptoms, retroflexion being only half as frequent as retroversion; hence the inference that retroversio flexion is a common condition requiring no treatment in itself.

Tetanus following Vaginal Fixation.—MENZER (*Zeitschrift für Geburtshilfe und Gyn.*, Band xliv., Heft 3) reports a case of tetanus which developed on the eighth day after a strictly aseptic operation, with afebrile convalescence and terminated fatally three days later. The wound presented an unhealthy discoloration and secreted a brownish pus, which, injected into mice, produced typical phenomena of tetanus in a few hours. No tetanus bacilli could be isolated. Injections of antitetanus serum and salt solution were used without benefit. The result of the autopsy was negative. There was no evidence of inflammation in or around the vaginal wound. The writer was unable to explain the source of infection unless on the theory that the

tetanus germ was present within the vagina at the time of operation and was not destroyed during the usual cleansing process. It is interesting to note that a hysterectomy was performed in the same room on the same day, the patient having a normal convalescence.

Vaginal Hysterectomy as a Radical Operation for Cancer of the Uterus.

—WINTER (*Zeitschrift für Geb. und Gyn.*, Band xliii., Heft 3) after careful analyses of the statistics of different countries refers to those of the Berlin clinic as the most favorable bearing on the question of the permanent cure of uterine cancer by vaginal extirpation. Up to the year 1892, 10 per cent. of the patients operated upon were free from recurrence at the end of five years. Since that year the number of cures was 15 or 20 per cent. The writer concludes that the vaginal operation is not satisfactory as a radical method of treatment, and turns to the abdominal as possibly the method of the future, although sufficient statistics have not yet been collected to allow any positive deductions.

Fibroma of the Abdominal Wall.—PINKUSS (*Zeitschrift für Geb. und Gyn.*, Band xliv., Heft 3) reports a case of fibrous tumor of the abdominal wall, of traumatic origin, in a young woman four months pregnant. She received a blow in the abdomen, which was followed by severe local pain and the rapid development of a swelling, so that it was decided to extirpate in spite of her condition.

The writer believes that this case confirms Olshausen's statement that such fibromata, which grow rapidly during pregnancy, originate as hæmatomata, due to tearing of the deeper muscular fibres, the fibrous growths subsequently springing from the fascia.

Incipient Carcinoma of the Portio Vaginalis.—FRANQUÉ (*Zeitschrift für Geb. und Gyn.*, Band xliv., Heft 2), from his studies of the lymphatics of the cervix uteri and broad ligaments in the normal and cancerous portio, arrives at the conclusion that supravaginal amputation is the preferable operation in the early stages of the disease. This view is based on the fact that metastases in the body of the uterus only occur at a later stage, and also that in commencing cancer the deeper lymphatics are not yet affected. Statistics show that in from 50 to 70 per cent. of these early cases, according to different observers, the pelvic glands were not affected. At the Würzburg clinic 27 per cent. of the patients were free from recurrence five years after high amputation had been performed. In ten out of thirteen radical abdominal operations for advanced carcinoma of the uterus no trace of disease could be found in the iliac glands. The writer further opposes Ries' teaching that it is imperative to remove the ovaries and tubes in every case of cancer of the cervix, and affirms that metastatic deposits are rarely or never found in operable cases. With reference to the radical abdominal operation, the writer raises the question whether the immensely greater risk incurred by the patient is offset by the greater number of patients who remain free from recurrence. According to his opinion, the favorable prognosis for the patient lies not so much in the extent of the operation as in the early diagnosis.

Paralysis of the Stomach and Intestines following Operation.—HERFF (*Zeitschrift für Geb. und Gyn.*, Band xlv., Heft 2) concludes a paper on this subject with the caution that especial care should be exercised in operating upon a patient with chronic disease of the stomach. Obstinate and prolonged vomiting after anaesthesia should lead to the suspicion of possibly commencing paralysis of that organ. When such paralysis occurs the stomach should be promptly and thoroughly washed out, all nourishment by the mouth being withheld. Enemata and infusions of saline solution and nutrient injections are important. Faradization of the stomach and the knee-elbow and Trendelenburg's position are also recommended.

Operation for Prolapsus Uteri.—DÜHRSEN (*Centralblatt für Gynäkologie* 1901, No. 29) now adopts the following method: After amputation of the cervix a transverse incision is made through the anterior vaginal fornix and a vertical incision is carried downward from the centre of the transverse. The vaginal flaps are dissected off laterally, exposing the bladder the slight attachments of which to the uterus are divided and the organ is freed as high as the peritoneal fold. The uterus is anteфлекed and the fundus drawn down into the wound, to which it is attached in the usual manner with three sutures of catgut or silkworm-gut, provided that the patient is not past the age of child-bearing. In older subjects it is better to remove a section of the peritoneum in order to secure a fibrous serous adhesion. After excising the vaginal flaps and closing the wound a posterior colpoperineorrhaphy is performed.

The patient is discharged at the end of two weeks, and is soon able to attend to her usual duties, the uterus remaining in normal position.

The writer replies to Gebhardt's objection to vaginofixation, especially the subsequent dystocia which may be caused, by stating that in only one case upon which he has operated has there been any complication during pregnancy and parturition. He calls attention to the most important point in his operation upon which depends the avoidance of future trouble—either not to incise the vesico-uterine fold of peritoneum at all, or, if it has been opened, to unite it carefully before tying the uterovaginal sutures.

Conservative Vaginal Compared with Abdominal Section.—DÜHRSEN (*Proceedings of the Thirtieth Congress of the German Surgical Association*) reports 780 cases of anterior vaginal section for different conditions. He believes that most of the cases formerly treated by laparotomy can be handled equally well by the vaginal route, with a mortality less than 3 per cent. There is no external cicatrix, no danger of intestinal adhesions (?) or hernia, and the convalescence is short, most of the patients being discharged at the end of from nine to twelve days. The only subsequent complication is the possible interference with parturition, which may be avoided by suturing the peritoneum separately.

Among the cases reported were seventy ovariectomies for large cystomata with no deaths, and twenty-eight operations for extra-uterine pregnancy with one death from an accidental cause. Among the conservative operations performed by the vaginal route the writer mentions closure of the tube to prevent conception, salpingostomy, and ignipuncture for cystic degeneration.

tion of the ovary. The latter procedure he has found an efficient way of relieving the pain and hemorrhages due to this condition.

There were seventy cases of myomectomy, in some of which the tumor exceeded in size the foetal head at term.

OBSTETRICS.

UNDER THE CHARGE OF

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Drainage after Abdominal Section.—BURCKHARDT (*Zeitschrift für Geburtshilfe und Gynäkologie*, 1901, Band xlv., Heft 2) reports from the clinic at Würzburg 31 cases in which, for some indication, drainage was employed after abdominal section. These cases were 6 of ovarian tumors, 18 of pyosalpinx, 2 of tubal pregnancy, 2 of suppurating uterine tumor, and 3 of peritonitis. Eight of these patients died after operation, 7 of them of peritonitis, 1 of pneumonia and pleurisy.

He concludes that drainage should be employed in the following cases: 1. When pus in large quantity has soiled the field of operation and the abdominal cavity. 2. When a large raw surface has been left by operation the walls of which are necrotic and infiltrated, and where sound tissue cannot be brought together by suture. In such a case he would employ iodoform gauze drain. 3. When a portion of a tumor must be left behind because of its firm adhesions, and especially where such adhesions are to the intestine. 4. When the bladder or intestine is injured, no matter how carefully they may be sutured. 5. When pus-tubes are so adherent that they cannot be separated from the surrounding tissue after they have been opened and emptied. In this class of cases also he would use iodoform gauze as a drain; in other cases he would drain with a glass tube.

He places very little reliance upon the microscopical examination or test by culture or puncture in cases of suppurative disease. He believes the results of such tests are too uncertain to be a safe guide in employing drainage.

The Treatment of Ectopic Gestation.—IHM, of Königsberg (*Zeitschrift für Geburtshilfe und Gynäkologie*, 1901, Band xlv., Heft 2), reports 44 cases of ectopic gestation and the results of treatment. But 1 of these had ruptured recently. In this case the patient died soon after admission to the hospital from very extensive and uncontrollable hemorrhage. In the other cases there were 2 of suppurating retro-uterine hæmatocele, 16 of retro-uterine hæmatocele without suppuration, 19 of hæmatocele surrounding the tube, 3 of pregnancy in the latter part of gestation, 1 with intact foetal sac at the beginning of the fifth month, 1 in which a large foetal sac had ruptured into

the bladder, and 1 in which a retro-uterine hæmatocele had already been opened by incision through the vagina. In no case did he see an intra-uterine pregnancy with living ovum during the first month.

From the 44 cases he takes 5 which were practically beyond treatment—one of very extensive hemorrhage immediately after admission; a second had been brought from the country, with acute diffuse peritonitis caused by a suppurating hæmatocele; a third, with hæmatocele surrounding the tube, declined to remain in the hospital, and left before treatment could be carried out; a fourth case had carcinoma of the uterus, and the ectopic gestation was discovered accidentally in operating for the cancer, and the fifth case was one in which, six months before admission, the vagina had been opened for retro-uterine hæmatocele. This leaves in his series 39 cases uninjured by attempts at previous treatment.

Of these 19 were treated by operation—17 by abdominal section and 2 by vaginal section; 20 were treated upon the expectant plan without operation. There was practically no mortality from operation. One patient died the day after a large suppurating hæmatocele was opened through the vagina by the thermocautery.

Ihm has collected the statistics of 12 operators, giving a mortality ranging from nothing to 25 per cent. The largest series of cases is by von Schenk—436 cases, with a mortality of 16.5 per cent. The largest number of cases without mortality is 17—that of Winter. In the 17 cases of abdominal section the writer made an effort to ascertain the subsequent history. He was enabled to investigate 15 of the 17 cases; 6 of these replied to letters and reported that they were well and able to work; 1 had had two children since the operation and 2 others had slight pain upon exertion, but normal menstruation. Nine patients were examined and personally questioned; 5 said that they had no pain after leaving the hospital; 1 complained of stitich pains on the left side after rapid walking or mounting stairs; 1 suffered from pain before menstruation, 1 from pain at the time of menstruation, and 1 had developed pain in the back and in the right side. In some of the patients examination found adhesions limiting the mobility of the pelvic organs. The tubes were thickened in some cases and the ovaries sensitive; 1 had a retroflexion of the uterus and parametritis upon the left side; 1 had a tumor the size of a hen's egg upon the left side, and adhesions behind the uterus. It was impossible to say that these changes were directly the result of the operation, or that they had not been caused by gonorrhœa or efforts made in working soon after leaving the hospital.

Of 20 cases treated without operation 13 had hæmatocele behind the uterus, and 7 hæmatocele surrounding a tube. In 4 the tumor was as large as a child's head; in 2 it filled the entire space behind the uterus; in 1 it extended two fingers beneath the navel; in 1 it extended to the anterior superior spine of the ilium; in 1 it was the breadth of a hand beneath the navel; in 1 it extended between the navel and the symphysis, and in 3 it extended to the navel. Of the cases of hæmatocele surrounding the tube, 1 had a tumor as large as a fist at the side of the womb; 2 had a tumor as large as a goose-egg at the side of the uterus, while in the others the tube was evidently enlarged. All cases were examined under an anæsthetic, and in 10 of the 20 cases there was the escape of decidual membrane from the uterus.

One of the patients, who had a hæmatocele reaching to the umbilicus, was operated upon two years before for ectopic gestation upon the right side. She had remained well after the operation.

The treatment of these cases without operation consisted of rest in bed, applications to the abdomen, regulation of the bowels, the use of stypticin; after the danger of hemorrhage had passed salt baths were given to increase absorption, and applications of iodides and glycerin or ichthyol and glycerin tampons were also made. The shortest stay in the hospital of these patients was nine days, and the longest nine weeks; 11 of these patients have since been heard from; 8 have been examined and 3 have replied to letters. One wrote that she was not as well as before; that she had great pain at menstruation and could not work hard; she had not become pregnant. A second wrote that she had irregular pains, and a third stated six months after discharge from the hospital that she could not do full work because of pain in the abdomen upon exertion. The 8 cases examined showed but partial absorption of blood, and abnormalities resulting from irritation of the peritoneum about the pelvic organs. The average stay in the hospital of patients treated without operation was five weeks. To secure the full absorption of such a hemorrhage at least six weeks must be required. The bad effects of ectopic gestation persisted for eight or nine months in patients not subjected to operation, and reducing this to the lowest possible point by allowing for other causes of ill health, it was found that patients not treated by operation suffered for 5 to 7 months from ill health after leaving the hospital. Cases subjected to operation were able to work in four weeks after leaving the hospital. The expectant treatment had no mortality, which agrees with the statistics of Winkel and Fehling, while Thorn places the mortality of patients not operated upon at 1 per cent.

When the two methods of treatment are compared it is seen that both give good results, but with an enormous difference in the time required for the cure. If the two methods of treatment are compared in the same patient, when the patient was treated by operation she speedily made a perfect recovery. When the expectant plan was pursued she was not entirely well in eight months.

Shall we, then, advise section in all cases of hæmatocele? Fehling, Küstner, and Schauta state the mortality from operation as ranging from 5 to 0.9 of 1 per cent., an average of about $1\frac{1}{2}$ per cent. Thorn believes the mortality of the expectant plan to be 1 per cent. The results seem practically the same.

Ectopic Gestation with Septic Infection of the Sac.—MACNAUGHTON-JONES (*The Lancet*, June 29, 1901) reports a case of ectopic gestation for which abdominal section was done about six weeks after the first symptoms. A tumor was present behind the uterus before the operation. In delivering the sac ruptured and some extremely fetid fluid escaped. The pelvic cavity was repeatedly cleansed with weak formalin solution and the bowel well protected from the sac, the only parts exposed being the edges of the incision. A gauze drain was left. The patient did well at first, but symptoms of septic infection developed; the abdomen was reopened, and the margins of the abdominal wound found to be sloughing. Death occurred on the seventh day after the operation.

On examining the specimen the wall of the sac was infiltrated with blood clot and fibrin, and pus was found between the inflamed tube and placental tissue. As the sac was adherent to the bowel, it is reasonable to suppose that infection occurred through the intestine.

Fibroid Tumors Complicating Pregnancy and Labor.—DONALD (*Transactions of the Obstetrical Society of London*, 1901, vol. xliii., p. 180) contributed a paper under the above title. He considers the risks which fibroids cause during pregnancy to be rapid increase in the size of the tumor, with severe pain and great distress, incarceration of the tumor in the pelvis, serious pressure on the bladder, degeneration of the tumor through diminished nutrition, excessive rotation of the pregnant uterus, and abortion or premature labor.

In these cases abortion or labor is often complicated by obstruction of the birth-canal from the tumor, by malpresentations, by retention of the placenta or membranes, or by extrusion of the tumor during labor. Fibroids render septic infection much more likely than in cases without them.

These cases may be divided into those in which pregnancy is allowed to go to term and those in which it is necessary to interfere in the earlier months. It is usually best to allow the pregnancy to go to term, and then to proceed in accordance with the conditions present. Cæsarean section is usually the best method of delivery. He reports a case in which pregnancy went until nearly term, when albuminuria and rapid failure in health made delivery necessary. Cæsarean section and hysterectomy were successfully performed. In the earlier months the writer does not believe that abortion should be induced. Septic infection occurred in one case coming under observation, and the patient was severely ill, but recovered. The tumor was removed later on by abdominal section. In three cases of abdominal hysterectomy for rapidly growing fibroids in the early months of pregnancy a successful result was secured.

When the tumor is so situated that it can be removed in the early months of pregnancy, this should be done, and thus the life of the child may be saved. The removal of subperitoneal pedunculated fibroids is not usually attended with much difficulty. Sessile tumors require enucleation and stitching of the gap in the uterine wall. Should this gap be extensively lacerated, hysterectomy is necessary.

Pseudocyesis—In *La Semaine Médicale*, July 10, 1901, RAPIN reports the case of a patient who had an abortion at six months during the first year of her marriage. After this she had a catarrhal metritis and an attack of acute peritonitis, from which she recovered.

She gradually developed enlargement of the abdomen, with many of the signs and symptoms of pregnancy, and with the firm belief that she was in that condition. She finally came into false labor, having pains for some hours with distention of the abdomen. During the time of her supposed pregnancy her menstruation continued regularly.

Facial Paralysis after Spontaneous Labor.—In the *Centralblatt für Gynäkologie*, 1901, No. 45, GRÖNE reports the case of a child born in face pre-

tation and delivered spontaneously after a tedious labor. The child's face was swollen from the mouth to the eyes. Upon examination an exostosis was found behind the symphysis, to the right of the median line, and extending parallel to the ramus of the pubes. This growth had a smooth surface, and hence had not lacerated the face of the child. The pelvis was otherwise normal in contour. The promontory of the sacrum could not be reached by internal examination. The facial paralysis was upon the left side, and disappeared spontaneously in a few weeks after labor.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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The Evolution of the Ophthalmoscope.—THEOBALD, of Baltimore (*New York Medical Journal*, June 22, 1901), in an address reviewing the history of the ophthalmoscope which has now reached the fiftieth anniversary since its invention, remarks that the problem which the investigators, with the exception of Cumming, were endeavoring to solve was: Why the pupil appears black ordinarily, while under certain unusual conditions it emits a reddish light. Prevost, of Geneva, about 1810, noticed that the luminosity of the eyes of certain animals which had long been known disappears in a perfectly dark room. Rudolphi further showed that it was necessary to look into the eye in a certain definite direction to perceive the reflex. It had also long been known that in certain pathological conditions, such as intraocular growths and detachments of the retina, the eye emits a faint light. Cumming, 1846, then a student in the London Hospital, showed how a luminous appearance or reflection might be obtained in the normal human eye by examination in a dark room with the patient ten or twelve feet from the light source, while the observer's eye looks as nearly as possible into the observed in a direct line between the light and the eye. He supposed that the reflex which could always be obtained in normal eyes would disappear under pathological conditions, and thus furnish a means of diagnosis. Brücke, in 1847, placed a tube through the flame of a candle, and thus obtained a reflex, besides employing a method similar to that of Cummings; he also related an observation of a medical friend who, wearing concave glasses, obtained a reflex from the pupil of a person standing in front of him when the light reflected from the glasses entered the observed eye. In 1849 Babbage contrived an ophthalmoscope by scraping off a little of the silvering from the back of a plane mirror which he set obliquely in a suitable tube. This instrument more nearly resembled the modern instrument than that of

Helmholtz. Babbage, who seems to have been unfortunate in nearly all of his ventures, failed to have his instrument brought before the profession and properly appreciated. His friend, Wharton Jones, published an account of it in 1854, and thus secured to Babbage the credit due him.

After according to Helmholtz the credit of inventing the parent of all later ophthalmoscopes, the first instrument with which the fundus of the living eye was distinctly seen, and mentioning him as the first to suggest the indirect method, Theobald asserts that what was essential in his ophthalmoscope was not wholly original, and what was original was not only distinctly not essential, but was the outcome of a fortunate misconception on his part. This consisted in the insertion of a concave lens between the glass plates and the eye of the observer. Helmholtz was led to employ the lens, supposing that the observed eye would be accommodated for the virtual reflected source of light, the rays would leave the cornea convergent to that point, and hence would need to be rendered parallel or divergent for the observing eye. Theobald claims that this is not the function the lens actually fulfils, that the observed eye has little inclination to employ its focusing power, but that the lens serves to overcome the involuntary focusing of the observer's eye. Beginners with the ophthalmoscope, as is well known, have trouble in controlling their accommodation when looking at a near object. The difficulties first experienced by Helmholtz were great. His words are: "It was at first so difficult to use that I doubt if I should have persevered unless I had felt that it must succeed; but in eight days I had the great joy of being the first who saw before him the living human retina." Eppens, an instrument-maker of Amsterdam, a few months after Helmholtz's publication, replaced the glass plates used by Helmholtz by a plane mirror with the silvering removed from a small space in the centre. In the meantime Rekoss, a German "mechaniker," added two revolving disks which carried a series of concave lenses. In 1852 Reute suggested a concave mirror in place of the plane mirror of Eppens. (The original papers of Brücke, Cumming, Reute, and Helmholtz, all in German, have been bound together in a small separate volume.) Ophthalmologists as such had taken no part in the scientific observations which led to the invention and improvement of the instrument up to the time of Reute; thereafter they were very active in constructing new models. Mery, nearly one hundred and fifty years before, had observed, as he accidentally held a cat under water, that the vessels of the retina and the color of the fundus could be seen. Upon this hint the so-called orthoscope was now invented.

Edward Jaeger, in 1856, was one of the first to appreciate the method of determining objectively the refraction of the eye by means of the ophthalmoscope. The late Dr. E. G. Loring, of New York, deserves the largest measure of credit for bringing the refraction ophthalmoscope to its present state of perfection. His first description was published in 1869. In 1872 Dr. O. F. Wadsworth, of Boston, suggested the use of a tilting mirror, which was subsequently adopted by Loring in a modified form. The older ophthalmologists were at first somewhat loath to admit the excellencies of the new instrument, but the latter rapidly made its way. It is curious to find certain names in connection with the early history of ophthalmoscopy, such as Mr. Spencer Wells and Dr. Christopher Johnson.

Ocular Complications of Mumps.—PÉCHIN (*Gaz. Heb. de Med. et de Chir.*, 1901, No. 45) calls attention to the ocular complications occasionally observed in mumps, and reports a case of iritis with infiltration of the cornea. A young man, aged twenty-two years, whose hereditary and personal medical history was negative, became affected with a severe attack of mumps (double parotiditis) February 17, 1897, followed March 1st by facial palsy of the right side and involvement of the gustatory sense of the right side of the tongue for its anterior two-thirds. The paralysis had disappeared by March 25th, but the parotid affection continued longer. At the end of April, nine weeks after the outbreak of the original disease, the ocular complications—iritis and keratitis—made their appearance. On May 31st he was first seen by Péchin. There was bilateral iritis with posterior synechia and a slight pericorneal zone. The right cornea was infiltrated in the form of a triangle with its apex above, near the centre; the width of the base at the limbus equalled about a quarter of the corneal circumference. The vision of the right eye was $1/2$, the left, 1 badly. The ocular disease was not accompanied by pain, photophobia, or lachrymation, unlike syphilitic or rheumatic iritis. The vision constantly diminished. By June 11th it had become: right, $1/5$; left, $1/4$. Atropine produced large and regular dilatation on the left side, very irregular on the right. Fundi well seen. On July 28th vision was: right, $1/4$; left, $1/3$. Bulbar conjunctiva less hyperæmic. On November 12th vision of right eye was $1/2$, badly, with muscæ volitantes; fundus visible, nerve very hyperæmic. Left eye vision was 1 on January 10, 1898; vision right was, $1/2$ badly; left 1, when treatment was discontinued.

The Micro-organisms of the Healthy and Diseased Conjunctiva, and their Etiological Importance.—SANNA (*Il Policlinico*, 1901, No. 39) arrives at the following conclusions as to the bacteriological findings in the conjunctival sac:

1. The human conjunctiva, healthy or diseased, always contains a varied bacterial population.
2. The pathogenic species are about the same in the animals ordinarily used in experiments (rabbits, dogs).
3. The etiological importance of these bacteria depends upon the physiological conditions of the conjunctiva at a given time.
4. Those bacteria which are found only in the human subject, like the gonococcus, the diplobacillus, and Weeks' bacillus, are an exception to rule 3.
5. The pseudodiphtheritic bacilli, which are ordinarily found in the human conjunctiva, have no pathogenic power and are unable to acquire such.
6. Fränkel's pneumococcus inoculated into the normal human conjunctiva from anærobic cultures always remains innocuous.
7. Cauterization of the conjunctiva of dogs with phenic acid and irritation by croton oil always excite an inflammation with bacterial accompaniment about like that found in other inflammations of the conjunctiva in man.
8. Inoculations of the healthy conjunctiva of dogs with the secretion derived from the conjunctivitis of cauterization and irritation with the substances above mentioned, have never been found to cause inflammation.
9. The micro-organisms found in such conjunctivitis are destroyed in large part by the epithelium and other phagocytic elements of the conjunctiva.

10. The conjunctival epithelium and the other cellular elements exercise a true bactericidal action upon the micro-organisms, while the tears and secretion of the conjunctiva have only a mechanical action upon them.

Glioma of the Retina.—LAGRANGE (*Journ. de Méd. de Bordeaux*, 1901, No. 19) reiterates the statement that glioma has been wrongly considered even to this day as being beyond the resources of therapeutics—a statement he first published in a work in 1890, contrary to the opinion of classic authors of all countries—which overestimates the malignancy of glioma of the retina. Instances of cures are becoming daily more numerous as proven by the works of Panas, Rochon-Duvigneaud and Wintersteiner. Although cures are no longer considered extraordinary, still they are sufficiently exceptional. He reports four additional cases, ranging in age from ten months to nine years. These were all operated upon, and remained well at the time of this report, February 4, 1901, two years in one case; a year and a half in the second; ten months in a third; one month in a fourth.

Sulphate of Copper in Affections of the Cornea and Lids other than Trachoma.—CLAIBORNE, New York (*Medical Record*, July 27, 1901), advocates this agent in the following conditions:

1. In all acute attacks of inflammation of the cornea in which there is thickening, with a succulent, velvety appearance of the upper lid.
2. In all recurrent attacks of superficial keratitis in which the same condition of the upper lid prevails.
3. In infiltrations of the cornea which are the result of preceding inflammations, associated with the same condition of the upper lid.
4. In maculæ of the cornea in children and adults which have occurred a reasonable time after an inflammation, whether the upper lid presents the characteristic appearance or not.
5. In chronic conjunctivitis attended by thickening of the lid associated with blepharitis.
6. In chronic dacryocystitis (particularly in those cases in which the canaliculus has been slit) attended by chronic conjunctivitis.

He applies the solid stick ground in the form of a parallelogram underneath the upper lid between it and the eyeball, so as to reach that part of the lid next to the ball, as well as to the cul-de-sac of the lower lid. The surplus blue stone is washed off with water and removed from the lower lid by a cotton-wrapped probe.

Pathogenesis of Choked Disk.—MERZ (*Archives of Ophthalmology*, July, 1901) draws the following conclusions, among others, from experiments upon dogs and rabbits: Increased intracranial tension alone is sufficient to cause choked disk. It is only necessary that the tension should be maintained uninterruptedly for a certain time. The tension need be but slightly increased—8 to 15 mm. of mercury or less. A transient increase of tension, even though often repeated, leads only to venous hyperæmia and arterial anæmia. The first clinical sign of increased intracranial tension seems to affect the retinal circulation—dilatation of the veins and constriction of the arteries. The nearer to the eye the vessels perforate the sheath of the nerve the sooner do the retinal changes appear.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Supravascular Pericardial Nodules.—KNOX, in a paper entitled "Supra-arterial Epicardial Fibroid Nodules" (*Journal of Experimental Medicine*, 1899, vol. iv., p. 245), describes small nodules which are frequently seen along the branches of the coronary arteries. These he regards as differing from the *periarteritis nodosa* first described by Kussmaul and Maier, and as having no relation to endarteritis. He finds that the outer elastic lamella of the artery immediately beneath the nodule is frequently reduced or has disappeared, and considers this due to irregularities and increase of blood-pressure affecting that part of the bloodvessel wall unsupported by myocardial tissue. In consequence of this the artery wall would here tend to bulge were this tendency not restrained. The formation of dense supra-arterial nodules over the weakened area holds this tendency in check, and may therefore be regarded as an adaptive or compensatory change.

HERXHEIMER ("Ueber supravasale Pericard-Knötchen und Sehnenfleck," *Virchow's Archiv*, 1901, Band clxv., p. 248), studying the same conditions, differs from Knox in some of his conclusions. He agrees that these nodules are distinct from *periarteritis nodosa*, but does not think they depend on any primary change in the bloodvessels, since, contrary to Knox, he finds the vascular changes slight and inconstant. The external elastic lamella, to which Knox attaches special importance, Herxheimer finds to vary greatly, sometimes being increased beneath the nodules, sometimes decreased, and the same variations occur where no nodules are superimposed on the blood-vessel.

Both find that the nodules develop outside the adventitia of the artery from the connective tissue lying between the endothelial and elastic layers of the epicardium—the point of origin, as shown by Meyer and Ribbert, of the milk patches so often found on the epicardium. In all of Herxheimer's cases milk patches also occur, and their mode of origin is the same as that of the nodules. Further, in addition to definite nodules, he finds along the course of the arteries areas of simple thickening of the epicardium, and these can be traced from their earliest beginnings in the epicardial surface. Finally, he describes transitions between these processes. The simple epicardial thickenings may extend along a vessel for some distance, and in some of these places nodules occur. Nodules occur independently of vessels, and by studying serial sections the thickenings along the course of vessels are sometimes seen to be continuous with large epicardial thickenings independent of vessels. From this he concludes that the supravascular nodules and milk patches are structurally and in origin identical.

Three theories have been adduced for the origin of milk patches: (1) They originate from a circumscribed pericarditis; (2) they have a mechanical explanation in chronic vascular pressure changes; (3) they arise from

developmental irregularities, perhaps in the endothelium. The weight of evidence is against the first. The third theory is urged from the frequent occurrence of splits lined by endothelium, but Herxheimer considers these as secondary changes arising from endothelial cells caught in from the endothelium of the epicardium, and points out the infrequency of the patches in the newborn as being against a developmental origin. The second theory he supports as explaining both the patches and nodules. Both occur most frequently on the left ventricle and in cases where the evidence points to high and irregular blood-pressure. The arteries, filled with blood, project above the epicardial level and so are more exposed to friction. The nodules occur oftenest in the region of a bifurcation, where pressure is highest. They do not occur in Herxheimer's cases on the arteries lying protected in the sulcus coronarius.—H. A. C.

Bacteriology of the Gall-bladder.—MIECZKOWSKI ("Zur Bakteriologie des Gallen-blaseninhaltes unter normalen Bedingungen und bei der Cholelithiasis," *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1900, Band. vi., p. 307) made a bacteriological study of the bile obtained from fifteen patients on whom laparotomy was performed for diseased conditions affecting other parts than the bile passages and in whom there was no evidence of a pathological condition of the gall-bladder or ducts. He finds the bile aspirated under these conditions to be sterile in every case, and concludes that human bile obtained from a normal gall-bladder is sterile. The same he finds to be true for the gall-bladder and hepatic and cystic ducts of animals, while in the common duct organisms are usually present. The organisms often found in human bile at autopsy he regards as post-mortem invaders.

Though the bile is normally sterile, he shows that it is nevertheless a medium in which bacteria can richly multiply, though not so energetically as in bouillon. This is especially true for *B. coli communis*.

Mieczkowski considers mechanical conditions to be of more importance in explaining the sterility of the gall-bladder than any bactericidal power of the gall. Bacteria are swept away by the stream of bile; but if this outflow is hindered, as in cases of cholelithiasis, infection takes place. Of twenty-three cases of cholelithiasis the bile in eighteen was infected, fifteen times with *B. coli communis* alone and three times with this organism plus one of the pyogenic cocci. From this he concludes that cases of cholelithiasis should be regarded as infected cases, since the gall-bladder is rarely sterile, and the absolute virulence of the organisms is heightened by the difficulty in absorption of the bile escaping at the time of operation into the cavity of the abdomen.

EHRET and STALZ ("Experimentale Beiträge zur Lehre v. der Cholelithiasis," *Ibid.*, Band vi., p. 350) do not agree with Mieczkowski in regarding the bile as absolutely sterile, since they claim to be able to demonstrate organisms in the bile of guinea-pigs, dogs, and cattle, provided larger quantities (1 to 4 c.c.) of bile are used in making the cultures. The organisms, however, are few in number, since they, as well as Mieczkowski and others, obtain no growths when small quantities of bile are used.

These two observers consider that the organisms reach the gall-bladder from the intestine by way of the gall-ducts. For the proof of this they

adduce the following : If the cystic duct or common duct is ligated, and the bile allowed to escape into the peritoneal cavity, no peritonitis results, while the reverse is true when the common or hepatic duct is ligated ; the last procedure interrupts the stream of bile, and so affords an opportunity for organisms to extend up the duct. In the common duct, close above the papilla biliaria, organisms are generally present in considerable numbers.

MIYAKI (" Zur experimentellen Erzeugung d. Gallensteine m. besond. Berücksichtigung d. bakt. Verhaltens d. Gallenwege," *Ibid.*, Band vi., p.479), in an examination of the bile of seventy-six dogs and rabbits, finds the gall-bladder sterile with one exception (small quantities of bile used in making cultures). From a study of a small number of animals he concludes that in the lower part of the common duct organisms are frequently present, and these are the normal inhabitants of the intestine.

Miyaki also investigated the pathological changes in the gall-bladder necessary to bring about the development of bacteria there. Ligation of the cystic duct produces no change, while ligation of the common duct close to the papilla biliaria causes an infection of the gall-bladder. The introduction of sterile foreign bodies into the gall-bladder produced infection in three out of eight experiments, while in three more animals in which the cystic duct was also ligated the gall-bladder remained sterile. Direct injury to the gall-bladder mucosa caused no infection. Organisms introduced into the gall-bladder, with or without the production of gall stasis, can be recovered in pure culture after periods varying from three days to one year and eight days.

This work seems to show : 1. That the bile is normally sterile in the gall-bladder and cystic and hepatic ducts. The common duct, except in the lower part, is also sterile. If organisms are normally present it is only in very scant numbers. 2. That organisms will multiply in the bile. 3. That conditions favorable to their entrance are not readily produced experimentally, but when introduced the organisms persist. 4. That gallstones favor their entrance, and in cases of cholelithiasis the gall-bladder is almost always infected. 5. That the mode of infection in these cases seems to be by way of the gall ducts from the intestine.—H. A. C.

The Source and Significance of the Cell Masses Occurring in the So-called Nævi of the Skin.—ABESSER (*Virchow's Archiv*, 1901, vol. clxvi., p. 40) says the characteristic feature of the nævus is composed of epithelial-like cells which occur singly, in clumps, and in bands in the papillæ and cutis of the skin. Two views obtain in regard to their origin : The first is that they are derived from the epiderm (Durante, 1871 ; Unna, 1893) ; the second, that they arise from the mesoderm (Demiéville, from the endothelium and perithelium of bloodvessels ; v. Recklinghausen, from the endothelium of lymph vessels ; Ribbert, from the chromatophores). Soldan (1899) regards nævi as neurofibromata. The epithelial origin was favored in 1897 by Waldeyer and Kölliker ; later by Marchand and Orth.

Abesser studied in serial sections sixteen small nævi from adults. He found three types of cells present. The most common, which he calls the typical nævus cell, is a round or oval cell of the size of those in the epidermis. It has a vesicular, epithelial-like nucleus, and lightly staining, often invisible, protoplasm. These cells occur usually in round balls in the

papillæ and the upper part of the cutis. The second kind of cell is small and flat, with a long oval nucleus, which is surrounded by a very little protoplasm; it occurs in thick bands of closely packed cells, which extend to the epidermis and deep into the cutis. The third form, called chromatophore, contains pigment; it varies greatly in size and shape; it may be branching, star-shaped, oval, or round.

The first noticeable characteristic of nævi is that a hypertrophy of the epidermis occurs in the region affected; the epidermis is thickened, the papillæ narrowed; the second characteristic is that the cells in the lowest layer of the epidermis undergo a metaplasia into nævus cells. A certain liquefaction first takes place, which separates the cells more or less from each other; the cells also lose their protoplasmic fibrillation. The cells are now free to escape into the underlying cutis. The writer compares the picture presented by the frayed epidermis between the papillæ as the nævus cells escape from it into the cutis to a splintered broom. Unna is inclined to believe that elastic and connective tissues play some part in forcing the nævus cells free from the epidermis. Frequently clumps of nævus cells are found within the epidermis surrounded by a single flattened layer of basal epithelium. Ribbert thinks that the nævus cells develop in the cutis and extend to and even press into the epidermis, thus forming the clumps of cells above mentioned.

The second, small, form of nævus cell is probably derived from the first, because all transitions in size between the two can be found.

The third form—the branched and polymorphous chromatophore—arises from the pigmented cells of the epidermis by the same process of metaplasia. This is shown by finding similarly branching pigmented cells in the epidermis and by tracing their escape into the cutis.

All three kinds of nævus cells are derived, therefore, in the writer's opinion, by metaplasia from the cells of the epidermis. They lose their protoplasmic fibrillation, but otherwise retain their epithelial character in that they produce no intercellular substance. The tumors which they give rise to should be classed as nævo-carcinomata.

[The fallacy in this work, if there is one, lies in drawing conclusions from the study of nævi from adults in whom it must be supposed that the process of development in the nævi has been quiescent for a great many years; therefore it is exceedingly doubtful if the lesions are in a stage in which they can be rightly interpreted. Nævi should be studied in young children at the time when the lesions are developing; then the process of metaplasia can be followed. The conclusions claimed as proved are too important to be accepted without further study of more favorable tissues.—F. B. M.]

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THE MEDICAL AND SURGICAL ASPECTS OF GANGRENE OF
THE LUNG.¹

BY FREDERICK A. PACKARD, M.D.,

AND

ROBERT G. LE CONTE, M.D.,
OF PHILADELPHIA.

On taking charge of the men's medical ward, a few weeks ago, I found among the patients one who, on May 10th, was admitted to the ward by my colleague, Dr. Morris J. Lewis. He was an Austrian, aged forty-two years, who had been ill for four months with cough and profuse expectoration. As far as could be learned the family history was negative, and he himself had always been well until the onset of his present illness. At the time of the beginning of his attack he was working in the coal mines. His illness began with fever, occasional chills, cough, expectoration, and pain in the right side of the chest. He was under treatment in a hospital at Pittsburg from two weeks after the beginning of his illness until three weeks before his admission to this hospital. On his admission his complaint was of cough, profuse expectoration, considerable pain in the right side of the chest, and aggravation of the cough when turning on the left side. Examination at the time of his admission showed him to be a swarthy, well-nourished man. Examination of the chest revealed nothing abnormal about the heart, either in regard to its position, size, or sounds. Over the left lung there were a few sibilant râles near the apex, but otherwise nothing abnormal was found. On the right side there was impairment of the percussion-note below the third interspace, uninfluenced by position, while both anteriorly and posteriorly the breath-sounds at the lower portion of the

¹ Combined clinic delivered at the Pennsylvania Hospital, October 6, 1901.

lung were tubular in character and accompanied by mucous râles on both inspiration and expiration. The liver and spleen were normal, as also was the urine. The sputum was of greenish color and mucopurulent in character. Examination of the sputum showed the presence of many diplococci and streptococci, but no tubercle bacilli were found on repeated examinations. In the notes made after his admission there is frequent mention of the fact that the sputum was profuse and very offensive. The note of May 20th states that the patient's temperature had continuously remained at about the normal point, and that there was but little change in the physical signs since his admission. Some change in these signs was noted on May 25th, it being stated that the percussion-note was flat laterally and posteriorly over the lower portion of the right chest, with absent fremitus over this area and with increased fremitus above, while the respiratory sounds were noted as being tubular, with a decided amphoric echo. By degrees the sputum became less abundant, and his general condition improved. Just before his discharge, on August 13th of this year, examination of the blood showed that the red cells numbered 4,630,000, the hæmoglobin was 82.5 per cent. of the normal, and the leucocytes were 11,000 per cubic millimetre. Five days after his discharge he was readmitted. For the first time there is then found the note that there was clubbing of the fingers.

The case ran an uneventful course, with almost complete absence of temperature and but little change in the general or local conditions until lately (September 12th), when he began to have irregular rises of temperature. Shortly after this time he began to expectorate small clots of blood, which at times tinged the sputum quite decidedly. Repeated examination again showed the absence of tubercle bacilli, of elastic tissue, and of fatty-acid crystals. On September 19th the notes state that distinct myoidema was present on both sides of the chest, and that examination of the anterior surface of the chest showed no new development except for the presence of a distinct friction-rub in the neighborhood of the right nipple. Posteriorly, on this date, examination of the right side showed dulness amounting almost to flatness from the level of the fourth thoracic spine downward, with cavernous breathing and numerous moist coarse râles over the upper portion of the dull area, while over the lower portion thereof the breath-sounds were almost amphoric. He was at that time still expectorating large quantities of fetid, greenish, mucopurulent material. This was most abundant in the morning, although on change of position cough was intensified and large quantities of mucopurulent material would be discharged. On September 25 he suddenly expectorated about a pint of blood. Examination yesterday (October 5th) gave the following results: He looked quite ill; the cheeks were sunken and the forehead covered with beads of perspiration. There was no cyanosis except of the nails;

the ends of the fingers and thumbs and of the toes were very markedly clubbed. The chest was quite barrel-shaped and showed some apparent diminution in size at the right base. The apex-beat was in the normal position, but the normal area of cardiac dullness was almost entirely covered by hyperresonant left lung. While the heart-sounds could be heard, they were extremely distant, a fact that was accounted for by compensatory emphysema of the left lung. The existence of the latter was shown by marked hyperresonance, with blowing breathing and prolonged expiration over the whole left chest. On the right side hyperresonance was also present over the upper portion of the lung, where the same character of the breath-sounds was noted, with the addition of coarse crackles on inspiration. Below the third rib anteriorly on the right side there was decided dullness, with very distant breath-sounds, and the lower interspaces on the right side, both anteriorly and laterally, were decidedly retracted. Posteriorly the left chest showed nothing except the signs of emphysema. On the right side was marked dullness, with great increase of resistance to the percussed finger over the right lung from the level of the sixth dorsal vertebra downward. Over this area the breath-sounds were loudly cavernous and accompanied by very many metallic râles. Whispering pectoriloquy was very distinct. These abnormal sounds were most marked and seemed nearest the ear at the level of the ninth dorsal vertebra, 6 cm. from the median line.

I need only add to what I have said in regard to the course of this case while in the medical ward, that from the 13th of September until the time he was transferred to the surgical side of the house his temperature never remained at the normal point for any consecutive length of time. After the date mentioned it became quite irregular, at times running up to 103° F. or more, but never remaining at that point for long. The chart thereafter shows irregular elevations and depressions, abruptly replacing the almost continuously normal temperature shown before September 13th. I would again emphasize the fact that at no time throughout his stay in the hospital were there found tubercle bacilli, elastic tissue, or fatty-acid crystals.

We have here, then, a man who has been ill for about eight months with an affection characterized by cough and profuse expectoration. Let us first occupy ourselves with the data given by the history of his illness in regard to their bearing upon the nature of his trouble.

In the family history there is no ascertainable fact having a bearing upon his illness. The patient's lack of understanding of the English language, and the general ignorance among this class in regard to the fate of other members of the family, makes this apparent absence of inherited disease tendency an unimportant factor in determining our diagnosis.

In his past history all inquiries are negative save for the fact that his occupation has been that of a coal-miner. This suggests to the mind several factors that may have a bearing upon the case. Workers in coal mines, like those engaged in such occupations as knife-grinding and stonemasonry, are subject to influences of so harmful a nature that all of those who have been exposed to them for a prolonged time have structural alterations in the respiratory apparatus of greater or less importance. In the coal-miner, or in the case of a man who has worked where coal-dust abounds, the lungs are of an almost uniform black color, while the bronchial glands are similarly altered from the normal as regards their color. These changes in color are due to the presence within the cells of the pulmonary and glandular parenchyma of carbon particles. Large particles of coal-dust, when carried into the air-passages, cause irritation of the sensory nerve endings and reflex cough, which causes their expulsion. Smaller particles, with which the air of the coal-mine is charged, are carried into the true pulmonary tissue, where they infiltrate the fixed cells or are engulfed by the phagocytic leucocytes and carried to the bronchial glands, in which they are retained mechanically by the admirable filtration apparatus of these structures. The continued entrance or presence of foreign materials into any tissue causes irritation of the latter, with consequent connective-tissue formation even in the absence of bacterial contamination. In the lung this increase of connective tissue beyond that normally present causes thickening of the walls of the bronchi and of the pulmonary septa, interfering with the perfect expansion and elastic retraction of the infundibula and alveoli, and weakening of the wall of the bronchi, whose normal coats are more resistant to pressure than are those thickened by cells resembling those of scar-tissue. The prevailing tendency of pathological connective tissue is contraction. In the interstitial tissue of the lung the chief result of this contraction is the interference with the proper vascular (sanguinary and lymphatic) circulation, and the same outcome is present in the bronchial walls. At first sight, interstitial inflammation and contraction of the resulting fibrous tissue would seem to presuppose narrowing of the air-tubes. Such, however, is not the result. Because of the interstitial inflammation of the walls of the bronchioles, the nutrition of these suffer and their strength is naturally impaired. The newly-formed connective tissue lying between the air-tubes in retracting tends to cause the approximation of the bronchioles. This, joined to the weakened condition of the walls of the latter, makes their dilatation a natural outcome of such interstitial inflammation of the lung. Dilatation of the air-tubes means the formation of pockets and reservoirs wherein secretion accumulates and is retained for a time sufficient to allow fermentative and putrefactive changes to occur, with resulting ulceration and progressively increasing

reactive inflammation—a typical illustration of the “vicious circle” so frequently seen in the processes of disease. Such, then, is the possible picture of the lung of one working in a coal-mine.

It might be well here to still further pursue this subject of the coal-miner's lung before proceeding with the history of the case before us. While encapsulation of foreign bodies by connective-tissue formation is a beautiful illustration of the natural methods of protection, this newly-formed connective tissue lessens the resisting power of the organ involved, partly by the disturbance of proper nutritive supply, by involvement of the blood-supply and lymphatic drainage of the part. Infection of such a thickened lung can, therefore, be supposed to be more serious than would that of an organ with normal circulation. The choking of the bronchial lymphatic glands with carbon particles and newly-formed connective-tissue cells would seem to be also a factor that should be considered in thinking of pulmonary infections among coal-miners. In chronic interstitial pneumonia, from whatever cause arising, there is a remarkable tendency to pleural thickening and formation of pleural adhesions. These may still further complicate the picture by the addition of infective accidents to the pleura, the functions and self-protective processes of which must be obstructed by such alterations.

To return to our patient. Four months before his admission he began to feel ill. His first symptoms were fever, with occasional chills, cough, expectoration, and pain in the right side. Bearing in mind the fact that his lungs were probably already in, at least to some extent, the condition of thickening and mild chronic interstitial inflammation present in all workers in coal-mines, what possible accidents may have occurred at that time to cause his acute symptoms? The facts obtainable are too few to permit of our drawing any absolute conclusions, but some clues may thus be obtained, the pursuance of which may help us to arrive at a correct diagnosis of his present condition.

An acute attack of bronchial inflammation would account for his fever, chills, cough, and expectoration. It would not explain the pain in the right side unless we also suppose that he had a coincident infective pleurisy, or possibly a pleural irritation caused by rupture of an old pleural adhesion in the act of coughing. The former of these is somewhat unlikely; the latter is rather far-fetched. An acute attack of pleurisy would be accompanied by all of his symptoms except expectoration. All miners have a chronic cough, due to irritation and inflammation of the bronchial mucous membrane; but the patient speaks as though the cough—marking, with other symptoms, the onset of his illness—were quite severe and accompanied by an increased amount of expectoration. It is probable, therefore, that there was an area of pneumonic consolidation with concomitant pleurisy somewhere in his lower right lung. This would best account for the symptoms present

at the outset of his illness and likewise for the prolonged stay in the hospital, the slowness of resolution being accounted for by a chronic interstitial change in the pulmonary tissue and the lymphatic apparatus of the lungs. In fact, the man has never recovered from this acute illness, and at the present time, now almost nine months therefrom, he is a seriously ill man.

One other point is mentioned by him in his meagre account of his illness. He has volunteered the statement that change of posture aggravates his cough. Such a statement has a quite important bearing upon the case. The easiest explanation of the production of cough upon change of posture is that on changing the position of the body some irritating material is shifted from a location where its presence is not resented to one where irritation is produced. Such conditions are presented by cases of bronchial dilatation. The saccular dilatation of a bronchus shows a mucous membrane destroyed or attenuated, with possibly fairly normal mucous membrane on either side of it. The acrid and irritating material filling such a sacculated dilatation may produce no outward sign of irritation, owing to destruction of the peripheral ends of the afferent nerve of the reflex arc concerned in coughing; but if the position of the body be changed this irritating acrid or putrescent collection flows by gravity out of the cavity and comes in contact with a mucous surface in a position to protect itself by reflex cough. So characteristic is this excitation of cough by change of posture that bronchial dilatation should be at once taken into consideration upon its presence being determined.

What bearing may this bronchial dilatation have upon the etiology of the acute illness affecting our patient? A frequent form of pneumonia is that due to the inhalation of foreign bodies, such as particles of food (called by the Germans "schluckpneumonie"). Ordinarily such particles are expelled from the air-passages at once through the spasmodic cough excited by their contact with the mucous membrane of the larynx or trachea. In certain conditions, such as in profound ether or chloroform narcosis, and in low states to which we apply the term "typhoid" as expressive, the reflex excitability of the mucous membrane of the air-passages is diminished or abolished, and particles of foreign material (food, blood-clots, etc.) gain their way to the deeper respiratory tissues. If our patient has dilated bronchi (and such patients may frequently consider themselves well and perform their daily labors), may we not have an explanation of his acute pulmonary illness in the entrance into the lung tissue proper of material from a dilatation of the bronchus? Such material is of a character to set up violent inflammation, as can be judged from its appearance and odor, even without a chemical or bacteriological examination.

The duration of his illness may be due to the persistence of a pneu-

monic consolidation or to a localized pleural effusion of irritating character.

I think that we have now run out our clues furnished by the history of our patient. Our diagnosis cannot be established on the facts given. Physical examination of the chest is our next resort. By this means we shall endeavor to determine as nearly as may be the physical conditions present, and especially that of the thoracic organs. In the first place, as you see, the man is emaciated, with hollow cheeks and sunken eyes. This throws no light upon the diagnosis, as any of the possible causes of his illness might be accompanied by loss of flesh. On examining his extremities a striking peculiarity is at once seen, and must be visible even to those sitting at a distance. The fingers and toes show a peculiar clubbing of their ends, making them resemble drumsticks as a whole, while the last joints have the appearance well likened to the beak of a paroquet. The nails show increase in their natural curvature, not only from side to side but also and especially from base to edge. In addition to this the soft parts of the last joint seem to be increased in bulk both at the sides of the nail and in the pad on the palmar and plantar surfaces. Just behind the nails the skin is thickened, forming a quite prominent crescentic ridge. In addition, those of you who are near at hand can see that the nails, instead of being a rosy pink color, are almost lilac in color, although there is no general cyanosis. We see here a typical example of "clubbing" of the fingers. This curious alteration in nutrition of the very remotest portions of the body is seen in a number of conditions, but is not thoroughly understood, in spite of the fact that a considerable amount of attention has been paid to it, especially since Marie particularly described this, with other tissue changes in the extremities, under the name of "osteo-arthropathie hypertrophiante pneumique." This nutritional change in the extremities has been occasionally observed in cases of hepatic cirrhosis; but, save for this rare association, it is seen only in certain diseases of the thoracic contents. These are congenital heart disease of certain kinds, long-standing pulmonary tuberculosis, with cavity formation, bronchiectasis, and chronic empyema. It is seen most typically in serious congenital heart lesions, such as patency of the ductus arteriosus with deficiency of the septa, and in empyema in children. It is seen most frequently in pulmonary tuberculosis. While an interesting feature, it throws but little light upon the diagnosis of our case.

Examination of the thorax shows interesting alterations. Superficially, we find a sign to which, at one time, too much importance was attached, chiefly because it was pronounced a valuable sign of pulmonary tuberculosis. This statement was made by no less an authority than Lawson Tait, whose field of usefulness, however, lay more in

gynecology than in phthisiology. The sign to which I refer is that known as myoidema. On striking the skin over any resisting tissue, such as the ribs, clavicle, or scapulæ, there is seen a sharp reflex movement of the skin. This takes two forms—one local or occurring immediately beneath the point of contact of the finger, the other more widely extended and running in both directions from the area struck. To the first of these is applied the term nodular, to the second, fibrillary myoidema. The latter consists of a wave-like contraction, which may be seen in marked instances to travel for many inches to either side of the point of impact, and may be repeated two or three times upon one application of the stimulus, running backward and forward to and from this point. The nodular form consists in a visible and palpable elevation of about the size of a pea. This arises at once on removal of the finger. If watched, the elevation may later be seen to travel backward and forward for a short distance along the same line as that pursued by the fibrillary contraction. In some cases this movable elevation is evidently not the same as that first appearing after the blow, but is apparently due to a meeting of two, so to speak, contraction-waves running in opposite directions. The phenomenon is evidently not the result of œdema or of changes in the vasomotor condition of the skin irritated, but is the result of reflexly excited contraction of the evolutionary remains of the platysma myoides muscle, which in man is grossly seen only in the neck and extreme upper portion of the thorax, but which is more extensively developed, or rather better preserved, in some of the lower animals, allowing, for example, the horse to move the skin of portions of the body in order to free itself from the presence of insects. Why this excessive reflex irritability should be present has never been satisfactorily determined. It possibly has some relation to toxin absorption, as it is as marked in the later stages of typhoid fever as it is in pulmonary tuberculosis. It is seen also in exophthalmic goitre, that epitome of curious phenomena, and in cases of extensive arterio-sclerosis. In most of these conditions there is more or less diminution in the amount of subcutaneous fat, a probable explanation of the vigor of the reflex result arising from sudden compression of the superficial tissues between the skin and the firm underlying bone. The cause and nature of this sign is one of the many questions worthy of careful study. This sign, while interesting, has but little weight in reaching a diagnosis in our case. Leaving this side-issue to return to the further examination of our patient's chest, we find a marked disparity on the two sides. The chest as a whole has, as you see, the circular shape which has caused the name of barrel-shaped to be applied to the type. The antero-posterior diameter is increased, the costo-sternal angle forms almost a straight line instead of a right angle, the scapulæ lie far more toward the lateral regions of the chest, and there

is marked forward curvature of the upper dorsal and lower cervical portions of the spinal column. Such a "barrel-shaped" chest is frequently seen among coal-miners, because of the prevalence among them of pulmonary emphysema. This change in the lungs is one of the many secondary results of the chronic fibroid inflammation produced by the prolonged inhalation of carbon particles. The elasticity of the lungs is impaired and the expiration collapse is rendered less complete, while their nutrition is decidedly impaired, with resulting structural weakness. To this cause for emphysema we have added bronchial catarrh, a very frequent precursor of emphysema. The limitation of the respiratory excursion of the chest as a whole is plainly seen to be more pronounced upon the right than upon the left side, at the right base more than at the right apex. Percussion shows over the whole left half of the chest, and over the right side down to the lower border of the third rib, a note more ample than is that obtained by percussing over a healthy lung; while over the same areas the breath-sounds are decidedly "blowing" and the expiratory sound is greatly prolonged. Evidently, therefore, the left lung and the upper portion of the right are emphysematous, but physical examination fails to tell us whether this condition antedated or was wholly or in part due to the illness which laid him low. The enlargement of his left lung prevents accurate determination of the area of cardiac dulness, but palpation detects a faint apparent apex-beat in the normal position, a point that is of considerable importance in further elaborating our differential diagnosis.

Turning now to the examination of the lower portion of the right side of the thorax we find evidences of trouble sufficient to account for the prolonged and serious illness. More careful examination of this portion of the chest shows decided retraction of the interspaces both anteriorly and laterally. Below the third rib there is decided dulness, with increase in resistance to the percussed finger, diminution of tactile fremitus, and very distant and feeble breath-sounds. The upper line of this area of dulness does not shift on change of posture—the material separating lung tissue from chest wall is not freely movable. Posteriorly the left side again shows the signs of emphysema, with at times mucous râles. On the right side, from the level of the sixth dorsal spine downward, there are dulness, great increase of resistance, loud cavernous breathing, metallic râles, and whispering pectoriloquy. The cavernous breathing and whispering pectoriloquy are most distinct and apparently nearest to the ear at the level of the ninth dorsal vertebra, 6 cm. to the right of the median line.

We have these additional clues furnished by physical examination: There is no evidence of active disease in the left lung or upper lobe of the right lung. There is something separating the lower portion of the

right lung from the anterior chest wall. This material is not air, and, if fluid, it is limited by adhesions. The interspaces of the lower right chest are retracted, showing that the material separating the lung and chest wall occupies less space than did the healthy lung, which would not be the case with pleural effusion or pure tumor formation. Mere retraction of the lung would cause sinking of the interspaces, but would not give us such impairment of resonance or feebleness of breath-sounds. Thickening of the pleura, with contraction of the newly-formed fibrous tissue, would account for all of the signs present on the front of the chest except for the normal position of the apparent apex-beat. The absence of dislocation of the heart should not, however, be allowed much weight, although it is worth noting. Posteriorly on the right side we have distinct signs of the formation of a cavity, with surrounding tissue condensed and airless. Is this cavity in the lung, or is it outside of that organ, but in communication with the air-passages? In favor of its being in the lung is the distinctness of the pectoriloquy as compared with the amphoric character of the breath-sounds, but especially are we aided in making the distinction by the absence of the peculiar bell-like sound conveyed to the applied ear when coins are struck together on the chest wall. If in the lung, is the cavity due to dilatation of a bronchus or to a destructive process involving the parenchyma of the lung? This question is one often presenting much difficulty in its solution, and one at times impossible to determine by physical examination alone. There are two facts weighing against the view that this is a bronchiectatic cavity. In the first place, the area showing signs of cavity is considerably larger than is that produced by a dilatation of a bronchus, and the extreme base is a rather unusual site for such a dilatation. Abscess of the lung might also give rise to just such signs as are present here, and must be considered in making our diagnosis.

Physical examination shows us merely the changes from the normal in the actual condition of the lungs and air-passages. It can do no more. To determine to what these changes are due we can call to our aid the past history of the illness and certain laboratory methods in order to determine the cause of these alterations in structure. The former of these we have considered somewhat, the latter we shall now consider. Throughout the patient's stay in the hospital, and from the beginning of his illness, expectoration has been a prominent feature. We can obtain from him no history as to the character of the sputum at the onset, and we are unable to state whether at the beginning its character was such as to indicate the nature of the early pulmonary lesion. Had we obtained the statement that at first the sputum was scanty, viscid, and of a rusty color, our thought would naturally be that the whole trouble might have followed an acute croupous pneumonia. Our most

careful inquiries only develop the fact that expectoration was a marked feature from the outset. Since his coming under observation the sputum has been abundant, nummular, with considerable fluid, of a brownish color, and for a long time of a very offensive odor. The quantity of the sputum indicates but very little that is significant. Any lesion involving a large extent of respiratory surface would give abundant expectoration. A chronic bronchitis, a large bronchiectatic cavity or cavities, an area of pulmonary excavation from any cause, or a long-standing pulmonary congestion would be accompanied by profuse expectoration. The curious coin-like masses of more viscid material, giving rise to the descriptive term "nummular," are seen most typically in the sputum of a tuberculous cavity, but similar characteristics are present in the absence of tuberculosis, and the "*sputa cocta et fundem petenta*" does not, as was at one time thought, point necessarily to pulmonary cavitation. The large amount of fluid present gives us no distinct clue, although the expectoration is known to be quite fluid in cases of cedema of the lung and in pulmonary gangrene. The odor of the sputum here has been for a long time extremely offensive; not the mawkish sweet odor present so frequently in cases of tuberculous breaking down and in abscess of the lung, but of an acrid, putrescent quality. This characteristic of the sputum is seen in two conditions—bronchiectasis and pulmonary gangrene. In neither of these conditions is there usually a pyogenic wall of granulation tissue, pouring out and covered by pus, but the material contained in the more or less "naked" cavities is less purulent and more serous in nature. The typical sputum of pulmonary gangrene is abundant, watery, and of a dull green color. On standing this separates into three layers—an upper turbid, sometimes frothy layer; a middle thin, watery stratum; and a lower viscid, opaque green or brown layer containing débris of cells, hæmatoidin crystals, fat droplets, fungi, and often protozoon forms. The sputum coming from bronchiectatic cavities has much the same characters, but frequently shows under the microscope fragments of elastic tissue without alveolar arrangement. The absence of the latter from the expectoration furnished from a gangrenous focus has been explained by the presence therein of a peculiar ferment which dissolves the elastic tissue much in the same manner as does the pancreatic juice. In repeated examinations of the sputum in our case elastic tissue was never found, either with or without alveolar arrangement. This fact would point to gangrene of the lung rather than to tuberculous softening, abscess of the lung or bronchial dilatation as a cause of the signs of cavity. The sputum has never been observed by us to be of a sufficiently purulent character to suggest abscess of the lung.

The hæmoptysis that has occurred lately in our case has but little

significance in a diagnostic sense. In "fibroid disease of the lung" blood in the sputum is not infrequent, owing to the high tension in the pulmonary circulation brought about by the obstruction to the blood-current consequent upon the overgrowth of fibrous tissue which constricts or obliterates the smaller bloodvessels. In bronchiectasis hemorrhage occasionally occurs from ulceration of the walls of the cavity. In tuberculosis the hemorrhage may occur early from reactive overfulness of bloodvessels, or, later, from erosion of their walls. In gangrene of the lung hemorrhage is frequent, owing to involvement of the bloodvessels' walls in the destructive process. In the absence of evidence of tuberculosis the more profuse hemorrhage seen here on one occasion would point rather toward gangrene of the lung than toward bronchiectasis, although, as I have said, its significance is diminished by the probable presence of fibroid infiltration from inhalation of carbon particles. Abscess of the lung is seldom accompanied by expectoration of blood, probably because there is usually thrombosis and obliteration of the surrounding bloodvessels before the latter are exposed on the surface of the cavity.

Examination of the blood shows moderate anæmia and slight leucocytosis, the latter being far less than would be expected in pulmonary abscess.

With the evidence before us, what judgment can we render in regard to the condition of this man's right lung? A working hypothesis is: That the man had chronic fibroid change in the lung, with emphysema and possibly some degree of bronchiectasis, prior to the onset of his acute illness. Inhalation of fetid material from a bronchiectatic cavity may have had something to do with the setting up of an acute pneumonic and pleuritic process in the lower lobe of the right lung, necrosis of the consolidated area being aided no doubt by the impaired nutritive state of the indurated pulmonary tissue. The gangrenous process, once started, would be accompanied by increasing thickening of the pleura, which would still further add to the vulnerability of the underlying lung.

Gangrene of the lung occasionally follows acute croupous pneumonia. There is no positive evidence of the latter in our case. It is frequent in "fibroid lung." This we have good reason to suspect here. It is sometimes seen as a result of pressure of a thoracic aneurism. This lesion we cannot find in our case. It is not uncommon in diabetes mellitus, an etiological factor certainly not present in this instance. It is very frequent as a result of the inhaling of a foreign body, especially if the lung were previously impaired in its vitality. This cause we cannot exclude here, yet the absence of known accident of this kind should not too readily lead us to exclude this as an etiological factor. I well remember a case of gangrene of the lung occurring in this ward

when I was resident physician here in 1887. The young man in whom the lesion occurred could give us no help in arriving at a conclusion as to the cause of his pulmonary gangrene; but after finding *post-mortem* a twig of evergreen in the midst of the gangrenous area his friends remembered that at Christmas time, some four or five months before his death, he had "swallowed the wrong way" a bit of Christmas green which he was chewing.

What would be the probable outcome if our patient were left unaided? The patient has been steadily losing ground for some weeks. Pyrexia, loss of flesh and strength, and profuse sweats show the absorption of toxins in abundance. Unless some favorable changes should occur toxæmia will certainly kill him. Can nature help him? It is possible for natural processes to heal the lesion through reactive inflammation walling off the septic material by a capsule of fibrous tissue, disintegration and expulsion of the necrotic tissue, and gradual obliteration of the cavity. Is this a probability in our case? There is no evidence of such natural repair, and this lack of protective power in the lung may be due to previous fibroid induration impairing the resisting power of the tissues. Our patient is in grave danger of death from toxæmia before nature can bring about a cure. With such a focus in the body there is constant danger of infection of other fairly healthy portions of the lungs, of septic emboli being carried from the lung to the brain or other organs, of infection of the endocardium, and of profuse and fatal hæmoptysis.

What assistance can we render to nature? Our only hope lies in supporting the patient's strength by food, tonics, and stimulants, in order to increase his general vitality and that of all his tissues and in lessening the production of toxins. In spite of all our supporting measures the effect of the constant dose of toxin absorbed is not neutralized by our remedies, as is shown by the increasing adynamia. From remedies to lessen toxin production but little is to be hoped. Inhalations of antiseptic volatile materials reach but a short way down in the respiratory tract. The drugs, such as creosote, guaiacol carbonate, and oil of eucalyptus, which we have given him in the hope that when eliminated by the lungs they would act as antiseptics, have appeared to be of little use. After a fair trial of these methods we have come to the conclusion that assisting nature by the removal of the actively diseased area, or rather of the necrotic tissue, by means of the knife presented the only hope of cure. From physical examination we can decide the seat of the lesion with accuracy. Our reasons for thinking operative removal or establishing effective drainage possible will be stated by Dr. Le Conte, to whose care we now transfer our patient, fully realizing that the surgeon's work is rendered less certainly beneficial by the toxic condition which we have in vain endeavored to

combat up to the time when it has become clear that nature cannot bring relief with sufficient rapidity to save our patient's life

Dr. Packard has given you a very clear picture of the medical status of this patient, and it only remains for me to bring out the surgical characteristics which it is important for us to note and consider, and then to describe the operation as it is being performed.

You will remember that the patient's occupation has produced a chronic fibroid condition of the lungs; that months ago some acute trouble was superadded; and that resolution followed very slowly and imperfectly, ending in a chronic inflammatory process, with putrefactive changes. For weeks he has been under our observation and has received the best of medical care, yet his condition is slowly and progressively growing worse, and the symptoms of septic intoxication are becoming more and more pronounced. No further benefits are to be hoped for from medical treatment; therefore we have strongly urged the patient to undergo operation. His condition, as you have seen, is not a very favorable one for operation; but he has some chance of recovery if we remove the gangrenous tissue and drain the cavity, while if it is allowed to remain a fatal termination is almost certain to ensue.

From a surgical stand-point, what are the important points elicited in Dr. Packard's examination? First, that the gangrenous process is confined to one lung. When both lungs are involved surgical interference is hopeless and useless. Second, evidence that the disease is localized to one portion of the lung and that probably only one cavity exists. When gangrenous foci are multiple in one lung we can scarcely hope to drain them all unless they are confined to a single lobe, and operation would be useless unless all of them were reached. For the same reason, when multiple cavities exist they must be very close to each other and capable of forming one cavity when the intervening barriers are broken down by the surgeon's finger. To drain only one cavity when others exist would be useless surgery. Third, that the process is limited and circumscribed, and not diffused. We have evidence of this in the constant, never-changing physical signs and also in the long duration of the case. It would be very poor surgery to attempt any interference in a gangrenous lung before nature had raised a barrier and limited the disease—that is, before a line of demarcation or separation has formed.

It is essential that these three points be determined before operation is undertaken. There are three other points that are important, although not absolutely essential to a successful issue, but which will materially influence our operative technique and also our prognosis in the case, namely, the accurate localization of the diseased area, its depth in the lung tissue, and the adherence of the pleural surfaces.

ACCURATE LOCALIZATION OF THE LESION. Very excellent clinicians have more than once made a mistake in the situation of the disease, and have inferred from the physical signs that the lesion was in the apex of the lung when it was found in the base, and *vice versa*. You can readily see how such an error would handicap the operator. Dr. Packard has spoken to you of the various means we have at our disposal for an accurate localization, and I only wish to add a word about exploratory puncture. In this we have a fairly accurate guide to mark the position of the disease, but in using the exploratory needle we subject our patient to the grave risk of infecting a healthy pleura, and making a single gangrenous area multiple by withdrawing the infected point of the needle through healthy lung tissue. For these reasons I should use an exploratory needle only at the time of operation, when it will give a maximum of information with a minimum of risk to the patient.

DEPTH OF THE LESION IN THE LUNG. Should it become necessary to traverse healthy lung tissue to reach the diseased portion the risk from septic absorption will be greatly increased, for putrid discharges are readily absorbed by healthy lung surfaces. Therefore, the nearer the lesion is to the pleural surface the greater the probability that normal lung tissue will not be exposed.

THE EXISTENCE OF PLEURAL ADHESIONS. These cannot always be positively foretold before operation, but we believe them to be present in this case for the following reasons: The patient's history of previous sharp, localized pain in the chest, suggestive of pleural pain; the present localized tenderness on pressure; the constant, unchanging position of the gangrenous lesion; the slight retraction of the intercostal spaces, with inspiration, and the diminished movements of the end of a needle which has been entered into the lung. All these signs point to adherence of the pleural surfaces. But how can we tell whether these adhesions will be sufficiently strong for our purpose, namely, to keep the lung firmly glued to the chest wall after we have penetrated to the diseased area? Our evidence of this is only circumstantial. We presume that the adhesions have existed for some weeks, and therefore, on account of their duration, that they must be firm and strong.

OPERATION. We make a broad, U-shaped incision down to the ribs in the posterior axillary line, and dissect up the flap, exposing the seventh and eighth ribs. The periosteum of the seventh rib is incised for a distance of three inches, and with a blunt elevator is carefully peeled from the entire circumference of the bone. The rib is divided with bone-cutting forceps in two places, about two and a half inches apart, and the section removed. The periosteum is now carefully incised, avoiding the position of the intercostal vessels, until the back of the parietal pleura is exposed. Inspection shows the bottom of the

wound of a uniform dull gray color with no to-and-fro motion of the lung, and the finger tells us that the lung within is immovable, resistant, and dense. This demonstrates the presence of adhesions. If they were not present the pleura would be a thin, semi-translucent membrane showing the movements of the pinkish lung within, and the finger would be able to appreciate its up-and-down movement. The sense of firmness and resistance imparted to the finger also tells us we shall expose infiltrated lung tissue. With a knife the incision is extended through the pleura into the lung to the depth of half an inch. We now see the lung tissue, of a gray-black color, filled with an inflammatory exudate, and equally resistant in all directions. The incision is again deepened with the knife for half an inch, directly inward toward the root of the lung, and the finger again carried in to palpate the surrounding tissue. In this way, by alternately cutting and feeling, we come to a place where there is a softer area, less resisting, and the fingertip enters a cavity at a depth of two and a half inches from the pleural surface. This cavity is irregular in outline, the size of a small egg, surrounded by infiltrated, friable lung tissue, and as the finger is withdrawn it is followed by a gush of very foul-smelling greenish pus. This pus has the same odor and some of the characteristics of the man's expectoration, showing that the cavity has a more or less large communication with the bronchus. Having reached the seat of the disease, what more shall we do? Irrigate? No, for we might drown the patient by flooding the bronchial tubes, or start a pneumonia, or multiple abscesses by disseminating this highly infectious material. Shall we curette? No, for even a dull instrument might start a fatal hemorrhage or one that would be difficult to control with gauze packing. We will first by touch assure ourselves that no other cavities exist in the immediate neighborhood. With the finger inside of the cavity the lung feels equally resistant in all directions. If a softer area were discernible in one place I should investigate it for a probable second abscess. Next, with the finger covered with gauze we gently swab out the cavity, breaking down any small, projecting septa or partitions. This will remove as much of the sloughing tissue as we can take away with safety. A soft large rubber tube is fastened in, the skin flap perforated for its reception, and the incision closed with silkworm-gut sutures. There has been scarcely any hemorrhage from the cavity, so there is no need for gauze packing. Iodoform and sterile dry gauze complete the dressing. The patient's condition is fairly good, and he has stood the operation very well.

The after-treatment will consist in keeping up the patient's strength with stimulants and tonics, daily dressing of the wound, at which time the position of the tube will be slightly changed by turning it around and withdrawing or pushing it in a little further, and perhaps later by

irrigating the cavity, when we find out the tolerance of the lung. Two serious complications may develop—persistent hard cough and secondary hemorrhage. Cough will probably be due to the mechanical irritation of the tube, and may be controlled by changing the tube's position—i. e., withdrawing it a little or possibly altogether for a time. Secondary hemorrhage is generally the result of ulceration from pressure of the tube, and we will try to prevent it by turning and changing the position of the tube, so that it will daily come in contact with different areas of the lung. Should hemorrhage occur the tube must be withdrawn and the cavity packed with gauze, and at the same time ice will be applied to the chest, and opium and suprarenal extract given. If the packing then brings on severe cough, opium will be freely given to allay it, for the danger of hemorrhage will prevent our removing the packing for some days.

The operation which you have just seen happened to be a very simple one, thanks to Dr. Packard's accurate localization of the diseased area, and to the existence of strong pleural adhesions. Unfortunately, many cases will be much more difficult, and it is well for us to consider now what we would do when the pleural surfaces are free or when the gangrenous area in the lung is not found.

If after resecting a rib and exposing the back of the parietal pleura we find that no adhesions are present, three procedures are open to us. First, more ribs may be resected, to the number of four or five, the parietal pleura carefully detached from the under surface of the chest wall, and a search made by touch and by sight for some point where adhesions exist. If they are found the lung is then opened over this point. While such a procedure has been successfully performed, I cannot recommend the method, for it seems to me that it blindly destroys a large area of the chest wall for a very small gain in knowledge. Second, a small opening may be immediately made into the pleura, a finger quickly introduced, and a search made for adhesions within the pleural cavity. If such a point is found the finger is withdrawn, the opening in the pleura closed, and the skin incision prolonged or a new one made, so as to attack the lung over the point of adherence. When the lung is partially adherent to the chest wall it cannot entirely collapse, and the dangers of a pneumothorax are greatly lessened. Third, if no point of adherence is found the finger will search for some indurated area on the lung surface, and over this area we will create adhesions artificially, by stitching the lung to the parietal pleura. A small curved needle with a long silk suture is passed through the parietal and visceral pleura a quarter of an inch deep into the lung, and the point of the needle brought out a quarter of an inch from the place of entrance. It is re-entered about half-way or an eighth of an inch back of the point of exit, and the same amount of lung tissue included in its grasp.

In this way an overlapping continuous suture is made to encircle a space the size of a quarter or a half-dollar. The wound is then filled with gauze and three or four days allowed to elapse for the formation of firm adhesions, when the operation is completed and the lung opened. If adhesions are encountered, but are found soft, friable, or treacherous, tampon the wound with gauze and complete the operation at a later period.

I cannot warn you too emphatically against the procedure of grasping the lung and opening a gangrenous cavity when adhesion of the pleural surfaces does not exist. In ten cases where this method was resorted to for purulent collections in the lung, five times the operation had to be abandoned, owing to alarming symptoms, two of the patients dying from acute purulent pleurisy and one as the result of the pneumothorax. Three times the cavity was opened, and death followed from acute purulent pleurisy, and in only two cases was the operation brought to a successful issue. Highly septic material when brought into contact with a healthy pleura is as fatal to the patient as it would be in the peritoneal cavity, and death will follow almost as rapidly from septic absorption.

Having successfully crossed the pleura, how shall we look for the diseased area in the lung if it is not immediately before us? Circumscribed gangrene in the lung is always surrounded by a zone of infiltrated inflammatory tissue—nature's barrier to the progress of the disease—so with the knife and finger we will explore any indurated area that may be present. If this fails to reveal the disease we will use the exploring or aspirating needle, and, by passing it in various directions in the lung, judge from the feeling imparted to the fingers the character of the tissue the point is traversing, and also from the discharges the needle may bring away. If this gives us negative results it is best to discontinue any further search, and complete the operation by leaving a drainage-tube in the incision in the lung. All hope of evacuating the septic material need not yet be abandoned, for several cases have been reported where the pus has found its way to the drainage-tube within a few days, and the patient has ultimately made a good recovery.

PROGNOSIS AND STATISTICS. The prognosis of gangrene of the lung is always grave, for many cases which at the time of operation seem favorable will later terminate fatally. In grouping all the cases together, irrespective of the etiological cause of gangrene, we find that under medical treatment about 80 per cent. die, and after surgical interference about 40 per cent. This great difference in mortality is not perhaps solely due to the brilliancy of surgery, for in all likelihood the cases that have been subjected to the knife have been more or less carefully chosen; but, on the other hand, it is undoubtedly true that many lives would have been saved had the surgeon been permitted to

operate at an earlier stage of the disease. Perhaps, then, these two conditions balance themselves, and we have in the numbers forty and eighty the true values of the two procedures. An examination of Tuffier's table of 71 cases where operation was undertaken shows that the etiology of the disease plays an important part in the prognosis. In 53 cases, where gangrene was the result of inflammatory affections of the lung, there were 15 deaths, or a mortality of 28 per cent. Where it was the result of bronchiectasis there were 4 cases, with 3 deaths, mortality 75 per cent.; of foreign bodies, 2 cases, with 1 death, mortality 50 per cent.; of embolus, 7 cases, with 5 deaths, mortality 71.5 per cent.; gunshot wound, 1 case, recovery; perforation of the œsophagus, 2 cases, 2 deaths, mortality 100 per cent.; the etiology is not given in two cases. In examining the causes of death in these 28 cases we find 8 from progressive septicæmia or exhaustion; 5 in which both lungs were involved in the gangrenous process, 4 where the lesions in the lung were multiple, 4 from secondary hemorrhage, 3 operated upon *in extremis*, 3 from cerebral complications, and 1 where the cavity was not found at the time of operation.

NOTE. Brief history of the case after operation: Cough and expectoration considerably diminished; discharge from the drainage-tube free, very offensive, filled with streptococci and staphylococci, and containing many small particles of necrotic tissue. Within twenty-four hours marked emphysema developed, extending from the scalp to the scrotum, and down the right arm to the hand. This gradually subsided, but traces persisted to the end. Temperature varied from normal to 101° F. At the end of a week the patient was evidently rapidly losing ground. His appetite was gone, and free stimulation gave but little response. A gangrenous odor persisted in the discharges from the drainage-tube, but the sputum, which had again become copious, was almost odorless. Irrigation of the cavity was tried, but had to be abandoned owing to violent cough. Dyspnoea became more and more marked, the pulse weaker, the temperature hectic, sweating profuse, until death intervened on October 25th, the twentieth day after operation.¹

¹ An autopsy was performed, and the specimens were shown to the class at a subsequent clinic. The results of the autopsy were briefly as follows: The left lung was twice the size of the right, very emphysematous and anthracotic, adherent for the most part, but where free the pleural cavity contained a small amount of bloody serum. On the right side the pleura was greatly thickened, especially at the base. The whole right lower lobe was the seat of a low-grade inflammation, with much excess of blood, with dense connective-tissue infiltration immediately around the gangrenous area in the right lower lobe. At the right apex there was a small bronchiectatic cavity. The bronchial glands were enlarged and deeply pigmented. The other organs showed no marked pathological lesions.

SCARLATINOUS EMPYEMA OF THE ANTERIOR SUPERIOR SQUAMOMASTOID CELLS.

SPONTANEOUS OPENING IN THE BONE ; BURROWING OF PUS BETWEEN THE
MASSETER AND BUCCINATOR ; OPERATIVE INCISION IN THE SUPRA-
AURICULAR REGION AND COUNTER-OPENING IN THE CHEEK ;
RECOVERY IN ALL RESPECTS.

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ON June 23, 1901, Alexina MacD., white, thirteen years of age, a farmer's child, living ten miles from Philadelphia, was admitted to the Presbyterian Hospital. The patient was at the end of the fourth week of an attack of scarlatina, and desquamation was well under way. The physician who accompanied her to the hospital stated that "almost at the onset of the disease her left ear had pained her and continued to do so for about two weeks, though a slight discharge had soon begun to flow from the external auditory canal." The ear had been freely syringed with peroxide of hydrogen and the meatus firmly stopped with the cotton. (This form of treatment I believe to have been the chief etiological factor in the production of the consecutive mastoiditis in this case, as it often is in other cases.) About the end of the second week the earache ceased, but the region above and in front of the auricle began to swell and grow tender to the touch, the tumefaction extending to the cheek.

At the time of her admission to the hospital the child was very pale and weak, being entirely unable to help herself. The left cheek was enormously swollen, the tumefaction extending from the zygoma and superior maxilla to the inferior maxilla. There was also great edema above the zygomatic arch over the entire temple and above the auricle, the latter standing out from the side of the head at its upper and anterior insertion. The left eye was half closed by edema, and the lids were ecchymotic. Just above and a little behind the upper wall of the bony auditory canal there was a prominent, pinkish fluctuating point. The mastoid region behind the auricle was entirely *devoid of any symptoms*. There was a slight discharge from the auditory canal, the membrana tympani was red and macerated, and there was a small perforation in its postero-inferior quadrant. The hearing was only slightly impaired. Her temperature was 103.8°; pulse, 124; respirations, 28. (She had just been brought in a carriage to the hospital, ten miles from her home.) Under the circumstances it was deemed best to place her in the isolation ward, with special nurse and resident physician. On account of her weakness and the lateness of the hour (5 P.M.) the patient was sponged and dressed in clean night-clothes, placed in bed and let alone until the next day. A milk diet was prescribed. Albumin was found in the urine. The next day the fluctuating point marking the position of a spontaneous opening in the squamomastoid region was frozen by means of ethyl chloride, and incised. About three fluidounces of creamy, odorless pus escaped, and was followed by some thinner, bloody pus. A probe inserted beneath the zygoma toward the cheek passed forward until its point could be

seen and felt projecting beneath the left eye, and when swept downward and backward it could be seen as it approached the angle of the lower jaw, following closely the lower insertion line of the masseter muscle. The probe point could also be felt on the inside of the cheek, by the finger placed in the mouth.

The buccal pus cavity having been emptied by gentle pressure, a female catheter was used as a drainage-tube, as its relative stiffness ensured its patulence and permitted its easy penetration to the depths of the pus cavity. Iodoform gauze was placed over the incision above the auricle, and the whole bandaged with sterilized gauze and let alone for twenty-four hours.

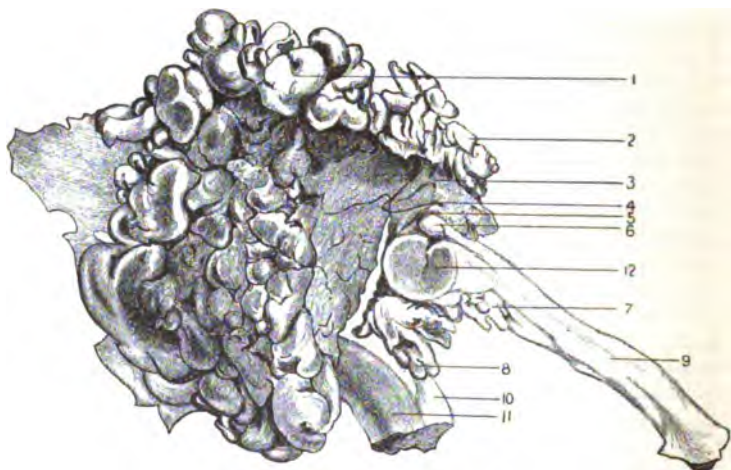
The next day the incision and the drainage catheter were found clogged with hardened pus and the buccal cavity again distended. The drainage-tube was then removed and the pus cavity well probed, with the result of evacuating about a fluidounce of bloody pus and débris. A fresh drainage-tube was inserted, the wound covered with iodoform gauze, and the whole region bandaged with sterilized gauze and let alone for another twenty-four hours. The patient was comfortable, slept well, and remained on milk diet. The percentage of albumin in the urine lessened.

Three days later (June 27) the notes state "the tube has been changed every day and the swelling in the cheek has greatly subsided. The patient's general condition is much improved and her temperature has gradually approached the normal, reaching 99° F. to-day. Albumin still present in the urine." I now observed that the opening above the auricle, supplemented by a drainage-tube, was not sufficient to drain the pus cavity in the cheek, as the latter lay largely below the supra-auricular opening. After emptying the buccal pus cavity by pressure with the hand, washing it out with bichloride solution (1 : 5000) and reinserting a drainage-tube, the temperature would go down, to rise again as the pus cavity in the cheek refilled. On July 1, a week after the supra-auricular incision, it was noted that "the patient's condition has been about the same, except that the temperature has remained somewhat higher than it was a few days after the above mentioned incision, reaching 103° F. on several occasions."

I resolved, therefore, to make the next day a larger mastoid incision for exploration of the mastoid surface, and also a counter-opening in the cheek, near the angle of the lower jaw. On July 2 the drainage-tube was removed from the wound above the auricle, and the patient prepared for operation at one o'clock P.M. On account of the intense heat of the day, 106° F. in the streets, and the exhausting demands on the entire hospital corps, necessitated by the large number of cases of sunstroke brought into the wards, the operation was postponed until the next day. The drainage-tube was not reinserted, pus reaccumulated in the buccal cavity, greatly distending it, the patient's temperature rose to 105° F., and albumin reappeared in her urine in large quantity. All these symptoms, however, promptly disappeared upon opening the wound with a probe and the removal of the retained pus by Dr. Joseph Ackerman, resident physician, in my absence. On July 3 the patient was etherized and a curved incision made from the temporal artery, and running from above and behind the auricle, to the tip of the mastoid process. Inspection revealed an opening in the surface of the squamomastoid region slightly above and behind the

bony external auditory canal, but the rest of the mastoid bone was entirely normal.

A curette was passed forward beneath the zygoma, and granulation tissue, slough, and thick bloody pus were scraped from beneath this bony ridge. Denuded bone was felt along this region for three inches. A grooved director was now passed from the anterior limit of the incision above the auricle directly downward into the pus cavity on the cheek, until its point could be seen beneath the swollen tissues, about two inches in front of the auricle, about half-way between the lobule of the latter and the angle of the lower maxilla. An incision was now made down upon the grooved director and the point of the latter thrust through the incision out upon the surface of the cheek. A rubber drainage-tube was then fastened by silk to the eye of a probe,



Cast of the middle ear and mastoid seen from without. (F. SIEBENMANN.)

1, upper external horizontal or squamomastoid cells; 2, anterior upper horizontal squamomastoid cells; 3, upper malleo-incudal space, attic; 4, external malleo-incudal fold; 5, lower malleo-incudo-squamous space, lower attic; 6, upper pouch of the membrana; 7, tubal cells; 8, tympanic cell, floor of the drum cavity over the jugular bulb; 9, Eustachian tube; 10, petrosal sinus, and 11, transverse sinus, lying together in the jugular fossa; 12, membrana tympani.

the point of which was made to follow the groove of the director until the probe point appeared at the counter-opening in the cheek, when the director was removed from above, and the probe armed with the drainage-tube was pulled through the buccal counter-opening. The posterior mastoid portion of the incision was sutured and healed by first intention. The anterior supra-auricular part of the incision was kept open for the upper end of the drainage-tube, the lower end of the latter being at the buccal counter-opening. The entire field of the operation was covered with iodoform gauze and bandaged.

The child's temperature went down gradually to normal and remained there. Albumin disappeared almost entirely from the urine, and she was removed from the isolation ward to a room in the Woman's Surgical Ward. The pus cavity in the cheek began at once to heal

from the bottom after I made the counter-opening in the cheek, and the latter was allowed to close in a week. The slight aural discharge ceased entirely. The opening above the ear was kept open with gauze drainage, which was gradually shortened as the buccal cavity became more shallow. As long as there was any discharge of pus from the pus-cavity the latter was syringed daily with bichloride solution (1:5000). By July 12th the patient was allowed to get out of bed and walk about the ward. There was still a faint trace of albumin in her urine. By July 20th all of the wound was healed. The membrana tympani still showed a small pin-head perforation in the postero-inferior quadrant. The hearing was normal. On August 1st the patient returned well in all respects to her home.

Pathology. The seat of the mastoid infection in the anterior upper, horizontal, squamomastoid cells was at about the point 2 (see illustration). Spontaneous perforation of the cortex of this region in the squama occurred at the end of the second week of aural mastoid inflammation, and was followed by cessation of the pain in and about the ear. The spontaneous opening in the bone being so far forward on the temporal region, and the recumbent position of the patient favoring a gravitation of pus beneath the more yielding tissues of the cheek rather than backward beneath less yielding tissues over the mastoid, thus there was formed a true gravitation abscess in the buccal tissues.

Etiology. In this case, as in many others, I think infection of the mastoid cells was induced by the use of hydrogen dioxide. The expansive power of this agent is so great that though it forces some pus from the ear it forces some inward, and the deeper cavities of the middle ear and its anterior and mastoid cells become infected. If this agent were not employed, and the natural siphonic drainage of the middle ear thus interfered with, secondary infection of the mastoid would not so readily take place.

Since I began this paper, the physician in whose practice occurred the case I have just reported asked me to see an infant of eight months in whom there was an abscess pointing in the mastoid region behind the auricle and some mucopurulent discharge from the ear. The story was the one I hear so often in these cases, viz.: Rhinopharyngitis about four weeks previous, earache a day or two later, discharge from the ear, then frequent syringing of the latter with hydrogen dioxide for two weeks, with lessening of the purely aural symptoms, *but the supervention of mastoid pain and swelling*, so that I usually see these cases in their *fourth* week. In the case of the infant just mentioned the physician volunteered the opinion that "syringing running ears with hydrogen dioxide did no good, but perhaps harm," to which I readily assented. In fact, I have never seen an acute empyema in previously normal mastoid cavities consecutive to acute otitis media that was not induced by the use of hydrogen dioxide. I am, therefore,

forced to conclude that such an empyema of the mastoid is not a necessary but an artificially secondary result of improper treatment of the primary acute otitis media.

The proper time to syringe or apply lavage in any way to an acutely inflamed previously normal ear is before the membrane ruptures and discharge sets in, in order to prepare an aseptic surface for the opening of the membrane, whether this be effected by the surgeon's knife or by nature.

After the membrana tympani is opened and the germ removing discharge is thus established, I find those cases do best in which the membrana is not touched by anything unless it be by a sterilized absorbent cotton mop by the aurist purely for purposes of examination. But there should be no syringing with anything, as this tends to infection and narrowing of the perforation, damming in of pus and consequent infection of deeper middle-ear cavities. No infectious mechanical obstruction should be thus placed in the way of the acute and beneficent tympanic discharge any more than in the way of a discharge from a pneumonia undergoing resolution, as the processes in both instances are analogous.

EMPYEMA OF THE FRONTAL SINUS: SOME OBSERVATIONS ON ITS TREATMENT.¹

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THE observations herewith presented are based more on my own experience than on an exhaustive survey of the literature of the subject and the reports of cases, and form, therefore, probably only a very limited view of a large subject, since the arc of the circle which any one man's experience subtends is necessarily very small; nevertheless, the sum total of human knowledge is made up of individual contributions.

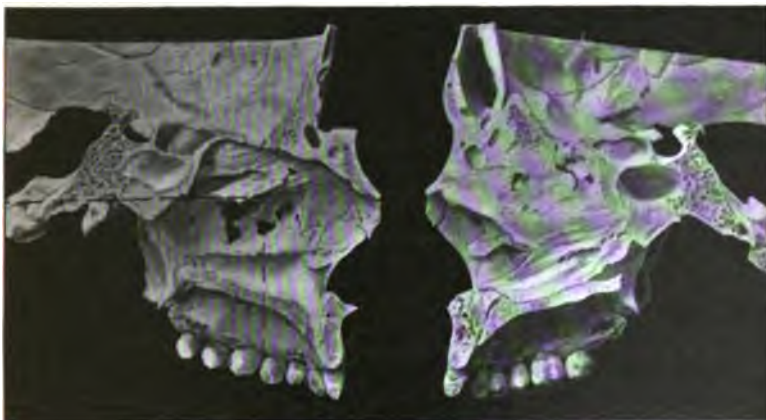
A brief review of the anatomy of the frontal sinus seems necessary in order to deal properly with the subject of treatment. The frontal sinus varies in its position, in size, and in the thickness of its walls. It is not developed in early childhood, but appears about the age of puberty, probably from an extension of the ethmoidal cells into the frontal bone, and attains its full development around the twenty-first year. The

¹ Presented before the annual meeting of the American Laryngological, Rhinological, and Otolological Society at New York City, 1901.

manner of its development may account for the great variation in its size, and for the relation which it bears to the anterior ethmoidal cells.

Normally, the most dependent portion of each sinus opens downward into the nose, through a duct called the fronto-nasal opening. This duct varies in diameter from one-twelfth to three-sixteenths of an inch, and in length from one-half to three-quarters of an inch. Its outlet is in the infundibulum, a term applied to the upper and anterior portion of the hiatus semilunaris, where it is bounded externally and behind by the bulla ethmoidalis, in front by the processus uncinatus of the ethmoid, and internally and above by the anterior third of the middle turbinate bone.

FIG. 1.



Right and left halves of the same skull. In the right there is a good-sized frontal sinus, the walls of which are thicker below the supra-orbital ridge than above, but an opening below the ridge would have given easy access to the fronto-nasal canal, and at the same time have been at the most dependent part of the sinus. In the left half the inferior turbinate and the anterior end of the middle turbinate have been removed. The natural opening into the maxillary antrum is indicated by the vertical dotted line, while above the processus uncinatus is plainly visible. The horizontal dotted line points to the infundibulum, which is prolonged into the frontal sinus as the fronto-nasal canal. In this case a probe or canula could probably have been passed into the frontal sinus. (Author's collection.)

The natural openings of the antrum of Highmore and those of the anterior ethmoidal cells are in the hiatus semilunaris, but behind the infundibular opening of the fronto-nasal duct. Between the frontal sinus and the infundibulum the fronto-nasal duct is separated by thin walls from the anterior ethmoidal cells, while in front of it are usually several small cell spaces, called by Hartmann the frontal cells of the ethmoid; of these he describes three—an anterior, an outer, and a posterior. These cells have very thin walls, sometimes almost as thin as paper. The direction and position of the infundibulum and fronto-nasal duct being dependent upon the size and development of all these

boundary structures, it is easy to understand how difficult it may be in many, or even in most instances, to satisfactorily reach the sinus, for purposes of exploration or treatment, through the natural opening.

The anterior wall of the frontal sinus is relatively thick; its posterior wall less so, but still quite thick; while its thinnest wall is over the orbit, a portion of the roof of which it forms, and at which point the wall may be very thin. Its extension outward and upward is very variable, being on an average from three-fourths to one and one-fourth

FIG. 2.



Two halves of the same skull. In the right half there is no frontal sinus above the supra-orbital ridge, diploic bone being found in its place. Just underneath the ridge, at the inner angle, is seen an opening; this leads to a small sinus which communicates with the nose through the usual fronto-nasal canal, and is large enough for an empyema. The sinus is present in the left half. It is about the average in vertical extent, but outwardly extends but a short distance. In this specimen opening over the roof of the orbit would have come upon the dura. (Author's collection.)

inches above the fronto-nasal opening, and about the same distance outward. It extends backward over the roof of the orbit about half an inch. These are average measurements, but by no means constant. The two sinuses do not usually connect, but may do so; this very rarely occurs, except as a result of disease. They are also variable in their relation to one another, one sometimes being much larger than the other, or one sinus may entirely fail. The division line may be so far to one side as

to allow of the wrong sinus being opened at an operation, or it may run diagonally, as in one of my specimens, and there may be septa dividing the sinus into compartments.

What danger has the collection of pus in these sinuses? What are the chances of cure and the best methods of bringing this about? As to the danger, it seems to me that so far as life is concerned this is relatively small. This statement I regard as correct, in spite of the fact of deaths being reported from chronic empyema of the frontal sinus; since anatomical anomalies, secondary infection after operation, or unusual virulence of the pus would account for these, as reports seem to show. We have all discovered cases of chronic empyema of the frontal sinus, with a constant discharge of foul pus, in which the general health was really very good, and the danger to life by the presence of the pus almost *nil*. I recall one such case, in which the pus discharge had probably existed for years without any great disturbance of health. Relief was sought on account of the nasal obstruction and the foul odor. Polypi were cleared out, the middle turbinate removed, and, this failing to cure, the sinus was later opened externally. It was found to be filled with pus and a low grade of granulation tissue, which latter bled most freely on being curetted. After the thorough removal of all diseased membrane recovery was prompt and uneventful, and there has been no recurrence of the trouble.

If one considers the anatomical relationship, and the fact that nature usually seeks an outlet through the path of least resistance, it will be found that there can be no outlet which nature is likely to force except downward, or downward and forward, or outward through the wall of the orbit; that is, nature will be most apt to force her way out in the region of the ethmoidal and frontal cells, or through the roof of the orbit, in which latter place it is probable that spontaneous evacuation sometimes takes place.

Aside from the symptoms of pain and headache in the neighborhood of the sinus, and the complaint of nasal obstruction, the diagnosis is made by inspection of the discharge, which appears underneath the middle turbinate or between it and the septum, and by exclusion of the other possible sources from which this pus might come. Pressure upward on the wall of the orbit will usually elicit pain, though this is not constant. Disease of the antrum and of the sinus may coexist, and in many cases it may be necessary to make an exploratory incision into the antrum to exclude that as a possible source of the pus. I have several times opened into the antrum in the canine fossa, under cocaine, to determine the presence or absence of pus. Puncture underneath the anterior end of the inferior turbinate is equally efficient for diagnosis. When antrum and sinus are both diseased it is likely that one has infected the other. The antrum is usually the first to be operated

upon under these conditions, as the discharge from the frontal sinus may take care of itself after the antrum is healed. If the exploratory puncture of the antrum has been negative, we then have the anterior ethmoidal cells and the frontal sinus as the possible sources of the pus. The diagnosis here may possibly be very difficult. Transillumination, while by no means decisive, has seemed to me of some value as a diagnostic measure. It certainly is, so far as antrum disease is concerned; and in two of my frontal sinus cases the information furnished by transillumination was positive and accurate.

In the text-books one is taught how to carry a probe through the hiatus semilunaris and the infundibulum directly into the frontal sinus. In practice, with a swollen mucous membrane and an enlarged middle turbinate, the whole of which may have been bathed with pus for a long time, and where polypi may also be present, this may be extremely difficult, or even impossible. The observer may think that the probe is in the frontal sinus when it has passed into some one of the frontal or anterior ethmoidal cells, or even when it is in the superior meatus. The first thing to do, then, in any case in which pus is evident, is to clear away any obstruction, such as polypi, that may be present. As a rule, it will be necessary to remove the entire anterior portion of the middle turbinate. This also applies to those cases in which the suppuration is acute—such cases as are not infrequently seen following the grip, and in which the drainage is obstructed by the swelling of the mucous membrane and its underlying connective tissue.

These acute cases tend to get well if the drainage is free enough; and on their first discovery the middle turbinate should be removed and the drainage made as free as possible. After this is done it is frequently comparatively easy to find the track up to the frontal sinus, and with the aid of a silver or hard-rubber tube, properly bent, to wash out the sinus, and in a relatively short time to bring about recovery. The proper curve of the tube is obtained by first passing a long silver probe and then copying its curve. In cases of this class the question of an external operation ought seldom to occur, and the diagnosis is usually not a difficult one to make or to differentiate from ethmoiditis or antrum disease, since the attack is acute, is accompanied by pain over the eyes and forehead, and the whole clinical picture is one that admits of comparatively little doubt as to the trouble. I have recently had an acute case of frontal sinus empyema, in which after removal of the middle turbinate it was easy to pass a curved canula into the sinus. Daily syringing produced a cure in six weeks, and after some months there has been no recurrence.

In that large number of cases in which the purulent discharge has lasted a long time, and in which the formation of polypi has been brought about from the constant draining of the pus over the struc-

tures, and all the adjacent ethmoidal cells are very likely more or less involved, the question of treatment becomes less easy to decide. These cases are of all degrees of severity. An example of one of the severer ones may make my meaning clearer.

Some three years ago a patient was referred to me by an oculist on account of some trouble in the nose following typhoid fever. She had

FIG. 3.



Specimen somewhat broken, but showing a good-sized frontal sinus, with very thick anterior wall. The ethmoidal spaces are very large, have extremely thin walls, and communicate with the cavity of the sphenoid. All of the turbinates and the infra-orbital wall have been removed. In case of chronic empyema, the entire frontal, ethmoidal, and sphenoidal areas might have been affected. In that case no single operation would have been thorough enough to have eradicated all of the disease. It is specimens like this which probably account for the long and unsatisfactory course which empyemas in this region sometimes take. (Author's specimen.)

complained for several years of severe headaches, for which she had been treated by many physicians. She finally drifted into the hands of an oculist, who, finding that glasses were of no avail, examined the nose. To his surprise, he found that breathing through each nostril was difficult, and that there was a constant discharge of pus from each

side. Thinking that the cause of the headaches might in be the nose, he referred her to me for treatment. Treatment was commenced by first removing at several sittings large numbers of mucous polypi from each nostril. I then made exploratory punctures into each antrum without finding any pus. The pus discharge diminished very much from the left nostril, but was no better on the right. I next removed each middle turbinate. Still the headaches were about the same, and the discharge of pus was constant; nor did syringing into the sinus, which was done fairly easily on the right side, less so on the left, seem to accomplish much. I then did the infra-orbital operation on the right side, in the angle formed by the eyebrow and the root of the nose, and opened freely down into the nose. Fluid pus, without granulations, was present. Thinking that I had now accomplished drainage by external and internal means, I allowed the external wound to close in a short time, and did not carry any drainage-tube down into the nose.

There was improvement for a while, but the pus discharge continued, and after a few months I again operated, this time on both sides, in the same locality, and found pus in each sinus. The opening into the nose was enlarged with large curettes, and iodoform gauze wicking passed through, followed in a few days by good-sized fenestrated rubber drainage-tubes passed clear into the nose; these latter were retained for four weeks. The external openings closed; the openings into the nose, in spite of their size, became so small that a silver probe could not be passed through without pain; still the pus discharge continued.

After a few weeks more she was again operated upon. This time the curette was used with greater vigor, so as to break up all of the cells in the region of the outlet; and larger drainage-tubes were put in each sinus. The amount of pus at this time was pretty small in the right side, but she had been complaining greatly of pain in it. Healing took place as before. The tubes were retained several weeks, but still the pus continued. She was allowed to go home, but as the pain was complained of as much as before, and the pus discharge was considerable, she was operated on again two months later.

This time the incisions were made higher up on the forehead, above the supra-orbital ridge, with the hope that the curette could be passed down into the nose more directly and the operation be a thorough success. It was found that there was still pus in the left sinus, but that the right one had completely closed and had become filled with new osseous growth. This fact would seem to harmonize with the statement of Hajek, that the pain is sometimes worse on the sound side, since, in spite of the healing, the pain did not disappear. At no time did the two sinuses communicate. The wound again healed, and I was under the impression that I had accomplished a cure, as relief was obtained for a time; but something over a year later she came to me with the

statement that there was still some slight pus discharge from each nostril and an occasional swelling at the inner angle of the right eye, with considerable throbbing pain in the region of the temples, especially on the right.

At her own request she was again operated on. Both frontal sinuses were found to be closed with new bone, and were therefore not opened throughout their whole extent. The natural opening into the nose was reopened and made as large as possible, and all anterior ethmoidal cells that could be reached were broken down and destroyed. The right antrum was opened, but contained no pus. An attempt was made to open the right sphenoid sinus, but it was not very successful. Large drainage-tubes were carried down into the nose, and the external openings closed. The throbbing pain ceased, and the general pain after operating was less than usual. The rubber tubes were removed in five days. On the right side a curved silver tube was carried from the external incision down into the nose and retained three weeks. She is improved in every way, though still under treatment, and the pus discharge from the nose has not entirely ceased. That an absolute cure has been obtained is doubtful, as improvement has followed all previous operative procedures. Considering the number of operations the cosmetic results are very good.

I am inclined to think that in this case I have had an empyema not only of the frontal sinus, but of all the adjacent ethmoidal cells, possibly extending as far as the sphenoid, and that the earlier operations failed to open sufficiently for efficient drainage some one or more of these cells; and if I were to draw any lesson from my own failures in this particular instance, it would be to suggest that when an external opening is made the anterior ethmoidal cells should be destroyed with the curette as far back as it can be made to reach when used from above; and if the cutting portion is kept backward and outward, or outward and forward, there is but little risk of injuring any important structures. If turned toward the centre line it will be possible to injure the lamina cribrosa, which is from 14 to 20 millimetres only from the root of the nose and about on the same level, though sometimes deeper.

The foregoing is an extreme case; but can any of the chronic cases be cured by intranasal operative methods? Hartmann thinks it will but seldom occur that they can. In the chronic cases, how long shall the operator be content with having secured fairly good drainage; for it is only exceptionally that the element of severe pain comes in, as in the case just cited? According to Hartmann, the external opening should be made when, after a fair trial of intranasal drainage, the discharge continues and there is still pain. Most of the chronic cases get a certain amount of relief after the removal of the middle turbinate, and are

then somewhat loath to consider the question of an external operation, fearing the deformity and having a certain dread of the length of time required for healing; though the latter, I am sure, would be very much shortened if a sufficiently large opening were made down into the nose at the time of operation; and the deformity, when the operation is done at the inner angle of the eye, under the infra-orbital ridge, is nothing. The washing out of the sinus, even after the removal of the turbinate, is still difficult, since the outlet is small; and if polypi or granulations are present in the sinus no cure will ever be effected until an external opening is made. Various solutions are used for syringing into the sinus. Personally, I have had better results from corrosive sublimate, 1 : 10,000 to 1 : 15,000, than from peroxide or any of the other solutions usually advocated. To be sure, the corrosive sublimate irritates sound tissues and is sometimes painful, but it does more good than any substitute. It should be followed by an alkaline douche through the nasal cavities and pharynx, to offset its irritant effect upon the sound tissues. Protargol, in 2 to 5 per cent. solution, has proved of service in the hands of several observers.

I should say, then, that after spending a reasonable length of time in the attempt to gain proper intranasal drainage by syringing and douching, the question of an external operation should be presented to the patient and he be allowed to make the choice. He will be very likely to ask if we know that there is pus in the frontal sinus, and if a cure will result; and to both of these questions we may be in doubt as to the answer. Hartmann reports a case in which, after opening the frontal sinus, no pus was found, but on further exploration pus was found in the frontal cells. I once opened the frontal sinus in a case of pus discharge which I had at first supposed to be purulent ethmoiditis, having found no pus on exploratory puncture of the antrum. The pus discharge not disappearing after removal of the middle turbinate, I concluded that the sinus must be infected. It was opened and no pus found. Healing took place in four or five days, and no unpleasant results ever came from the operation. The pus, without doubt, came from some unopened ethmoidal cells or from the frontal cells in the region of the fronto-nasal canal.

Will a cure result? This is the other question certain to be asked. Sooner or later, yes; but how long it will take will depend on the extent of bone involved; and this is a question difficult to determine before the sinus has been explored. Diseased bone is notoriously slow to heal so long as any portion of the disease is left, since so long as there is any left it is liable to break down and form pus. Then, again, I have found the tendency of the canal in the nose to close to be a constant source of difficulty. To illustrate:

A case of empyema of the antrum was opened, cleansed, curetted,

cured. Pus appeared in the nose. The middle turbinate was removed and the discharge found to come from the frontal sinus. The sinus was opened thoroughly, and, having in mind previous experiences, the opening into the nostril was made, as I supposed, sufficiently large. A drainage-tube was retained two weeks, and the opening then allowed to close. Five weeks later there was a swelling at the region of the external wound, and the sinus, or at least a portion of it, was found filled with pus. Under cocaine the walls of the sinus were thoroughly curetted and an endeavor made for weeks to bring about healing, but without avail. An ether operation was again done, more bone cut away, the sinus curetted in its whole extent, and the opening down into the nose very much enlarged, a drainage-tube reinserted, and the opening allowed to close. The case was discharged, apparently well, but returned some weeks later with pus as before. The sinus was curetted, drained, irrigated with corrosive sublimate, and at last healed. This last time I was able, with the aid of a silver tube, to prevent the opening into the nose from closing until the bone was apparently healthy. Some months have now elapsed without any return of the trouble, and there is no deformity.

In another somewhat similar case, which also combined antrum and sinus trouble, a gold tube was made and kept in the external wound for a long time, until the last vestige of pus had disappeared.

So far, then, as the external operation is concerned, it must be allowed that the question is by no means so simple as it at first might be supposed, since the so-called radical operation is not always certain to result in a cure.

At the meeting of the Laryngological Society of London in February, 1901, this subject was discussed, and the weight of the argument seemed to be in favor of the more conservative methods of dealing with these cases intranasally. Dr. St. Clair Thompson thought that we should be slow to claim a complete cure in these cases, and recorded a case in which, although the operation had been done two years before, the patient still had to syringe her nose three times a day. The pus, he thought, had come from the ethmoidal cells rather than from the frontal sinus, as the fronto-nasal duct was quite obliterated before he allowed the sinus wound to heal. This, however, only confirms my statement that in the chronic cases we probably usually have to deal with an inflammation of the ethmoidal cells as well as of the frontal sinus.

At this same meeting Dr. Herbert Tilley said that in cases in which only a drop or so of pus showed itself at the end of each fronto-nasal canal he did not consider himself justified in advising a radical operation, and that the nose should be merely cleansed once or twice daily and nothing else done.

Sir Felix Semon replied in answer to the question, "When ought one to perform a radical operation in these cases?" that it was a great achievement to be able to diagnose these cases better and to treat them more successfully than in the past. Over a practice of twenty-five years devoted to special work he had seen plenty of these cases, and, so far as he knew, very few of them had come to grief prior to the discovery of the modern forms of treatment. There had been a few cases with threatening symptoms, but they were and are very few. He had seen cases in which suppuration still continued after the performance of the radical operation, further operations were necessary, and the patient finally was not much better off than before. In his opinion, it is a matter deserving very great consideration as to whether or not the discovery of a little pus coming from the frontal sinus demands a radical operation in every case; and the surgeon is bound to tell the patient that the big operation occasionally leaves some deformity.

Dr. Lambert Lack thought that the indications for external operation are rather indefinite. If the symptoms are of only slight purulent discharge, he thought the cases best left alone. The middle turbinate should first be removed, the anterior ethmoidal cells opened, and the approach to the infundibulum thoroughly cleansed. If this failed he operated externally, endeavoring always to obliterate the sinus, as the only certain method of obtaining a cure. He thought that many cases reported as cures by intranasal operation are really only cases of ethmoidal cell disease.

As a whole, this discussion seems to me to be leaning rather to the extreme of conservatism. It is certainly much more conservative than the other extreme, viz., the operation which Hartmann describes, of removing the whole of the anterior wall of the frontal sinus (*Die Verödung der Stirnhöhle durch Abtragung der ganzen vorderen Wand*). This would doubtless produce a cure, but it is a question whether our American patients would care for the operation. Hartmann himself is opposed to such an operation on account of the great deformity which results, and says that it does not seem to be necessary, as healing can be brought about through the establishment of a free communication with the interior of the nose.

In the final analysis the question of operation must depend in each individual case upon symptoms, such as recurring attacks of pain and cerebral irritation, which suggest dangers to the organism from septic absorption; and upon the judgment of the operator and the confidence reposed in him by the patient. In many persons the question of confinement, or the length of time they will be kept from work, will have a large influence, and they will prefer to endure the discharge of pus which is not producing alarming symptoms rather than be kept

from their daily vocation long enough to produce a cure. I have such a patient at the present time. The daily washing keeps her comfortable; there is no retention of discharge; and she thinks she cannot afford to lose the time required for the radical operation. In another case removal of the polypi and middle turbinate has so much improved all the conditions that the radical operation is declined on the ground

FIG. 4.



Specimen with good-sized accessory cavities, in which operation in any of the places recommended by various authors would have reached the sinus and allowed abundant room. On the right side is indicated the site for the opening above the orbital ridge and also the openings for operation in the orbital roof. In this specimen there are several septa in the sinus floor and large frontal cells; and had there been an empyema it might have been necessary to do one of the radical operations in order to entirely eradicate the disease, though the opening below the ridge could easily have been enlarged sufficiently to give access to the entire sinus area. Below on the left side is indicated the site for operation on the maxillary antrum, while the small openings on the right side show somewhat of the boundaries of the antrum. (Author's collection.)

of lack of urgent symptoms. The drainage is good, the pain on pressure over the eyebrow has disappeared, the odor though still present is much less, and there is but little discharge. As this patient is over seventy years old, and his cardiac conditions are poor, I am limited to intranasal methods of treatment.

If the external operation is decided upon, where shall the opening be made, and by what means? The question of scar is one not to be lightly passed by, and I am decidedly opposed to such an operation as Hartmann describes, in which, after first making an incision and cutting away the bone in the line of the eyebrow, but underneath the ridge, he then opens at right angles down to the eye, lays aside the soft parts, and cuts away the frontal cells, taking care that he does not remove any of the important muscles or soft parts of the eye. This is undoubtedly effective in laying bare the sinus, but leaves an unnecessarily large opening. He does this only when the simpler operation has not opened the cells sufficiently or brought about a free enough communication with the nose.

From my own experience I prefer always to make the opening between the supra-orbital notch and the root of the nose and underneath the ridge. Whereas the wall of the sinus is comparatively thick at this point, the sinus itself when opened is as roomy as at any place, and the bone opening is easily enlarged upward as far as the upper border of the ridge, and outward, so as to give a sufficient amount of room to enter a fairly good-sized curette and explore every part of the region. While the fronto-nasal canal is easily accessible at this point, it is at the same time also the most dependent portion of the sinus. So long as the upper border of the supra-orbital ridge is left the resulting deformity is practically nothing, as the new growth of the eyebrow takes care of the scar.

Before beginning the operation the post-nasal space can be plugged, to prevent the swallowing of blood. A string should be attached to the plug, one end being brought out through the free nostril and the other out of the mouth. After shaving the eyebrow and properly preparing the surface a circular incision is made from just inside the supra-orbital notch and along the lower third of the eyebrow as far as the root of the nose or a little below. The periosteum is laid back, the bone bared, and an opening made down to the sinus at the centre-point of the incision. The sinus being opened and explored with the probe, the external opening is still further enlarged as in the individual case may seem necessary. When this is done sufficiently a probe can be passed downward into the nose through the natural opening and its direction noted. This opening is then enlarged with the curette, so as to give all the room that is needed, more of the bony wall being cut away if required and in whatever direction needed; and, bearing in mind my own experience, I should urge upon the operator to make a larger opening into the nose than he thinks he needs—in fact, the largest opening possible, especially backward and outward, so as to avoid any danger of injuring the lamina cribrosa. Use small curettes at first, and larger ones if needed; those with curved shanks are pre-

ferable. The whole area of the sinus which can be reached is to be curetted and its ramifications followed up. The extent and vigor of the curetting will depend upon the condition of the mucosa and the presence or absence of granulations. As far as possible, all of the ethmoidal cells in the immediate vicinity should be opened. When using the curette in the fronto-nasal canal or in the ethmoidal region the little or index finger should be kept in the nostril as a guide, the middle turbinate having been in all cases previously removed.

I have tried gauze wicking of good size as a means of keeping the fronto-nasal canal open, but do not like it, as it is very painful to remove. Drainage is best attained by means of rubber tubes slightly fenestrated, one end of which presents at the external wound and the other out of the nostril. These may be changed every other day, the new one being readily attached to the old one by carrying a needle with a long thread twice through each one and leaving the threads, cutting out the needle at the eye. The old tube is then drawn down, the new one follows it, and the threads can be cut off. The advantage of putting the thread through twice and then leaving it long is, that if there is any interference on the way and the rubber pulls the threads might pull out and the following tube be lost; but if the threads are sufficiently long the following tube will not be lost, and the first one can be drawn back in case the second tube will not follow, and a smaller tube can then be used. These tubes should be retained as long as may be necessary, certainly two or three weeks. After that time if a tube is required it is better to use one of silver, properly curved. At the time of operation a stitch is put at each side of the wound, leaving an opening sufficiently large for the drainage-tube. In cases which have lasted but a short time, if the opening into the nose is very large, it may do to close the external wound; personally, I prefer to keep it open for a while, when if the case progresses well the tubes can be removed and the opening allowed to close. As the incision is a curved one, when the tube is finally withdrawn the tendency is for the opening to close perfectly, leaving only a slightly depressed scar, completely covered by the eyebrow and not noticeable in any of my cases except on close inspection. When the opening is made on the forehead above the ridge there is more likelihood of an unsightly scar if it cannot be closed at once and perfect apposition obtained; there is also more likelihood of a depression than when the opening is made at the angle of the eye. The only possible advantage of opening above is that the opening down into the nose is rendered straighter. I think, however, that this advantage is more theoretical than real. If the operator were certain that he had removed all of the diseased mucous membrane, and that the outlet into the nose was sufficiently large, the external wound might be closed at once; of this, however, one cannot be sure.

In case the cavity is filled with granulations, Hartmann would take off the frontal wall. As already cited, one of my cases was as full of granulations as it could be, and yet it healed in less time than any of the others; so that it does not seem to me to be necessary to take off the frontal wall.

FIG. 5.



Specimen showing large frontal sinus extending backward and outward over the entire roof of the orbit. In case of empyema, operation underneath the supra-orbital ridge and between the supra-orbital notch and the root of the nose would give ready access to the whole interior of the sinus and to the fronto-nasal canal as well. (Author's collection.)

Considerable swelling of the loose tissue about the eye always follows any external operation, but lasts only a few days.

The after-treatment will depend upon how thoroughly the disease can be eradicated at the first operation. In many cases recovery will be prompt and satisfactory; in others final healing will be tedious and long delayed, and there may be some pus discharge for some time. The pus discharge may come from some of the ethmoidal cells, which

in long-standing cases are almost always involved. Re-operation may be necessary, even when the first operation was apparently absolutely thorough. The principle which must be our guide is, remove all diseased bone and mucous membrane that it is possible to remove, provide free drainage, and have as little remaining deformity as possible. The time required for a cure varies, according to the degree of severity of the disease, from a few weeks to several months.

Beside the operations described there are a number of other procedures for the cure of empyema of the frontal sinus which bear the names of their originators, and which I shall briefly describe, since they appear frequently in literature, and there is sometimes some confusion of terms.

The Operation of Nebinger-Praun. The essential point of this is the wide opening of the anterior frontal sinus wall. An incision is made from the naso-frontal suture along the orbital border of the supra-orbital notch. The anterior bony wall is laid bare, whatever is necessary, first of the nasal, and then of the frontal bone, being chiselled away. A vertical incision, 4 to 6 cm. long, is next made, following the border of the frontal vein. Bone is now chiselled away corresponding to the breadth of the sinus, the horizontal portion being laid bare. After the horizontal portion is laid bare, the purulent contents removed, and the wall curetted, the opening into the nose is enlarged. Nebinger recommends draining the sinus at the place of junction of the horizontal and vertical cuts.

The Operation of Jansen. The chief feature of this operation is the removal of the inferior wall of the frontal sinus, and, in very large sinuses, partial resection of the anterior wall. A curved incision is made underneath and parallel to the eyebrow from the lateral border of the orbit to the side wall of the root of the nose. After loosening the periosteum the superior wall of the orbit is laid bare. The inferior wall of the sinus is next removed. In the case of a very large sinus the inferior border of the anterior wall is also removed. The diseased mucous membrane and granulation tissue are removed with sharp curettes.

While this method gives free access to the floor of the sinus, the opening into the nose is not so readily attained as by some of the other methods. Having seen some cases some time after the Jansen operation, I am not especially pleased with the ultimate cosmetic results; in one case, particularly, the eye on the operated side seemed to sag.

Method of Ogston-Luc. To open the sinus and ethmoidal cells an incision is made along the inner third of the orbital wall, extending about 1 cm. until the root of the nose is reached, the periosteum laid back on one side toward the forehead and on the other from the orbit, and the sinus opened a little to the outward from the median line and mostly above the orbital ridge. After opening the sinus an opening

is next made into the ethmoidal cells, and after establishing a wide communication between the sinus and the nose a drainage-tube is carried down into the nose, and the external wound at once closed.

This operation has many advocates, and is frequently performed. When the external wound is at once closed it is probable that the resulting scar is very slight. In those cases in which it becomes necessary to keep the external wound open for a time—and these are quite common—it does not seem to me likely to produce as good cosmetic results as the infra-orbital operation I have described.

Method of Czerny. Czerny makes an osteoplastic flap, enlarges the opening into the nose, tampons with iodoform gauze for a sufficient length of time, and as soon as healing is brought about lays the skin flap, with its attached lamella of bone, on the side of the original wound. This is said to produce good results, without deformity.

Method of Kuhnt. This consists in the removal of the entire anterior frontal sinus wall and the entire diseased mucous membrane, including the upper portion of the fronto-nasal canal. A horizontal incision is made along the inner two-thirds of the supra-orbital ridge; then a vertical upward incision is made at the inner angle of the first incision. After pushing back the soft parts and the periosteum the entire anterior wall of the frontal sinus is removed with chisel and bone-cutting forceps. The bone edges are left smooth and all septa in the sinus are removed. The entire mucous membrane is next removed from the sinus and from the upper portion of the fronto-nasal canal. A drainage-tube is then inserted and the external wound closed with sutures. The after-treatment consists in the daily syringing of the sinus with sublimate solution. The stitches are removed on the sixth or seventh day.

Röpke has modified this operation so as to make a wide passage from the floor of the sinus to the ethmoidal cells, clearing out the latter as far as necessary and then packing the cavity with iodoform gauze. When both sinuses are affected the vertical incision is made in the middle line, and the anterior walls of both sinuses, together with the division line and the prominent portion of the nasal process of the superior maxilla, are removed.

The cosmetic results are said to be good, there being marked depression in only one of Röpke's cases. Double vision was present in three cases, but passed away after a while. Unless the mucous membrane is entirely diseased it would seem almost superfluous to remove every portion of it, since it must all be re-formed again for the proper nourishment of the interior of the sinus unless the sinus is to be obliterated. In this latter case I fail to see how there can help being some deformity after such an operation as this.

Operation of Killian. This consists in chiselling and curetting the frontal sinus, with temporary resection of the nasal bones.

Operation of Spiess. Spiess opens the frontal sinus intranasally with the drill and the end of a flexible shaft, under the X-ray.

This operation would seem to be a rather difficult one to do, and not entirely devoid of danger, since where there are so many sinuses and cells the X-ray picture might easily mislead. I have seen some of the X-ray pictures of the operation in Dr. Spiess' clinic. Both Dr. Spiess and Prof. Moritz Schmidt regard this method of operating as valuable and certain, since it gives a large opening into the sinus without external opening. So far as I know, this method has not as yet been followed by other operators, at least not to any extent.

It would be much simpler if these various operations were described and known according to their anatomical characteristics rather than by the names of their real or fancied originators. Too many things in medicine and surgery are described in this manner. It seems to me that it is sufficient, and far better, to describe a procedure as such, rather than as A.'s, B.'s, or C.'s. The reader is then enabled to know what is being described without first looking up a biographical dictionary of medicine.

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THE DIAGNOSIS OF LATENT FRONTAL SINUITIS.¹

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THE inflammatory diseases of the frontal sinus may be either manifest or latent. Under manifest inflammations are included all those cases where the inflammatory discharge, often on account of obstructions to its normal outflow, seeks an outlet through the walls of the sinus, and in so doing produces symptoms which point unmistakably to involvement of the sinus.² These symptoms and their relation to disease of the frontal sinus have long been understood. This is the classical empyema.

Aside from these comparatively rare cases of manifest frontal sinusitis,³ there exists a far greater number of cases where the disease produces no outward or manifest symptoms and where the only abnormal conditions are the secondary changes produced by the pus where it is discharged into the nose, with, perhaps, a few uncertain subjective symptoms. This so-called latent sinusitis passes often not only unrecognized, but unsuspected. The detection and differential diagnosis of this condition is distinctly an achievement of modern rhinology, and is generally considered to be the most difficult to demonstrate of all the accessory sinus diseases.

Latent frontal sinusitis often lasts over a period of many years, with longer or shorter intervals when an acute exacerbation of the trouble produces symptoms which, while they do not point positively to disease of the sinus, at least should lead one to suspect this condition.

Among these symptoms frontal headache is the most constant. This is sometimes only a dull, heavy feeling or sensation of pressure, sometimes of a more severe, throbbing character. More characteristic is an intermittent type of frontal headache coming on in the morning and disappearing toward the middle of the day, only to return with systematic regularity the next morning at the same hour. The intermittent character and the location just above the orbit have often led to a mistaken diagnosis of supra-orbital neuralgia. This typical frontal headache is commonly seen in cases of acute inflammation which so frequently follow coryza, and especially influenza. Here the congestion

¹ Read before the Chicago Medical Society, December 18, 1901.

² The old view that the cause of pus breaking through the sinus walls is always obstruction or closure of the nasal orifice, is not tenable, since it has been repeatedly observed that the passage was freely open in cases where ectasia took place.

³ The term *sinusitis* has been used in some recent publications in English. The term signifying inflammation of a sinus should be formed by adding to the noun stem *sinu-* the suffix *-itis*, which gives the term *sinusitis*. This is the term used by the best foreign authorities, and it is desirable that we should adopt it.

about the orifice of the nasofrontal duct causes a temporary closure analogous to the closure of the Eustachian tube under similar conditions. The headache is often relieved by the application of cocaine about the orifice of the duct in the middle meatus. The intermittent character of this headache, coming on shortly after rising and disappearing when the patient has been up several hours, has been explained by the secretion accumulating while the patient is in the horizontal position, for then the opening is not at the lowest point of the sinus. When the upright position is assumed, this accumulated secretion sinks into the opening of the nasofrontal duct, which now forms the lowest point of the sinus, and by its pressure there causes pain until it has finally escaped into the nose.

Characteristic as frontal headache often is in cases of frontal sinus disease, it is not a positive diagnostic sign. Empyema of the antrum often causes a similar headache, and neurasthenic headache may readily be mistaken for that of frontal sinuitis. In cases of unilateral inflammation it has occasionally been observed that the pain is more severe over the normal sinus. I myself have never seen this in connection with frontal sinus disease, but in a case of unilateral empyema of the antrum I have seen a severe headache of long standing, which was limited to the opposite side, disappear completely upon irrigation of the diseased sinus.

Of greater diagnostic significance is the tenderness on pressure often felt over the affected sinus. This can sometimes be felt over the entire anterior wall; sometimes the orbital wall alone shows tenderness, this being the thinnest wall of the sinus. Percussion of the anterior wall will often show distinctly more tenderness over the diseased sinus. In several cases I have been able to verify the experience of Kuhnt, who was able to outline the extent of the sinus by noting the extent of the tenderness. Tenderness over the sinus often fails when the intranasal examination shows the presence of pus in the sinus. On the other hand, tenderness of a marked degree is occasionally found over a normal sinus.

Transillumination does not give such reliable results as with the maxillary antrum. I have seen several cases where a deeper shadow was found over one frontal sinus, and where afterward an empyema limited to that sinus was demonstrated. More often, however, I have found transillumination give negative results, when a further examination disclosed the presence of pus.

In cases of long standing empyema of the frontal sinus secondary degenerative changes of the mucous membrane are often found about the orifice of the nasofrontal duct in the middle meatus. The anterior end of the middle turbinated body is frequently found in a state of polypoid degeneration. The point opposite on the septum in the region

of the tuberculum septi sometimes undergoes a similar more diffuse polypoid degeneration, the presence of which Schaeffer considered an important sign in diagnosing frontal sinus empyema. The mucous membrane about the anterior end of the hiatus semilunaris and the infundibulum undergoes sometimes a diffuse, sometimes a more circumscribed degeneration, often giving rise to a cluster of true mucous polyps at that point. A fleshy polypoid mass sometimes develops in chronic cases on the unciform process which forms the lower lip of the hiatus semilunaris. This is often referred to by the Germans as the "lateral schleimhaut-wulst." These secondary changes about the anterior part of the middle meatus should always lead one to suspect frontal sinus disease. It is not possible, however, to base a diagnosis upon their presence, since suppuration of the antrum or of the anterior ethmoid cells may produce quite similar changes.

More characteristic are the appearances about the middle meatus in cases of acute frontal sinuitis. One often sees along the concave edge of the middle turbinated body a red, oedematous area. A drop of cocaine applied to this point will be followed in a few minutes by a partial reduction of the oedema, and often a suspension of a severe frontal headache. The inflation of the sinus by the Politzer method, as suggested by Hartmann, is often followed by as striking a relief as that following the inflation of the middle ear when the Eustachian tube is closed.

A continuous discharge of pus into the middle meatus has been considered a valuable indication of frontal sinus empyema, for the opening, being at the lowest point of the sinus, does not allow the secretion to accumulate. The antrum of Highmore, which also drains into the middle meatus, has its opening at the very top, so that secretion accumulates in the sinus until a change in the position of the head favors its outflow, when it is poured in quantity into the nose. A continuous discharge of pus into the middle meatus may come from the anterior ethmoid cells, the frontal or the infundibular cells as they drain directly into the anterior end of the infundibulum. (See Figs. 2, 3, and 4.)

On the other hand, discharge from the frontal sinus is often intermittent when only a limited amount is secreted, giving rise to the well-known morning discharge of pus which accumulates while in the horizontal position. This is followed by cessation of discharge for a number of hours. An intermittent flow may also be caused by a partial obstruction of the duct by polyps. The secretion accumulates behind this barrier until the pressure is sufficient to force open the passage, when a considerable quantity is discharged at once, followed again by a period of retention.

It is evident that whether all of the above symptoms fail in a given case, or whether part or all of them are present, we can neither posi-

tively exclude frontal sinus disease nor positively diagnose its presence. *The final diagnosis of latent frontal sinuitis must rest upon the actual demonstration of pus in the sinus.*

The course taken by pus in its passage from the frontal sinus into the nose is first through the nasofrontal duct into the anterior end of the infundibulum, then through the hiatus semilunaris into the middle meatus. This formation of the passage is the *typical* one, and results

FIG. 1.



Right lateral wall of nasal chamber, showing method of removing anterior end of middle turbinate body with a wire snare.

from the indirect mode of development of the frontal sinus. (Killian.) In other cases the pus passes directly from the sinus into the middle meatus at a point in front and a little to the median side of the hiatus semilunaris, and has nothing to do with the infundibulum. This is the *atypical* formation or the direct mode of development of the sinus. (Killian.)

In cases where the middle meatus is free from polyps and where the middle turbinated body stands well out from the outer wall, one may proceed at once to the examination of the nasofrontal duct and to the introduction of the catheter into the sinus. In most cases the view into the middle meatus is obstructed by polyps or by a curling under of the middle turbinated body. In such cases the first step is to com-

FIG. 2.



Right lateral wall of nasal chamber with anterior end of middle turbinated body removed. The catheter is introduced into the frontal sinus through the typical passage, which is a continuation of the infundibulum upward and forward.

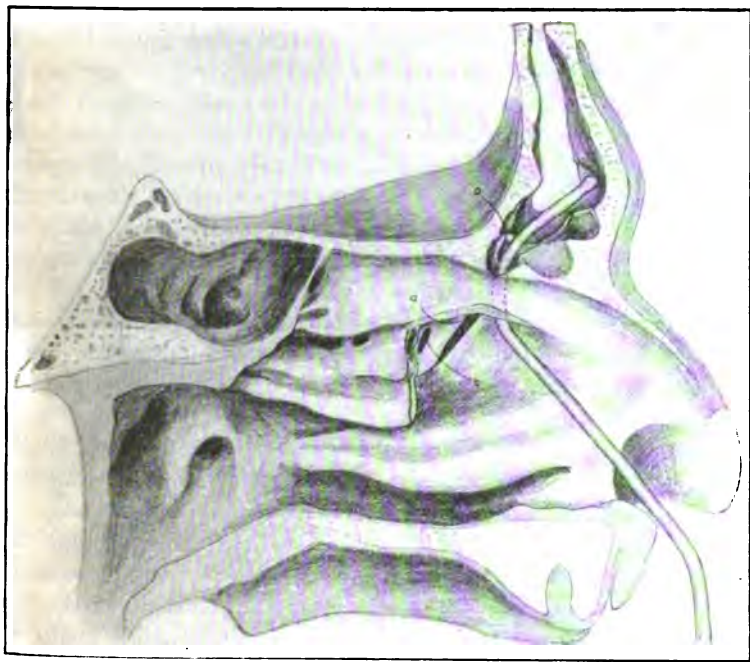
a, a. Frontal cell opening into the anterior end of the infundibulum just to the outer side of the ductus nasofrontalis.

b, b. Anterior ethmoid cell located in the unciform process and opening into the infundibulum.

pletely remove all the polyps, and if the middle turbinated body obstructs the view into the middle meatus, or prevents the introduction of a catheter, the anterior end of this body should be resected. The resection of the middle turbinated body not only opens the way for the inspection of the middle meatus and the catheterization of the frontal

sinus, but its removal is the key to the conservative intranasal treatment of many cases of frontal sinuitis where, to establish free drainage, the removal of the anterior part of the middle turbinated body is demanded. This operation, so necessary in many cases before the drainage can be established, is accomplished by the use of the cold wire snare. The loop is passed either above or below the body. (See Fig. 1.) When it is impossible to grasp the turbinated body with the snare a notch may be cut in the inferior or anterior border in which

FIG. 3.



Left lateral wall of nasal chamber, with anterior end of middle turbinated body removed. The catheter is introduced into the frontal sinus through the atypical passage, which lies a little in front and to the median side of the infundibulum.

a, a. Infundibulum opening into a frontal cell.

b. Typical opening of anterior ethmoid cells.

the loop will catch. The Hartmann conchotome is often found to be a better instrument than the snare for this operation.

With the middle meatus freely opened there are three methods of determining the presence of pus in the frontal sinus:

1. The introduction of a catheter and irrigation of the sinus.
2. The diagnosis by exclusion.
3. Making an exploratory opening into the sinus.

Catheterization of the Frontal Sinus. To be successful in catheterizing the frontal sinus one must be familiar with the anatomy of the middle meatus by the study of sagittal sections of the nose on the cadaver. In the typical formation the anterior end of the infundibulum opens into the nasofrontal duct.¹ The direction of the passage is from below upward and forward. (See Fig. 2.) In the atypical formation the ostium frontalis has nothing to do with the infundibulum, but opens at a point a little in front and to the median side of the hiatus semilunaris. The direction of the passage here is almost straight upward. (See Fig. 3.)

The first step in catheterizing the frontal sinus should be to anesthetize the middle meatus with cocaine. Before attempting to introduce the catheter it is best to determine the exact location of the orifice and the direction of the passage with a flexible blunt-pointed probe. Bending the probe one inch from the tip at about a right angle upward, try to introduce it by the typical passage through the infundibulum upward and forward. (See Fig. 2.) If this fails, try to reach the sinus through the direct or atypical passage at a point in front and a little to the median side of the hiatus semilunaris, changing the curve of the probe to suit the direction of the passage in the particular case.

The obstacles one encounters in passing a sound into the frontal sinus are, in the first place, the conditions which obstruct the middle meatus—namely, a curvature of the septum narium, the anterior end of the middle turbinated body, and masses of mucous polyps about the meatus. After the resection of the middle turbinated body, and with the middle meatus freely open, there are still obstacles which sometimes interfere with the passage of a sound. A very narrow infundibulum or an excessively large bulla ethmoidalis may obstruct. In some cases the infundibulum opens into a frontal cell and does not enter the sinus. (See Figs. 3 and 4.) In other cases where the infundibulum opens into the frontal sinus there are one or more cells, the so-called frontal or infundibular cells, which open into its anterior end. (See Fig. 2.) It may readily happen that the probe enters one of these cells in place of the frontal sinus. In such cases, where the frontal sinus opens into the infundibulum together with other cells, the opening into the sinus, as a rule, lies nearest the septum narium. (See Fig. 2.) To avoid entering a frontal cell the rule is to bend the tip of the sound slightly inward. To introduce the sound in a difficult case often requires many trials at repeated sittings. In other cases, especially in the atypical formation

¹ The application of the term ductus nasofrontalis to the passage leading from the frontal sinus into the nose, if taken to signify the existence of a canal having a certain length between the opening into the sinus and that into the nose, is not strictly correct, for often, especially in the atypical formation of this passage, no true duct exists, but the frontal sinus opens into the anterior part of the middle meatus through a simple ostium.

of the passage, one is often surprised at the readiness with which the sound slips into the sinus.

The introduction of a catheter is a comparatively simple matter after the location and direction of the passage have been determined by the probe. A soft silver catheter is bent to correspond exactly to the curve of the probe, and is introduced at the point where the probe had entered.

FIG. 4.



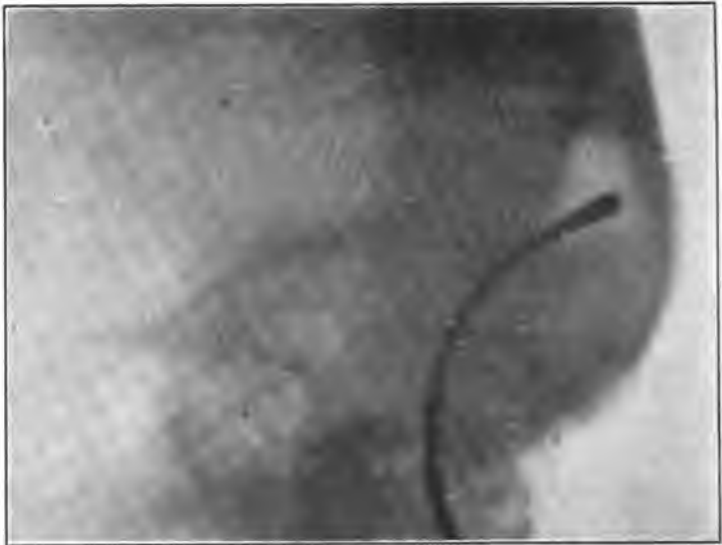
Right lateral bony wall of nasal chamber, showing Infundibulum opening into an anterior ethmoid cell.

To ascertain whether the catheter has actually passed into the frontal sinus is sometimes quite difficult. The ability to feel the instrument scraping against the anterior wall of the sinus has been reported by Engelmann in four cases.¹ I have never been able to verify this. The method of measuring the distance the catheter has been intro-

¹ Arch. f. Laryng., Bd. I., S. 812.

duced into the nose, and of placing it for comparison along the side of the nose, may be deceptive, for the course of the catheter within the nose is oblique, while on the outside it is more vertical, and the impression is readily given that the instrument has passed into the frontal sinus when the tip has really lodged in the infundibulum or in a frontal cell. (See Figs. 2 and 3.) To ascertain the position of the point of the probe, Struycken¹ has used a compass after magnetizing the point of the probe. To determine whether the catheter has passed into the frontal sinus I have for several years used the X-ray, with satisfactory results.² There is a possibility for error even when using the X-ray.

FIG. 5.



Skiagraph showing position of catheter in frontal sinus.

The catheter may enter a large frontal cell (Fig. 2), the bulla frontalis of Zückerkandl, in which case the X-ray might give the same result as if the frontal sinus had been entered. We can in some cases be sure that the catheter is not confined in a frontal cell when we are able to rotate the tip freely, for this shows that it has lodged in a large sinus. *In cases where this experiment fails we have no means of determining whether the point of the probe is in the sinus or a frontal cell.* The skiagraph (Fig. 5) shows the position of a catheter in the frontal

¹ Arch. f. Laryng., Bd. II., S. 154.

² The application of the X-ray for diagnosing conditions in the frontal sinus was suggested by Max Scheler in 1897 (Arch. f. Laryng., Bd. VI., S. 57). In 1899 Gustave Spleen used it for directing a burr while opening the sinus from the nose (Ibid., Bd. IX., S. 285). Glatzel, in 1900, used the X-ray for locating the position of a probe in the frontal sinus (Ibid., Bd. XI., S. 155).

sinus. This skiagraph was taken of a man who had suffered for many years from purulent discharge from the nose. After the resection of the anterior end of the middle turbinated body and the removal of a large number of polyps from the middle meatus the catheter was introduced and a large quantity of fetid pus was washed out of the sinus. With the catheter once in the sinus it is a simple matter to irrigate the cavity and examine the water for the presence of pus. A single negative result cannot be considered conclusive, but repeated examinations must be made before the possibility of a latent suppuration can be excluded.

Diagnosis by Exclusion. The number of cases where, for anatomical reasons, the catheter cannot be passed into the frontal sinus is greatly reduced by the resection of the anterior end of the middle turbinated body. There are still, however, certain cases where the catheter cannot be introduced. In these cases the method of making a diagnosis by exclusion may be tried.

The maxillary sinus and the anterior ethmoid cells as well as the frontal sinus open into the middle meatus. Pus found in this meatus may have come, therefore, from one or all of these cavities. To determine whether a part or all of the pus comes from the frontal sinus we proceed first to eliminate the maxillary sinus. This is done by irrigating the sinus either through its normal opening or by introducing a curved trocar just above the inferior turbinated body. The point of the trocar should be directed down and outward in order to avoid the orbit.¹ After the sinus has been washed out the nose is thoroughly cleansed with a pledget of cotton. The patient is now allowed to wait ten or twenty minutes. If pus is again found in the middle meatus it must come either from the anterior ethmoid cells or the frontal sinus. The opening into the anterior ethmoid cells, placed in the angle between the bulla and the base of the middle turbinated body (Fig. 3), is separated far enough from the ostium frontalis so that pus coming from the former can often be distinguished from that coming from the opening of the nasofrontal duct.

In latent empyema the discharge from the frontal sinus is often intermittent, due to a partial obstruction of the duct or to the slight amount of pus secreted. In such cases, therefore, where the pus does not reappear after cleansing the nose a piece of dry gauze may be packed about the ostium frontalis and left there for several hours, or even overnight. Upon its removal, if any pus has exuded from the frontal sinus, the gauze will be marked with it. A negative result, just as when catheterizing the sinus, cannot be considered conclusive until repeated examinations show its absence. In making a diagnosis by exclusion,

¹ Study of the Anatomy of the Maxillary Sinus, with Special Reference to Points of Practical Interest. G. E. Shambaugh, Chicago Medical Recorder, 1898, vol. xiv. p. 398.

suppuration from the frontal cells which open into the infundibulum will give the same results as that from the frontal sinus.

Exploratory Puncture. In cases where the sinus cannot be catheterized, and where the method by exclusion fails to give satisfactory results, we have still at our command, as a last resort, a means of arriving at a diagnosis by making an exploratory opening into the sinus. An intranasal opening has been made by Schaeffer by boring with a narrow, sharp spoon through the floor of the sinus, between the middle turbinated body and the septum. The opening must be made well in front, to avoid breaking through the cribriform plate into the brain cavity. Others have directed a trocar from the middle meatus upward and outward into the sinus. Spiess¹ has used the X-ray for guiding a burr through the floor of the sinus. The danger of entering the brain cavity in puncturing the sinus from the nose is so great that few specialists care to undertake it. An unfortunate case is related by Grünwald,² where a Parisian specialist, suffering from what he supposed was an acute attack of frontal sinuitis, attempted to open the sinus himself through the nose, and died four days later from meningitis.

When the symptoms demand an exploratory opening of the frontal sinus the safest method is to make the opening from without. It requires but a small incision, which can be made under the inner end of the eyebrow, where the slight scar will not be seen. It is well to get the consent of the patient to complete the operation of cleaning out the sinus in case pus should be found.

CAUSES OF SALPINGITIS OTHER THAN GONORRHOEAL.

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AND

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IN approaching this subject two phases present themselves, the surgical and the sociological. From either point of view the subject is of interest, and the sociological is of no less importance, if not of more, than is the surgical. There is an idea very prevalent among the laity, due perhaps to a careless manner of speaking among the medical profession, that salpingitis and pyosalpinx have their origin alone in gonorrhœa. This conviction is so deeply rooted in the minds of the laity, and in a great measure in the minds of physicians, that it is difficult to alter.

¹ Loc. cit.

² Die Lehre von den Nasenerkrankungen, München, 1896, p. 132.

It is true that the great majority of cases of salpingitis are of gonorrhœal origin, but yet from a sociological stand-point the recognition that there are other causes is most important.

To so great an extent is this view prevalent that many women who have this trouble are grossly misjudged, and their disease is considered one for which they are in great measure responsible, and, therefore, not deserving of the sympathy that would otherwise be accorded them.

The general recognition that there are causes of salpingitis other than gonorrhœal is especially important to innocent husbands and unmarried women, for in many instances are these members of society done a great injustice in the minds of the community. It may be said in reply, that the existence of other causes is well known; but yet, in spite of this knowledge, we think that we are justified in holding that there is a careless manner of thinking and speaking in the profession that is capable of doing a great deal of harm.

Perhaps we have gone out of our way to call attention to the sociological aspect of this subject, yet, nevertheless, we believe it is of great enough importance to be worthy of our attention.

Just how great a proportion of the cases of salpingitis and pyosalpinx are of gonorrhœal origin is very difficult to determine. The proportion has been placed by various eminent authors all the way from 20 to 80 per cent. Such a wide discrepancy in their conclusions is in itself enough to show how difficult is the true percentage to determine. At the time these cases come to operation the disease has usually been long established, and cultures, as is well known, commonly show no growth, or, if successful, a doubt remains as to whether the micro-organism found is the one which caused the trouble or is only present as a secondary infection. The difficulty of growing the gonococci also greatly interferes with our statistics.

Under the microscope the tubercular cases can be differentiated, and in some cases the gonorrhœal can be separated from those caused by the other pus-producing micro-organisms. This latter differentiation, however, can only be surmised rather than definitely and certainly determined.

The causes of salpingitis other than gonorrhœal that have been especially called to our notice are post-puerperal infection, appendicitis, and tuberculosis, and we may add, as an existing cause of acute exacerbations, minor gynecological operations. These causes are operative in frequency, probably in the order named, and we will briefly discuss each of them.

One of the most important causes of this trouble is what may be well called the "terrible douche habit." The douche is often a remedial agent of great value, and if properly and not too frequently used is entirely harmless; but its indiscriminate and careless use is greatly to

be deplored. By its too constant use the vagina is robbed of its natural protection against infection, and carelessness in asepsis often implants an infection of the utmost gravity. We believe that there is hardly one of our readers of any considerable experience in either a general or a gynecological practice who has not seen at least one case of endometritis or salpingitis that he has attributed to the unsurgical use of the douche. Personally, so many of these cases have been met with that we are afraid of a fountain syringe, and feel it our duty to warn our patients against this apparently harmless procedure. The careless use of the douche in one instance in our knowledge was responsible for a severe gonorrhoeal infection in an entirely innocent woman. In a reprehensible manner the douche was allowed to hang in the bathroom and was used by a servant who had a virulent gonorrhoea, and its subsequent use by her mistress resulted in her contracting the disease.

Minor gynecological operations are responsible for many acute exacerbations of latent disease of the tubes, and it is our opinion that the question whether a cervix should be repaired or dilated, or a uterus curetted, calls for as much or more experience than any other operation in gynecology. A dilatation of the cervix for the cure of dysmenorrhoea or sterility is done often with practically no thought of the dangers; but in too many instances this slight operation is followed by the most distressing consequences. Besides the element of danger in these cases the chances of cure are so uncertain that a dilatation should not be done without the most careful consideration. All authorities give salpingitis as a contraindication for these procedures, but the great difficulty lies in definitely determining that a moderate latent salpingitis does not exist.

Some years ago the practice of introducing a sound into the uterus for methods of diagnosis and uterine applications of Churchill's tincture of iodine and other drugs was much in vogue in office practice. Of late years intrauterine applications in the office have become much less popular with the majority of practitioners, on account of their dangers as well as their lack of efficiency. During the past year there have been published, by German surgeons, elaborate technique of intrauterine treatment for use in the office; one man even going so far as to advocate curetting in the office, and allowing the patient to go home in an hour or two afterward. Already there are beginning to appear in American medical journals reports of series of cases treated by these methods. This we deplore as a step backward and not forward in medicine, for if our modern experience in aseptic surgery teaches anything, it should teach that a woman with a piece of gauze in the uterus, or immediately after a dilatation and curettement, is in too great danger of infection to permit her to go about on the streets or carry on the usual vocations of life.

Post-puerperal infection is undoubtedly responsible for many cases of salpingitis, and calls to our minds the trite statement that if it were not for bad obstetrics there would be very little gynecology. The post-puerperal cases may develop directly after labor, or after a long interval. In the latter cases an endometritis alone develops at the time of labor, which may be mild in character and occasion very few symptoms, but which subsequently results in a salpingitis.

We hardly dare say in the majority, but certainly in a large percentage of cases of appendicitis in the female is the right Fallopian tube infected. Indeed, in a case of periappendicular inflammation it is hard to see how, from its anatomical position, it can escape becoming infected. Not only are the two structures so close in their anatomical relations, but also the communication by their lymphatic channels is so free that the transmission of infection from one to the other is to be expected. In many cases there is direct lymphatic and vascular connection by means of the appendiculo-ovarian ligament of Claudio. Through this free lymphatic connection we sometimes find the right tube and ovary seriously involved in mild grades of appendicitis, making the primary cause of the salpingitis hard to explain but for the knowledge of this connection.

Tubercular salpingitis is occasionally met with and arises in two ways, either secondary to tuberculosis in other parts of the body, or from direct implantation from tuberculosis of the seminal vesicles, testicle, or prostate. In forty consecutive cases of salpingitis subjected to microscopical study at the German Hospital tuberculosis was found but once. Of course, this is not a large enough series of cases to determine with any degree of accuracy the percentage of tubercular salpingitis, yet it is our opinion that it does not occur much more frequently than our figures would indicate.

AN EXPERIMENTAL INVESTIGATION OF PUERPERAL PYÆMIA.

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WHEN we follow up the investigation of the development of pyæmic diseases it is, without doubt, only by the progress of bacteriology, by its enlarged and improved methods of examination, that clearness, system, and experimental confirmation has been infused into vague, unsatisfactory ideas and suppositions.

Kehrer, in his thesis on *Diseases of Childbed*, says, in paragraph 89, that pyæmia in puerperio is due to a phlebitis of the placental

veins or the veins of the wall of the uterus. Occasionally it originates from inflamed veins of the vagina or of the external genitals—the vena cruralis or saphena. From these primary sources of infection pus or thrombi containing pus pass into the bloodvessels and cause those secondary symptoms which are characteristic of pyæmia.

This theory of the origin of pyæmic infection is generally accepted, and will probably remain so for all time. But what kinds of bacteria cause pyæmia, and what are the conditions upon which the different degrees of pyæmic infection depend? It is upon these two points that opinions are divergent.

Some years ago the writer proved, in "A Contribution to the Study of Septic and Pyæmic Infection of the Uterus," that the origin of pyæmia and sepsis is not due alone, first, to the staphylococcus, and, second, to the streptococcus, and that these cocci are not confined to certain definite places of entrance into the system, but that each of these two kinds of bacteria, no matter whether they penetrate by lymph-vessel or bloodvessel, or by both at the same time, are able to cause pyæmia or sepsis. This is apparently directly in opposition to the result of the histological study of puerperal endometritis by Bumm, who in all his experiments found only the streptococcus. The apparent contradiction of these two results is due, undoubtedly, to accidental occurrences, and proves most conclusively that neither pyæmia nor sepsis is caused from only one of these bacteria. Kehrer had previously warned against the theory that the streptococcus is the only bacteria which causes puerperal fever. He, on the contrary, believes this fever to be caused by both staphylococci and streptococci. Hahn confirms this by his paper on "Post-mortem Diagnosis of the Septic and Pyæmic Processes." Out of fifteen fatal cases of sepsis and pyæmia he could find both streptococci and staphylococci as the cause of the puerperal infection.

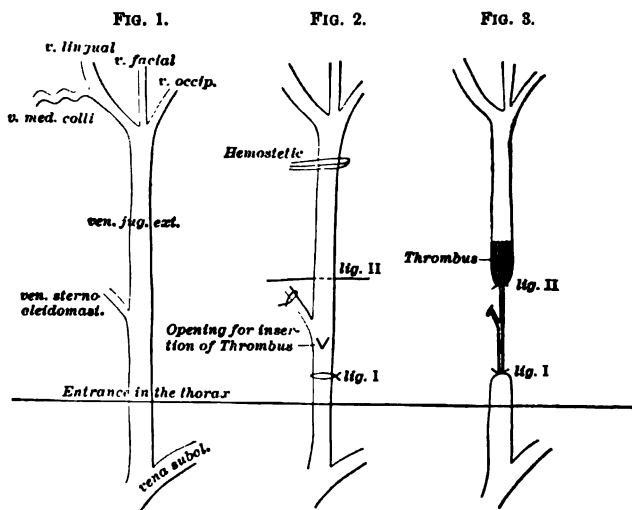
Preiyer adds to this result, in his bacteriological investigations of some cases of puerperal fever, by finding in the pus of seven cases of puerperal pyæmia both streptococci and staphylococci, but principally the latter. Mixius confirms the same, and proved, by cultivation, staphylococci and streptococci in the pus taken from different organs, and also in the blood of six fatally terminating puerperal fever cases. Rubecka also, in his "Contributions to the Pathogenesis of Purulent Puerperal Diseases," obtains the same result. From a series of further publications—Cornil, Babes, Levy, etc.—in which they name only the staphylococcus or streptococcus as being present in pyæmia, I select only that of Baginsky, because it best illustrates how cautious one must be about accepting the majority of the reports of "found" bacteria. The story is best told in the author's own words: "The bacteriological examination was done according to Koch's method. In the test-tubes was placed

agar-agar which had been infected directly from the corpse; on this, after having been two days in the incubator at 35° C., there were little whitish substances, resembling pearls somewhat. They were in groups or ridges, which were only raised a very little from the surface of the cultivation mass. The microscopical examination showed a pure culture of a small, round coccus, which was easily stained by fuchsin, gentiana, and Gram's method. In another test-tube of the same infection there were, after twenty-four hours' exposure, numerous colonies, which looked like very small pearls, and besides these there was a white, straight line, elevated above the surface a little. This line of bacteria represents the place where the infection was made with the platinum loop. In the condensed water of this tube a whitish mass was easily perceived. In all the cultures the microscopical examination proved successful, the result each time being to find cocci formed into chains and heaps." He who practices bacteriology even in a small way must find this way of investigation doubtful, to say the least; because it is a real mistake, according to our knowledge of bacteriology to-day, if a fluid which is to be examined for its germs is not mixed with agar or gelatin and spread on a plate. In an agar-culture made in a test-tube by merely infecting it with a platinum loop one never succeeds in isolating different germs, which are always to be presupposed in a bacteriological investigation. If Baginsky, in spite of this, had found a unitary growth of both cultures, even then he would not have proved that one germ did overgrow another; but, as Baginsky describes in the second culture, a growth which is not characteristic of the streptococcus, but rather of the staphylococcus, because in his further microscopical investigations he found little heaps of cocci, as well as little chains, there is no doubt that Baginsky presented a mixed infection of two cocci as a unitary one. Although the theory that the staphylococcus and streptococcus are the causes of pyæmic diseases predominates almost exclusively, yet contrary theories may only be repudiated by reasons gained from experience. The experimental proof of this theory is still lacking. It therefore shall be my task to advance the proof that the origin of pyæmic diseases is, indeed, independent of the immigration of different kinds of pus germs; that it is sufficient if either staphylococcus or streptococcus, or both at the same time, develop their inflammatory properties.

The first step toward advancing this task was the question, "How may we produce the same symptoms in animals that appear in the pyæmic infections in man?" In other words, how is it possible to create by embolic processes infectious thrombosis, phlebitis, and supuration in tissues adjoining the entrance of infection, and in more distant organs?

Judging from the knowledge that the cause of a pyæmic infection in

puerperio generally is due to an infectious thrombus of a vein of the placenta, and that after operations pyæmic symptoms take the same course as puerperal diseases, I tried the following experiment: I brought an artificial thrombus charged with pus cocci into a vein of the test animal, without infecting the surrounding tissue. In all my experiments I used the vena jugularis externa of dogs and rabbits as test animals. The technique for the three kinds of thrombi infected and charged with streptococcus or staphylococcus, single or united, was the same, and may therefore be described in advance, and thus avoid the description of each infection of each animal. The vena jugularis sinistra of all the test animals was laid open from the place of confluence of the facial and cranial veins to the thorax, where the vena jugularis was ligated permanently, while the place where the facial and



cranial veins flow together was closed only temporarily by a hæmostat. The branches of this hæmostat were protected by rubber tubes, to avoid injuring the walls of the bloodvessel. The artificial thrombus was placed one-half inch below the ligature. In rabbits this is above the inosculation of the vena mediana colli and the jugularis externa; in dogs, just below the inosculation of the vein coming from the musculus sternocleidomastoideus. This latter vein is also ligated permanently. A sterilized silk thread was now placed under the vein, the vein opened, the thrombus pushed in peripherally, and the vein reclosed with the same silk thread. In removing the hæmostat at the temporary ligature the vein was instantly filled with blood up to the thrombus. The external wound was now carefully closed with silk. This entire operation was performed aseptically, all disinfectants being avoided.

The selection of the material for the artificial thrombus was attended with great difficulties. First, I tried thrombi from agar-agar infected in small glass tubes of the same diameter as the vena jugularis. After filing off the bottom of the small test-tubes the agar-agar thrombus was pushed into the open vein by a sterilized glass rod. Here I was obliged to discontinue the use of this material, because the agar-agar was absorbed too quickly, and thus a main momentum of the nature of a thrombus was absent. The natural thrombus, as a solid mass before its final softening, keeps the bloodvessel filled with blood for a certain length of time; but the quick absorption of the agar-agar in connection with blood did not resemble the reality. To avoid this disadvantage the entire test-tube (one inch long), charged with agar-agar culture, was sterilized half an hour each day for three successive days at 60° C. and then inserted in the vena jugularis. But the advantage of this solid artificial thrombus was offset by the disadvantage that the part of the infected agar-agar which came in contact with the blood was too small to have a proper infectious result. After some further fruitless experiments to find a proper material for the artificial thrombus I finally found that a round cotton wick of the same diameter as the vena jugularis would answer the purpose best. Pieces of wick one-third of an inch long were first carefully sterilized, then charged with a bouillon culture of pus germs, and dried in an exsiccator over H_2SO_4 . To protect these pieces of charged wick from any outside infection, and to render their insertion in the vein easier, I gave them a thin coating of gum arabic. Dried once more in an exsiccator, they became as hard as stone. To prove whether these artificial thrombi were free from any other germs than those used for infection, several were selected from the series, put in bouillon, and kept for twenty-four hours in a thermostat at 37° C. Plate-cultures spread with this bouillon placed under aerobic and anaerobic conditions and microscopical examination revealed their purity. Further experiments with these thrombi proved that their composition was a successful one, and their effect came nearest to the one of a natural thrombus. Having completed these preliminary investigations, I started with the main experiments to demonstrate that puerperal pyæmia is solely due to infectious thrombi, no matter what kind of pus germs are in question.

EXPERIMENT 1. Large, full-grown dog. Thrombus with staphylococcus pyogenes aureus from fresh carbuncle pus inserted in the left vena jugularis externa nine hours after its completion.

Five days later dog refuses to eat; has an unsteady, tottering gait; three days later dog was found dead.

Autopsy. Vena jugularis externa sinistra ligated one-half inch above the entrance in the thorax. Centrally from this ligature the bloodvessel contains fluid blood; peripherally the vein is obliterated up to the second ligature. Starting at the second ligature a two and one-half inch long

softened thrombus up to the inosculation of the vena lingualis, facialis and occipitalis. From here on the veins again contain fluid blood. Other organs normal. In the median third of the sulcus centralis of the left hemisphere of the cerebrum was an abscess, the size of a hazelnut, which destroyed the pia mater of the lobus parietalis and frontalis and penetrated into the brain substance of the lobus parietalis and frontalis. Pia mater, about one-half inch in the circumference of this abscess, inflamed. Dura mater normal. From this abscess, from the pulp of the spleen, from the heart's blood, and from the softened thrombus, staphylococcus pyogenes aureus could be proven in pure culture, microscopically, and by plate-cultures. The thrombus was carefully hardened in alcohol, and together with the walls of the veins and the surrounding tissue examined according to Gram's method and alum carmine. Result: Walls of the vein thickened, infiltrated, and partially necrotic; vasa vasorum filled with staphylococcus; thrombus consisted of detritus containing staphylococci and a few red and white blood-corpuscles; surrounding tissue infiltrated, necrotic, and populated by numerous staphylococci. The microscopical examination of the hardened spleen showed that some lumina of its bloodvessels were filled with staphylococci.

It would become too tiresome for the reader if all our eighteen experiments were told as minutely as Experiment 1. The writer, therefore, thinks it sufficient when he reports in general the course which each one took:

Insertion of infected wick thrombus; reports of symptoms which the animal shows after operation up to its death, which occurred on the tenth day after the insertion, with chloroform; in all cases the animal did not die earlier. Examination of the pus germs found in thrombi, etc., upon their identity with cocci used for infections by plate-cultures and microscopically. Autopsy: Report of thrombus found in vena jugularis externa, whether perivascular inflammation and pus formation occurred; report of embolic abscesses in different glands, etc.; state of different organs. Thrombus with vein, surrounding tissue and spleen hardened in alcohol, and sections stained with alum carmine and Gram's method; microscopical examination of sections.

From the tabulated compilation may be learned what kind of animal the writer used, the kind and origin of germs, how often they passed through agar-agar before use, how soon the artificial thrombi were used after their completion, their identity with the germs originally employed for infection of the inserted wick thrombus, and, finally, the result of the infection ten days after insertion of the thrombi.

From our experiments, and especially from their tabulated compilation, we learn that the results of the insertion of thrombi in the vena jugularis externa are the same for staphylococcus pyogenes aureus and streptococcus pyogenes alone or when combined. Thrombosis of the vena jugularis externa is common to all cases; now more, now less extended; now confined only to the main branch of the jugularis; now

spreading over a large number of the veins of the head and face; a thrombosis the thrombus of which is either entirely softened, or shows only signs of decay, or produces itself as a clear thrombosis, with or without any transformation by bacteria.

In all the cases a phlebitis was connected with the thrombosis, the intensity of which depended upon the degenerative changes of the thrombus.

In Experiments 3, 7, 10, and 14, in which the thrombus underwent no degeneration, for reasons which I will explain later, there was a mere thickening of the walls of the bloodvessel, with more or less infiltration of it and the surrounding tissue. In all the other cases where the substance of the thrombi passed through all the steps of a purulent softening the inflammatory changes of the walls of the blood-vessels were accordingly increased. Dense infiltration of the thickened wall of the vein and of the surrounding tissue with pus-corpuscles, necrotic decay, and suppuration are now the common pictures. These, stained by Gram's method, show cocci varying greatly in number. In other words, we have macroscopically and microscopically a pronounced thrombophlebitis purulenta, the true companion of a pyæmic infection.

The accurate microscopical examination of these eighteen cases gives the writer a certain privilege to participate in the discussion of Metschnikoff's theory of phagocytosis. Just by these microscopical slides, concentrated in a small and easily controllable space, which were at my disposal, the facts to be disclosed were: The effect of bacteria, the changes made by bacteria, and, above all, that arrangement of protection which Metschnikoff supposed existed in the consuming of the bacteria by leucocytes. Not in a single case, nor in one of the numerous cuts of any case, could it be observed that a coccus was enclosed in the cells of the innumerable leucocytes. On the contrary, it appeared to the writer, after the present and previous observations, that the leucocytes were eaten by the bacteria—i. e., that the bacteria caused their decay.

This is a long known and forcible contradiction of the theory of phagocytosis—that hand-in-hand with the multiplication of the bacteria within the zone of the leucocytes there is also a decaying of these bacteria; it is just in the detritus where the most bacteria are noticed. This decaying effect of bacteria in the leucocytes was proven experimentally by Loewit. Our microscopical observance is therefore in the best accordance with the natural process, which I will explain later.

Although the writer, as a result of his microscopical examinations, is placed on the side of the opponents of Metschnikoff's theory of phagocytes, he will yet admit, with other authors, that a certain value has to be conferred to the leucocytosis in infectious inflammation. But this advantage does not lie in a mere mechanical effect, as Bumm

thought to be able to prove—that the “rampart” of leucocytes around the centre of an inflammation is impermeable to bacteria (the writer himself, in a previous communication, proved this to be wrong, by directly contrary results from examination)—but, according to Buchner, in a chemical effect.

Above all, the bacteria, after their content becomes nascent by decay of their cells, act attracting—i. e., chemotactically on the leucocytes, as we learn from the experiments of Leber, Buchner, Janowski, etc.; then they act as solvents—leucolytic—on the same; and, finally, perish in their own composition. (Baumgarten, Hoenfeld, and others.)

The latest efforts of Metschnikoff to sustain his theory of phagocytosis are also not effective enough. If Metschnikoff quotes against the main opponent of his phagocytosis—against the theory of the microbicide power of the blood-serum—that the same was only studied in diphtheria and tetanus (two diseases where the resorption of the locally produced poison is prevalent), and not on septicæmic diseases, the writer needs only refer to the experiments of Reichel, Brieger, Frankel, Everard, Szekely, and Szana in order to disarm this allegation; for not only at septic general infections, but also especially at pyæmic local infections, it is confirmed beyond any doubt. By the microscopical examination of Experiments 3, 7, 10, and 14, I am further able to support the theories of Virchow and Aschoff, in opposition to the French school, that thrombosis is the primary process and phlebitis the secondary one. The writer could recognize without any difficulty in the above cases that the farther and fresh end of the thrombus was still surrounded by a normal wall of the vein, while already, a few millimetres distant from it, thickening and infiltration of the blood-vessel occurred. Thus this result of examination again is an affirmation for the fact that the progressing thrombosis of a bloodvessel is not the consequence of an inflammatory change of the wall of the bloodvessel, but independent from it, due only to a chemical process of the blood itself.

Because in none of our eighteen experiments there could be found any embolic-pyæmic processes in the lungs the writer failed to confirm the remarkable presence of hyaline emboli in the ramifications of the arteries of the lungs, especially in cases of specific fatal character as are described from Litter, Heller, and Deutsch. The obstructions of the bloodvessels in the spleen which were observed in our fatally terminating cases—1, 6, and 8—are of a more bacteritic nature and caused by the same cocci which were used for the infection.

Resulting from thrombophlebitis purulenta we note in all the eighteen cases perivascular inflammation from serous saturation extending to purulent reduction of the tissue. Not only staphylococci and streptococci alone, but also both combined, produced those changes of

tissue which in pyæmic diseases find their cause in the wandering of the cocci through the wall of the bloodvessels and their penetration in the surrounding tissue.

Embolic abscesses and suppuration of glands reach in our eighteen cases the percentage of 44.5 per cent., of which three ended fatally—17 per cent. The transportation of these emboli occurred into different organs. In four cases there was parotitis, and in three others the glandula submaxillaris sinistra suppurred; in one case there was an abscess in the left hemisphere of the cerebrum, in one case a meningitis purulenta, and in one an abscess behind the left ear—processes which do not permit any other explanation than of an embolic origin, because in all these suppurations the same cocci as those used for infection of the artificial thrombi could be proved as a pure culture. In the three mentioned fatal cases there could still be found a general infection of the blood, since in the blood of the heart and in the pulp of the enlarged spleen there could be cultivated the same cocci as there were in the embolic abscesses and in the thrombus of the vena jugularis externa.

Transferring these results of investigation to pyæmia in puerperio, we come to the conclusion that the transformation of the physiological formation of thrombi into a pathological one, as in thrombophlebitis purulenta, is principally due to the presence and effects of pus germs. The recent theory of the coagulation of the blood furnishes a decided confirmation to the above results of our experiments, and the writer therefore takes advantage of quoting its principal points, which are of importance in order to understand the statement of the deleterious effect of the cocci.

The thesis of Alexander Schmidt, the founder of the theory of the coagulation of the blood—"without leucocytosis no coagulation of the blood"—experienced an extension: "The colorless blood-corpuscles are not the only cause for coagulation of the blood, but they accelerate in an eminent degree, by their extracorporeal effect, a process already in operation; this process would be brought to a termination also without the colorless blood-corpuscles, by the disengagement of the fibrin in the blood separated from the organism;" because the filtered, from all corpuscular elements eliminated, blood-plasma of the horse also coagulates, though a little slower than the blood which contains cells. This is not at all in contradiction with the theory of Pekelharing and Lilienfeld, which will be quoted later, because Alexander Schmidt admits that the constituent elements for coagulation which are present in the blood in a dissolved condition derive themselves from cells and come from there in the blood.

Schmidt derives fibrinogen by a process of dissolution from a body higher than albumin—from cytin, the radical of the protoplasma; but Schmidt was only able up to now to transform the cytin, by dissolution,

into cytoglobin and preglobulin, and by co-operation of these two into paraglobulin. Fibrinogen, which he derived from paraglobulin only speculatively, is just this constituent element for coagulation which Schmidt was unable yet to obtain directly from paraglobulin.

Still more hypothetical is the derivation of fibrin ferment, the "thrombin," which is said to develop from another constituent of cells, the prothrombin by the influence of zymoplastic substances. Schmidt admits but recently that the elementary bodies which are present in each cell may act in plasma free from ferments, either promoting or hindering the coagulation. The latter substance he found in his cytoglobin. The same observations of Schmidt we already meet in a publication of Wooldrige, in the year 1890, who found that fibrinogen of tissue, injected intravenously or intra-arterially, caused at first an increased and then a diminished capability of coagulation of the blood. Wright, who extended Wooldrige's experiments, could analyze his "tissue-fibrinogen" into two albumoses—a protein which hinders coagulation, and a nuclein which favors it.

The investigations of Pekelharing "About the Importance of the Lime Salts for the Coagulation of the Blood" explains, further, "that the fibrin ferment—the 'zymogen' of Pekelharing—is a chemical compound of lime, which is able to transfer lime to the fibrinogen, so that from the soluble fibrinogen there is originating an insoluble lime-like compound of albumin, the fibrin."

Pekelharing found in this compound of lime and albumin as an organic component a nucleo-albumin, which he also could educe from it, and which originates by decay of the leucocytes. These give up their plasma to the nucleo-albumin and combine with the lime which the plasma contains.

A valuable confirmation of this theory of coagulation are the experiments of Loewit about the decay of the leucocytes—the leucolysis, by intravenous and intra-arterial injections of hæmialbumose, peptone, pepsin, nuclein, extract of leeches, pyocyanin, tuberculin, curare, urea, uric acid, and urate of sodium. Of these substances the albuminoids, inclusive of the proteins of bacteria, proved the most effective leucolytic ones. Exceptionally these injections brought about thrombosis, but the same could be produced on a large scale by the injection of several cubic centimetres of a 1 to 2 per cent. CaCl solution in the venous system after having injected the albuminoids.

Arthur and Pagis proved that blood mixed with small quantities of oxalate of lime—by which chemical process the lime in the plasma precipitates—becomes uncoagulable. With an admixture of chlorcalcium this blood, free from lime, coagulates again. Hence the fibrin is a lime-albuminate. Also, Freund accedes to the same opinion, that the formation of insoluble phosphate of lime has to be looked upon as

the essential and exclusive cause of coagulation of the blood. Griesbach, too, agrees that coagulation of the blood depends upon the plasmochysis—i. e., the decay of leucocytes in combination with calcareous substances.

It cannot be denied, says Griesbach, that there is indeed calcium oxide present in the plasma, as Pekelharing supposed; but it cannot be overlooked that, also, the amœboid cells—this at least tallies with the blood of *astacus fluviatilis* and of the invertebrates—are abounding in calcium oxides, and that, therefore, the products of decay of cells represent already compounds of calx and albumins which were formed either very quickly or in which the calcium atom was present *a priori*.

The chemical process of coagulation of the blood becomes still more obvious by Lilienfeld's "hæmatological searches." Out of a water extract of leucocytes (thymus, lymph glands) Lilienfeld could precipitate by acetic acid and alcohol a substance of constant compound—the nucleo-histon—which, again, by addition of mineral acids is decomposed into nuclein and a substance free of phosphorus, "the histon," already formerly discovered by A. Knopel in the red and white blood-corpuscles of birds.

This nuclein, even as the thrombin of Schmidt or as the nucleo-albumin of Pekelharing, promotes coagulation in all fluids capable of coagulation, and intravascularly injected produces wide-spread thrombosis.

Histon has the property of an albumose, and therefore intercepts coagulation, and that in such an exclusive way that in blood gained by bleeding and mixed with histon the morphological elements are, after twenty-four hours, still as unchanged as in circulating blood.

Nuclein and histon, acting together as nucleo-histon, are also hindering coagulation. Nucleo-histon can be analyzed not only by mineral acids, as mentioned above, but also by lime-water; and it is just this analysis in histon, its compound with calx and the consequently nascent nuclein, which causes coagulation, that the process of coagulation is based upon.

Hauser supports the theory of coagulation of Schmidt by his histological investigations. He could prove in cuts of the lungs of a child who died of diphtheria, and in several other cases, delicate nets of fibrin, with numerous central points. These latter ones were formed by closer nets of fibrin in whose centre a blood-corpuscle or tissue-corpuscle could be recognized. Hauser found the same picture also in some thrombotic bloodvessels. He therefore thinks that the ferment for coagulation becomes nascent from the decaying cells, and that consequently round these cells fibrin will be deposited.

The continuation of the physiological thrombosis of the placental bloodvessels, the thrombophlebitis purulenta, which originates from

infection of the physiological thrombus with pus germs, has to be explained as follows: The proteins of the cocci present in the infected thrombus of placental veins act at first chemotactically (Buchner, Leber, Pomorski, etc.) in gathering a large quantity of leucocytes; then they are plasmochitic and leucolytic. By this decomposition of the leucocytes the nucleo-histon which is contained in the cells is becoming nascent. Is the system not any more able to eliminate the nucleo-histon, the same will be analyzed, by the presence of calx in the plasma of the blood, in nuclein (which promotes coagulation) and histon (which delays it). The latter one, forming a compound with calx, becomes ineffective and leaves the whole field of operation to the nuclein, the only originator of coagulation.

Hence it is that where no leucolytic substance is present—this exists in a mere ligated vein—also no coagulation or thrombosis follows. The theory of Brücke, that the walls of a bloodvessel *in vivo* possess certain qualities adverse to coagulation, can therefore be laid aside; and, indeed, the writer could prove, what Zezeck ere now had published in his "Contribution to the Physiology of the Circulation of the Blood," that the blood remains fluid at a plain ligature of arteries or veins—under avoidance of infection—above and below the ligature.

The writer had the same experience with two control experiments which he performed with sterilized non-infected thrombi, in order to obviate the objection that thrombosis of a bloodvessel could be produced by insertion of any sterilized foreign body in the vena jugularis externa. A sterilized, non-infected wick thrombus was therefore inserted in the vena jugularis externa of a full-grown dog and rabbit. Ten days later the same wick thrombi were both, without reaction, grown into the veins of the animals. The thrombi were then cut out and put in bouillon for control, to see whether an accidental infection occurred; but the bouillon remained sterile. The blood above and below the artificial thrombus in both cases was found fluid.

As mentioned, in each single case of our eighteen experiments the cocci of the pyæmic thrombi, the pus of the perivascular and embolic abscesses, were examined upon their identity with the germs used for the infection of the respective artificial thrombi, which caused abscesses, etc.; and in all eighteen cases the identical cocci could be proved not only by plate-cultures, but also by microscopical examination of the fresh pus.

Hence, by imitation of the natural cause of pyæmia the writer obtained the same phenomena which are characteristic for this infectious disease—thrombosis of the infected vein in the various stages of softening; phlebitis from the mere infiltration and thickening to the entire necrosis of the walls of the bloodvessel; seropurulent inflammation, with formation of phlegmons in the surrounding tissue; embolic

suppuration of the glandula submaxillaris and parotis; embolic abscesses in different parts of the caput and in the cerebrum, and, at last, general infection and death of the animal.

Jordan claims, in his publication upon "Acute Osteomyelitis," that, first, the quantity, and, second, the virulence of germs is decisive for the result of invading and tissue-destroying bacteria. He deducts the first requirement of a successful action of bacteria from experiments made by Watson Cheyne and Herman. Watson Cheyne claims 250,000,000 of staphylococci for bringing about a mere abscess in rabbits, and Herman concludes, from his numerous experiments with animals, that the coefficient for a subcutaneous suppuration is 520,000,000 in rabbits and 52,000,000 in dogs. Bumm found in his experiments as to the importance of pyogenous micro-organisms for progressive suppuration, that it is very difficult to equal the same results in animals with those in man unless the entire system of the animal were inundated with the respective bacteria.

Since the writer, with his infected thrombi, obtained the same pyæmic symptoms in dogs or rabbits as there are in man, he was very curious to learn how many germs were necessary for it, and especially whether there was a difference in the quantity of germs in comparison with the different quality of infection.

For this purpose he cut pieces 1 mm. long from different series of our infected wick-thrombi, ground them in 1 c.cm. of bouillon, mixed the same with agar-agar, poured it on sterile culture-plates, and kept them in the thermostat at 37° C. After forty-eight hours the number of germs grown on these plates varied from 80 to 150, which makes for the entire thrombus a total number of about 800 to 1000 cocci.

There is an enormous difference between the results of the experiments of Watson Cheyne, Herman, Bumm, and the writer's. But it cannot change anything in the fact that our infected wick-thrombi, although they only contain a number of cocci from 800 to 1000, caused, indeed, pyæmic symptoms in dogs and rabbits of the same intensity as in man. For that very reason we can conclude that it is not the quantity of pyogenous micro-organisms from which depends the intensity of an infection, but their virulence.

Before all, it will be of interest for us to find out the cause of the difference of this virulence in our eighteen cases. From columns 1, 2, and 4 of our synopsis we cannot perceive anything. Whether dogs or rabbits, whether streptococci or staphylococci, or both combined, were used for our experiments, or whether the cocci passed through agar-agar only once or several times before their employment—all this did not have any effect on the intensity of the pyæmic infection; also, Knorr found that streptococci did not lose any of their virulent properties even after their sixtieth passage of cultivation.

| I. | II. | III. | IV. | V. | VI. | VII. |
|-------------------------------|--|--|------------------------------------|--|---|--|
| Animals. Experi- ments. | Species of cocci. | Place where cocci were found. | Passage of cultiva- tion. | Duration of preser- vation of com- pleted thrombi before in- sertion. | Identity of germs before and after insertion. | Result of insertion of artificial infected wick-thrombi in vena jugularis externa. |
| 1 Dog | Staphylo- coccus pyogenes aureus. | Fresh car- buncle pus. | 1 | 9 hours. | Same. | Death after eight days. 4½ cm. long puriform softened thrombus; abscess in cerebrum; enlargement of spleen; general infection. |
| 2 Dog | " | " | 1 | 6 w'ks. | " | 8 cm. long softened thrombus; perivascular, sero-purulent inflammation. |
| 3 Dog | " | " | 1 | 8 w'ks. | " | 2 cm. long not softened thrombus; perivascular serous inflammation. |
| 4 Rabbit | " | Pyæmic liver ab- scess. | 3 | 14 hours. | " | 3 cm. long softened thrombus; suppuration of gland, submax. sin.; abscess from ligat. il. size of hazelnut; enlargement of spleen. |
| 5 Rabbit | " | " | 3 | 3 w'ks. | " | 2 cm. long thrombus in beginning softening; perivascular sero-purulent inflammation. |
| 6 Rabbit | " | Carbuncle pus. | 6 | 6 hours. | " | Death after five days; 4 cm. long softened thrombus; suppuration of parotis; perivascular sero-purulent inflammation; enlargement of spleen. |
| 7 Rabbit | " | " | 6 | 9 w'ks. | " | 2 cm. long thrombus; perivascular serous inflammation. |
| 8 Dog | Strepto- coccus pyogenes | Erysipelas. | 1 | 9 hours. | " | Death after 7 days; 4 cm. long softened thrombus; suppuration of parotis; meningitis purulenta; enlargement of spleen. |
| 9 Dog | " | " | 1 | 6 w'ks. | " | 2½ cm. long softened thrombus; suppuration of gland, submax. sin.; perivascular sero-purulent inflammation. |
| 10 Dog | Strepto- coccus pyogenes | Erysipelas. | 1 | 8 w'ks. | " | 2 cm. long thrombus; perivascular serous inflammation. |
| 11 Rabbit | " | Parame- tritis exudativa. | 4 | 14 hours. | " | 5 cm. long softened thrombus; suppuration of parotis; perivascular sero-purulent inflammation. |
| 12 Rabbit | " | " | 4 | 3 w'ks. | " | 3½ cm. long thrombus with beginning softening; perivascular sero-purulent inflammation. |
| 13 Rabbit | " | Furuncle pus. | 6 | 6 hours. | " | 3½ cm. long softened thrombus; phlegmon from lig. il. toward median line of neck. |
| 14 Rabbit | " | " | 6 | 9 w'ks. | " | 2 cm. long thrombus; perivascular serous inflammation. |
| 15 Dog | Strepto. & staphylo- cocci. | Pus from abscess in arm. | 1 | 9 hours. | " | 4½ cm. long softened thrombus; suppuration of parotis; embolic abscess behind left ear; enlargement of spleen. |
| 16 Dog | " | " | 1 | 6 w'ks. | " | 3 cm. long thrombus with beginning softening; perivascular serous inflammation. |
| 17 Rabbit | " | Parame- tritis exudativa. | 4 | 14 hours. | " | 6 cm. long thrombus with beginning softening; suppuration of submaxillaries; phlegmon toward median line of neck. |
| 18 Rabbit | " | " | 4 | 5 w'ks. | " | 4½ cm. long thrombus with beginning softening; perivascular sero-purulent inflammation. |

Entirely different, however, stands the comparison of our cases in column 3—"place where the pus germs originated from"—and column 5—"duration of preservation of artificial thrombi from the time of their completion until their insertion in the vein." In column 3 we note a distinct difference in the virulence of the same cocci among themselves, and not, as one supposed, of the different species of cocci in comparison with each other. Staphylococci from carbuncle pus used immediately after their obtaining caused fatal pyæmic symptoms; whereas the same species of cocci obtained from a pyæmic abscess created again pyæmic symptoms, but with less intensity and without fatal termination. The same proportion existed between streptococci obtained from erysipelas and ordinary abscesses.

In column 5 at last we perceive a very distinct difference in the violence of the different pyæmic symptoms. The greatest effect of the inserted thrombi was noted, independent from the place where the cocci used for infection were found, if the same were inserted in the vein of the animals directly or only a few hours after their completion. (See Experiments 1, 4, 6, 8, 11, 13, 15, and 17.) The longer, however, the completed thrombus was preserved in its dry condition the less was the virulence of infection. (See Experiments 2, 3, 5, 7, 9, 10, 12, 14, 16, and 18.) The virulence degree of staphylococci and streptococci is therefore in inverse proportion to their preservation in a dry state.

Although we know from Axel Holst that desiccated bouillon cultures of streptococci lose their virulence, and from Hausman and Fröhlich that staphylococci undergo the same loss of virulence by a dry, protracted preservation, an accurate experimental confirmation of this long known theory was still lacking. The writer, therefore, is confident he has proved by his experiments the fact that an abode of cocci in a dry state—i. e., a loss of their water molecules—is extremely detrimental to their virulence, and in time abolishes it entirely. That this loss is due to a chemical destruction or ineffectual transposition of the albumin of the bacteria, and not to a mere decay of the same, is proved from the fact that the infected wick-thrombi, the cocci of which became ineffective by too long a preservation, healed in the veins of the two control animals without any reaction. Even the chemotactic working proteins were absent—a substance which, according to Buchner, could be obtained only by boiling the bacteria for hours in 0.05 per cent. liquor potassi.

The questions which the writer asked in the preface, Whether pyæmia is due to a special species of pus germs, and what reason causes a different vehemence of a pyæmic infection, may be answered at the conclusion of this paper, as follows:

It did not make any difference whether staphylococci or streptococci alone, or both together, were used for infection of our artificial cotton-

wick thrombi; the insertion of the same in the vena jugularis externa of eighteen animals caused uniformly pyæmia, with its characteristic symptoms—phlebitis, suppuration in the surrounding tissue, embolic abscesses in submaxillaris, parotis, cerebrum, and general blood-poisoning and death of animals.

The intensity of the pyæmic infection depended:

First, from the place where the employed cocci were found (staphylococci from carbuncle pus and streptococci from erysipelas proved the most poisonous ones, no matter how many passages of their cultivation they experienced).

Second, from the duration of the preservation of the completed artificial thrombi before their insertion in the vena jugularis externa (the longer staphylococci and streptococci were kept in a dry state the greater was the loss of virulence, no matter how virulent the cocci proved at their first examination).

REPORT OF A CASE OF HEMIANÆSTHESIA OF OVER EIGHT YEARS' DURATION, RESULTING FROM DESTRUCTION OF THE CARREFOUR SENSITIF AND LENTICULAR NUCLEUS WITHOUT DIRECT IMPLICATION OF THE OPTIC THALAMUS.¹

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THE optic thalamus was first regarded as a sensory centre many years ago. Carpenter's theory, as given by W. H. Broadbent,² in 1866, was that the thalamus is the organ of conscious sensibility, to which all impressions made on peripheral sensory nerve fibres must be transmitted in order to be recognized as sensations; and that the corpus striatum is the organ or instrument of voluntary motion—the downward starting-point of volitional motor impulses, or, it might be said, of all cerebral motor impulses. This is very different from the views now held regarding the thalamus. The theory that the thalamus is a sen-

¹ Read at the meeting of the American Neurological Association, June, 1901.

² The British and Foreign Medico-Chirurgical Review, January to April, 1866, vol. xxvii., p. 468.

sory centre, as held in 1866, was not founded on careful anatomical investigations, and erroneous views were entertained at that time regarding the course of the sensory fibres; indeed, the course of these fibres is not fully known, even at the present time.

Broadbent also looked upon the thalamus as a sensory centre, as did Hughlings Jackson. The latter, writing in 1866, said: "Hemiplegic paralysis especially is common, but hemiplegic anæsthesia is very rare. Both, however, occur together from disease of the same centre of the brain, viz., the optic thalamus," and he reported a case as proof of this statement. In his case the degree of loss of sensation was so unusually great, according to his experience of chronic cases of hemiplegia, that he diagnosed disease limited to the optic thalamus. He had carefully ascertained during the patient's life that sensation was impaired in the region of all the sensory branches of the fifth nerve, which supply the skin, and also in one-half of the trunk as well as in the arm and leg; but sensation was not lost, nor was it so much impaired that pinching was not painful.

His description of the necropsical findings is as follows: "The disease was not strictly limited to the thalamus. At the autopsy I cut through it from within outward, beginning at about the line of the posterior commissure. This incision went through the remains of a clot. The diseased part did not extend to within a quarter of an inch of the commissure, nor to within about that distance of the geniculate bodies.

"In front of the incision the quantity of the centre disorganized would be equal to a small hazelnut; outward *the disease extended through the small tongue of corpus striatum, which curves round the outside of the thalamus, and thence up to the gray matter of the convolutions of the Sylvian fissure*" [italics ours]. As this was not a chronic case, there is no way of deciding how much of the loss of sensation may have been due to pressure on sensory fibres outside of the thalamus and how much to injury of the thalamus; and Jackson's¹ description seems to indicate that the *carrefour sensitif* may have been implicated.

The *carrefour sensitif*, as defined by Verger,² comprises the last third of the posterior limb of the internal capsule in the most inferior portion of the opto-striate region.

Those who accepted the views of Meynert believed in the existence of a sensory tract in the external portion of the foot of the cerebral peduncle, and the supposed continuation of this tract in the posterior part of the internal capsule gave the foundation for the theory of the

¹ Clinical Lectures and Reports by the Medical and Surgical Staff of the London Hospital, 1866, vol. III, p. 373.

² Archives Générales de Médecine, 1900, vol. II.

carrefour sensitif. Charcot,¹ in 1876, said that only a lesion of the internal capsule caused symptoms permitting an exact cerebral localization. A lesion at the posterior third of this part was supposed to give rise to anæsthesia implicating the general sensation as well as the special senses. This lesion, if it extended a little further forward, caused hemiplegia. Below the position of the *carrefour sensitif*—i. e., in the cerebral peduncle—a lesion was not supposed to cause disturbance of the special senses as well as of the general sensation. The same may be said of a lesion above the *carrefour sensitif*.

The form of hemianæsthesia with disturbance of the special senses is now quite generally regarded as hysterical; but though Meynert's views relative to a sensory tract in the external bundle of the foot of the cerebral peduncle can hardly be accepted at the present day, the *carrefour sensitif* is still held by many to be the location of the sensory fibres. Some believe that the median lemniscus, in whole or in part, in passing to the cerebral cortex has a situation in the internal capsule behind the motor fibres; others, while acknowledging that the median lemniscus terminates in the optic thalamus, believe that the *carrefour sensitif* contains thalamo-cortical sensory fibres.

The views of Dejerine are directly in opposition with these ideas, and he has expressed them many times. It is not possible, according to him, to admit the existence in the posterior segment of the internal capsule of a region for sensory fibres only. These sensory fibres are intimately mingled in the internal capsule with the fibres of projection. Hemianæsthesia of general sensation, of central origin, can exist only when a lesion in the optic thalamus destroys the terminal fibres of the median lemniscus and the cortico-thalamic fibres; or when the connections of the thalamus with the sensori-motor cortex are destroyed, even though the thalamus may be intact; and in the latter case the lesion is always very extensive. Hemianæsthesia is much more likely to be persistent when the thalamus is injured.²

Probably the most recent statement of Dejerine on this subject is made in the second volume of his anatomy of the nervous system, which, when translated, reads: "When one examines the observations of capsular hemianæsthesia with autopsy reported until the present time, one recognizes that in the immense majority of cases the optic thalamus was implicated together with the posterior segment of the internal capsule; and in the very rare cases in which the lesion of the thalamus is not indicated one cannot assert that this ganglion was intact, because in all these observations the localization was made with the naked eye and on fresh material. On the other hand, there exist observations of lesions of the posterior segment of the internal capsule with integrity of

¹ Comptes rendus de la Soc. de Biologie, 1876, p. 254.

² Dejerine and Long. Comptes rendus de la Soc. de Biologie, 1898, p. 1174.

the thalamus, and in these cases the patients were simple hemiplegics without anæsthesia. We ourselves have observed two very positive cases of this character studied by the method of serial microscopical sections."¹

Long,² holding very much the same views as Dejerine, believes that the sensory fibres coming from the spinal cord pass to the inferior and external portion of the thalamus, and that sensory thalamo-cortical fibres pass by the internal capsule, but not in an area posterior to that occupied by the motor fibres.

The external bundle of the peduncle, according to Dejerine, is formed by a band of fibres that comes from the middle portion of the second and third temporal convolutions, and enters the retrolenticular portion of the internal capsule in the hypothalamic region. It is not a sensory tract, because it degenerates downward and terminates in the pons.

The views of Verger³ on the subject of the position of the sensory fibres are of interest. He says that the conclusions of Dejerine and Long concerning the localization of lesions causative of hemianæsthesia may be considered exact, as they are in harmony with facts revealed by a study of normal anatomy and anatomico-clinical investigation; and it is easy to demonstrate that they are applicable to all cases with autopsy published under the name of capsular hemianæsthesia. The capsular localization, in the narrow sense as was formerly understood, he regards as manifestly incorrect; and he cites a case published by Long (Case X.) as proof of this statement, since in this case a circumscribed lesion of the internal capsule, he says, caused some sensory disturbances, although the *carrefour sensitif* was intact. The sensory disturbances, however, were not pronounced.

In order that a case may be of value in reference to the question whether a lesion confined to the *carrefour sensitif* may cause hemianæsthesia; or whether the lesion must implicate the whole of the posterior limb of the internal capsule, because of the mingling of sensory with motor fibres, according to Dejerine; or whether the lesion must implicate the thalamus in order that hemianæsthesia may exist, it is necessary that the hemianæsthesia should have existed a long time—more than a few weeks at least—and therefore was not merely a symptom at distance; that the lesion should have been of such a character as to cause little pressure on surrounding tissue, therefore not a tumor; and that only one of the three areas—the *carrefour sensitif*, or the posterior limb of the internal capsule, or the thalamus—was much injured. Such a case, of course, is exceedingly rare. In Verger's Cases XXX. and XXXI. the lesions were recent. In his Case XXXII.

¹ A. and J. Dejerine. Anatomie des Centres Nerveux, Tome deuxième, Fascicule 1, p. 257.

² Les voies centrales de la sensibilité générale.

³ Archives Générales de Médecine, 1900, vol. II.

the hemianæsthesia persisted only about a month, and was due to a hemorrhagic focus in the postero-external part of the thalamus implicating slightly the middle portion of the posterior segment of the internal capsule.

Long¹ reports two cases of central lesion of the brain without hemianæsthesia (Cases X. and XII.). In one case the posterior segment of the internal capsule was destroyed and the optic thalamus was intact; but in the detailed report of this case he says the lesion did not extend to the retrolenticular segment of the internal capsule, and this is also clearly shown by his drawing. This case would seem to show that destruction of the internal capsule does not cause intense hemianæsthesia. In the other case the lesion implicated only a part of the posterior limb of the internal capsule and only the anterior and internal nuclei of the thalamus, leaving the external nucleus intact as well as the region of the *carrefour sensitif*. In regard to his Case X., in which the posterior limb of the internal capsule was so seriously implicated, it was difficult, Long says, to explain the absence of pronounced sensory disturbances, because the thalamo-cortical fibres were probably destroyed by the lesion of the internal capsule, and yet sensation was little affected.

The hemianæsthesia from central lesion of the brain, as shown by all cases with necropsy, Long says is never absolute, even in the first weeks following the onset of the hemiplegia. The organic hemianæsthesia described by Verger² is principally an anæsthesia of the extremities. If the trunk is implicated it is not as much so as the limbs, and the upper limb is more anæsthetic than the lower. In the limbs the anæsthesia is more marked in the distal parts than at the root of the limbs, and it diminishes gradually as parts of the limbs nearer the trunk are examined. The borders of the anæsthesia may vary slightly from time to time, but not so much as Long believes. This anæsthesia is really incomplete; it is merely hypæsthesia, and contact is felt, but not so keenly in the affected limbs as in those of the other side of the body. The qualitative change in perception of tactile sensation is greater than the quantitative, and the patient cannot distinguish the rubbing of a finger from that of a piece of wood or cloth, and he is unable also to localize the area touched. Hypalgesia is less common than hypæsthesia, but the power to localize the pain produced is often affected. Temperature sense is less often implicated. Dejerine³ has expressed views similar to these in many particulars.

The following case which seems of much importance in regard to the lesion causing central hemianæsthesia was presented *intra vitam* by one of us (Dr. Dercum) before the Philadelphia Neurological Society on

¹ Loc. cit.

² Loc. cit.

³ Semaine Médicale, July 6, 1899, No. 32, p. 249.

January 23, 1899,¹ and seems to be the first case to demonstrate clearly that a lesion of the *carrefour sensitif* and lenticular nucleus without implication of the optic thalamus may cause hemianæsthesia. The history was as follows:

CASE I.—J. B., male, aged forty-nine years, mulatto, laborer, was admitted to the Philadelphia Hospital, November, 1892.

The family history is unimportant and unsatisfactory. Father died at forty-eight, cause unknown; mother died of "dropsy and asthma" at middle age. One brother and one sister are dead, cause unknown.

Personal History. He had various diseases of childhood: measles, chicken-pox, mumps and whooping-cough. He has had no illness of moment, except an occasional cold, until he had an attack of paralysis about 1892. This attack he describes as follows: He "suddenly fell fast asleep while at work." A fellow-workman shook him and he awoke to find his right arm, his right leg, and the right side of his face paralyzed. He could not move the right arm and right leg, nor did he have any feeling in them; and he could not talk. He recovered his speech in about two months, and motion in the arm and leg in about three months. Sensation, however, has never returned in the right side. He denies a venereal history, but acknowledges that he has used both alcohol and tobacco.

Through an inadvertence at the time the case was presented, the word "left" was used instead of "right" in speaking of the hemiplegia and the hemianæsthesia. It was the *right* side which was involved. There was present in 1899 a spastic hemiplegia of the right side, moderate in severity, together with a moderate degree of contracture of the right arm and spastic rigidity and exaggeration of the knee-jerk of the right leg. There was a very slight involvement of the right side of the face. The hemianæsthesia was well defined by the middle line of the body and appeared to be everywhere complete. Right-sided homonymous hemianopsia was associated with this hemianæsthesia. There was no impairment of hearing upon the right side, nor was there any involvement of taste or smell. The sensory symptoms were limited to those concerned in the various forms of cutaneous sensibility and to the hemianopsia.

The hemianæsthesia was studied repeatedly and while intense it presented the following peculiarities: It was noted that the results varied slightly at various examinations. It was found for instance that upon several occasions the patient would respond when the trunk upon the hemianæsthetic side was tested or when the proximal parts of the limbs were tested, that is, the upper arm and shoulder or thigh and hip. It was noticed also that occasionally he would respond when the right side of the face below the eye and above the mouth was touched. At most of the examinations, however, no responses were obtained in these situations. At different times also it was noted that the line defining the hemianæsthesia from the side with normal sensation varied somewhat, at times retreating a little from, and at other times advancing toward, the sound side. In the distal portions of the limbs, however, such as the forearms and hands and legs and feet, no variations in the tests were ever observed. The hemianæsthesia appeared to involve equally all the

¹ Journal of Nervous and Mental Disease, vol. xxvi., No. 4.

forms of cutaneous sensation. Occasionally upon the trunk the patient made attempts to differentiate between hot and cold, but the answers were more frequently incorrect than correct.

The reflexes were as follows: The knee-jerk upon the right side was exaggerated; upon the left side, normal. There was present a pronounced front tap reaction upon the right side; none upon the left. There was no ankle clonus upon either side. The tendon reactions in the right arm were exaggerated while those in the left arm were absent.

The diagnosis was made of a lesion in the left hemisphere involving especially the posterior third of the posterior limb of the internal capsule, together with the fibres of the optic radiation; some involvement of the anterior two-thirds of the posterior limb was also considered probable. It was further remarked that cases of hemiplegia, even of organic origin, with persistent hemianæsthesia, are comparatively rare; that hemianæsthesia and hemianopsia, while not infrequently observed during an apoplectic seizure or immediately subsequent to a seizure, commonly disappear at a relatively early period; that the sensory phenomena as a rule prove to be distance symptoms, and that actual lesions of the posterior portion of the internal capsule and optic radiation as a result of apoplexy are of rare occurrence.

On May 24, 1900, the patient developed a nephritis which persisted until his death. On January 6, 1901, he suffered from a fresh apoplectic seizure. The attack came on during the night, and the left arm and the left leg were involved. The paralysis was not, however, complete, and he was able to move the limbs, though slightly. The physical examination revealed a mitral systolic murmur and some roughness of the second sound. The lungs were resonant throughout, though there was some dulness posteriorly on the right side. He was conscious, but developed Cheyne-Stokes' respiration. On the morning of January 9th, he was still conscious but continued to have Cheyne-Stokes' respiration. At noon he became suddenly unconscious. Upon raising the left arm and left leg, they fell as though completely paralyzed. The right side, the seat of the old hemiplegia, was very rigid, however, more so than before. Marked œdema of the lungs now developed and the patient died twelve hours later.

A clinical report of this case was published by Drs. C. K. Mills and G. E. de Schweinitz¹ in 1896. Dr. de Schweinitz found right lateral quadrant anopsia (1896), whereas in Dr. Dercum's clinical report (1899) the man is said to have had right-sided homonymous hemianopsia. The report of Drs. Mills and de Schweinitz is as follows:

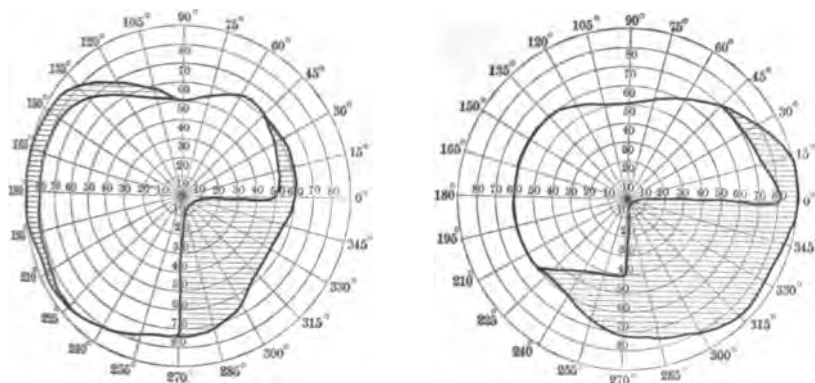
"CASE II.—*Right lateral quadrant anopsia, absence of Wernicke's symptom; dyslexia; right hemiparesis; partial right hemianæsthesia; partial word-deafness and word-blindness.*—J. B., aged forty-two years, a laborer, May 3, 1892, had an apoplectic attack with loss of speech, and was taken to the Hospital of the University of Pennsylvania, where he remained until November, 1892, when he came to the Philadelphia Hospital. He improved slowly. During his stay in the University Hospital, he noticed when he grasped an object with his right hand that he could not release it as promptly as when he seized it with his left.

¹ The Philadelphia Hospital Reports, 1896, vol. III.

"Examination showed that he was paretic in the right arm, and his field of vision toward the right was decreased. He says that at times he could not understand what was said to him, especially if the person speaking stood to his right. Examination showed paresis of the right forearm, partial right hemianæsthesia, tongue slightly tremulous on protrusion, pointed and deviating a little to the right. He had gradually recovered his speech.

"From the first he says he knew what he wanted to say, but could not put it into such words as would be understood by his hearers. He could recognize objects, but could not pronounce their names. He could not read writing or print because everything ran together; he recognized the letters, but could not pronounce them. He understood sentences, but could not repeat them. He had almost completely recovered from his aphasia, although his voice was a little thick and difficult to understand. One of the most marked phenomena was that which has been described as dyslexia.

FIG. 1.



Visual fields taken in 1896, showing right lateral quadrant anopsia.

"The examination of his eyes gave the following results:

"*R. E.*—Oval disk, shallow, dish-like excavation, almost complete. Vessels about normal in size; no fundus lesions.

"*L. E.*—Small, nearly round disk, and small physiological cup. Vessels almost normal in size.

"Pupils equal and round; mobility of iris normal; no hemiopic pupillary inaction.

"*Fields.*—Right lateral quadrant anopsia."

It is perhaps wiser not to make any extended comments on the condition of aphasia, as the statements relative to this were made by the patient. It is important to note that although an extensive subcortical lesion of the left temporal lobe existed, the patient had regained his speech.

Post-mortem Record.—Body of a well-developed man, showing considerable post-mortem rigidity. Some oedema of hands and ankles.

Chest well formed, abdomen slightly distended. Right arm shows some atrophy, hand partially flexed on forearm.

Upon opening abdomen a thin layer of subcutaneous fat is noted. Distention of stomach and intestines is seen. The appendix is about 11 cm. ($4\frac{1}{2}$ inches) post-cæcal, and points toward the liver. The liver extends about 1 cm. ($\frac{1}{2}$ inch) below costal arch.

Left pleural cavity is free. Right pleura shows adhesions, posterior at lower lobe and apex. Adhesions were old.

Both lungs show extensive oedema and congestion.

Spleen measures $10\frac{1}{2} \times 8 \times 3$ cm. ($4\frac{1}{2} \times 3\frac{1}{2} \times 1\frac{1}{2}$ inches). Congested and firm.

Left kidney measures $11\frac{1}{2} \times 5\frac{1}{2} \times 3$ cm. ($4\frac{1}{2} \times 2\frac{1}{2} \times 1\frac{1}{2}$ inches). Capsule strips with some difficulty, leaving a rough, uneven surface. The kidney substance is normal. Some congestion; small infarcts.

Right kidney, $12 \times 5\frac{1}{2} \times 3$ cm. ($4\frac{1}{2} \times 2\frac{1}{2} \times 1\frac{1}{2}$ inches), shows advanced interstitial nephritis. Small cysts under capsule.

The heart shows extensive hypertrophy, especially of left ventricle. Right ventricle is dilated and only slight hypertrophy. Heart weighs 630 grammes. The coronary arteries show extensive atheroma; this involves the anterior arteries especially. The heart muscle is somewhat fibrous. The anterior and posterior mitral leaflets present a few sclerotic patches. Valves are competent. Other valves normal.

The left adrenal normal.

The liver appears small, and the surface is smooth and edges are short. On section the liver is congested.

Pancreas is normal.

Intestines are congested, as is also the stomach. No other pathological changes of these organs are found.

The brain membranes show considerable oedema. A recent hemorrhage is found in the right cerebral hemisphere in the region of the lenticular nucleus. An old cyst is present in the left cerebral hemisphere and is described below in detail.

Pathological Diagnosis.—Edema and congestion of lungs. Hypertrophy of heart. Atheroma of coronary arteries. Interstitial myocarditis. Interstitial nephritis. Cerebral hemorrhage and cerebral cyst.

When the lateral ventricles of the brain were opened by a horizontal cut just above the upper surface of the optic thalami, the caudate nucleus on the right side was found destroyed by a recent hemorrhage, the right optic thalamus was intact and presented a strong contrast with the optic thalamus of the left side, which was shrivelled in its posterior portion. No cyst was apparent on the left side at this level. The left optic thalamus on its superior surface measured antero-posteriorly 3.3 cm. ($1\frac{1}{8}$ inches); the right 4.5 cm. ($1\frac{1}{2}$ inches). The left optic thalamus in transverse diameter in its widest portion on its upper surface measured 1.8 cm. ($\frac{3}{4}$ inch); the right in a corresponding portion measured 2.5 cm. (1 inch). To the naked eye the left optic thalamus appeared to be about one-half the size of the right thalamus on its upper surface. The anterior part of the left optic thalamus was rounded and convex, but the posterior portion was sunken and concave. In the photograph (Fig. 2) the piece of tissue behind the left optic thalamus is a part of the hippocampal gyrus. A horizontal section made through the left optic thalamus at the part where a crack is seen in the photograph (Fig. 2), cutting off only the anterior

superior portion of the left thalamus, revealed a condition represented in Fig. 3. A cavity, probably resulting from an old hemorrhage, 2.8 cm. (1½ inches) in depth from above downward, and 2.5 cm. (1 inch) in its longest diameter at the level, represented in Fig. 3, was found in the extreme capsule, claustrum and external capsule of the left side. The

FIG. 2.



Photograph showing the optic thalami. A. Portion of hippocampal convolution; B. Atrophied portion of left optic thalamus; C. Line showing the level at which the horizontal section represented in Fig. 3 was made.

FIG. 3.



Photograph showing the upper portion of the cyst in its relation to the optic thalamus. A. Cyst situated chiefly in the lenticular nucleus; B. Posterior limb of the internal capsule; C. Atrophied portion of the left optic thalamus, shown also in Fig. 2 at B; D. Line showing the level at which the horizontal section represented in Fig. 3 was made; shown also in Fig. 2 at C.

left lenticular nucleus appeared to be almost destroyed and the extreme posterior part of the posterior limb of the internal capsule and the optic radiations were involved. The anterior half of the posterior limb of the internal capsule was not implicated in the cyst. The anterior limb of the internal capsule was entirely normal. The optic thalamus was not

implicated at all in the cyst, and the external medullary lamina of the thalamus was distinct and normal in appearance. The anterior tubercle of the left thalamus was relatively larger than the external nucleus, and seemed to be of about normal size. A transverse cut made 1.5 cm. ($\frac{3}{4}$ inch) below the level represented in Fig. 3 and passing through the hypothalamic region showed the posterior limb of the internal capsule apparently fully normal. The lenticular nucleus, except in its extreme ventral portion, was destroyed.

The portion of the thalamus that showed atrophic change was the pulvinar, and this alteration was the result of the destruction of the optic radiations. The left optic tract was much smaller than the right,

FIG. 4.



Photograph of a microscopical section. *A.* Anterior part of the lenticular nucleus; *B.* Anterior limb of the internal capsule; *C.* Head of the caudate nucleus; *D.* Posterior limb of the internal capsule; *E.* *Carrefour sensitif* degenerated; *F.* Cyst.

and the external geniculate body was not distinct. The left mammillary body was one-third or one-half smaller than the right.

The basal ganglia of the left hemisphere with the parts adjoining were cut in microscopical serial sections; the result of a study of these sections is as follows:

The cyst implicated the posterior portion of the lenticular nucleus, the outer part of the posterior portion of the posterior limb of the inner capsule, the area of the *carrefour sensitif*, and the optic radiations. The greater portion of the posterior limb of the internal capsule was intact. The optic thalamus was not implicated at any part in the cyst. The

cyst was limited on its outer side in its upper portion by the cortex of the insula. The posterior part of the optic thalamus in its superior portion contained few nerve fibres, because of the secondary degeneration from the lesion of the optic radiations, but at a little lower level more fibres were found entering the posterior part of the thalamus. The cyst maintained the same relative position in all the sections as far as the lower part of the temporal lobe. In sections from lower levels the posterior part of the posterior limb of the internal capsule was even less implicated than in those from higher levels. The fasciculus of

FIG. 5.



Photograph of a microscopical section at a lower level than that shown in Fig. 4. A. Anterior part of the lenticular nucleus; B. Anterior limb of the internal capsule; C. Head of the caudate nucleus; D. Posterior limb of the internal capsule; E. *Corresponding sensitive degenerated*; F. Cyst.

Türk, which, according to Dejerine, comes from the second and third temporal convolutions, was not entirely degenerated, and some fibres of this fasciculus were found passing to the posterior part of the posterior limb of the internal capsule. The left external geniculate body was much smaller than the right, because of the implication of the left optic radiations. The foot of the left cerebral peduncle was much narrower than that of the right, and the external portion of the former was not so deeply stained as the rest of the foot. This external bundle was not completely degenerated, because the fasciculus of Türk was

only partially degenerated. The lemniscus on the left side was one-fourth to one-fifth narrower than that on the right side in the interolivary region. Sections of the medulla oblongata were obtained as low as the upper part of the nuclei of Burdach. The right-crossed pyramidal tract in the spinal cord was only slightly degenerated.

The hemianæsthesia in this case was such as has been observed after organic cerebral lesions, and persisted probably over eight years. While variations in sensations occurred at times on the trunk and face, they were never observed in the distal portions of the limbs. The hemianæsthesia corresponded to that produced by cerebral lesion and could hardly have been of hysterical nature. It was much more intense than the hemiparesis, and the reason for this is found in the slight degeneration of the motor part of the posterior limb of the internal capsule. The hemianæsthesia was so remarkable that the case was presented by Dr. Dercum before the Philadelphia Neurological Society in 1899 simply because of this phenomenon.

The microscopical serial sections of the left cerebral hemisphere show that at no place did the cyst implicate the optic thalamus, but the lenticular nucleus, except in its most anterior portion, and the area at the posterior part of the posterior limb of the internal capsule, known as the *carrefour sensitif*, were destroyed. The optic radiations were also implicated in the cyst, and thereby the partial blindness is explained.

This case seems to show that organic hemianæsthesia may be caused by a lesion in the *carrefour sensitif* and lenticular nucleus, without implication of the optic thalamus, except such as occurs from secondary degeneration. We emphasize the statement that in this case the inferior and external portion of the thalamus was intact. It seems to be the first case of the kind carefully studied in the literature. Whether or not the implication of the lenticular nucleus is necessary for the existence of organic hemianæsthesia we cannot determine by a study of our specimens. The integrity of almost the whole of the posterior limb of the internal capsule seems to indicate that the sensory fibres are located chiefly, if not entirely, in the area of the *carrefour sensitif*, or it may be that some sensory fibres pass through the lenticular nucleus. This view is in accord with the teaching of Edinger,¹ inasmuch as he states that a portion of the sensory tract passes through the posterior third of the internal capsule and a portion through the lenticular nucleus. The lemniscus in its interolivary portion on the left side was one-fourth to one-fifth narrower than that on the right side, and from this we conclude that the lemniscus on the left side had undergone retrograde atrophy. Some of its fibres, therefore, were probably cut in the lesion of the *carrefour sensitif* and lenticular nucleus.

¹ Vorlesungen über den Bau der nervösen Centralorgane des Menschen und der Thiere. Sechste Auflage, pp. 261, 422.

THE IMPORTANCE OF PROPER DIETARY REGIMEN IN THE
TREATMENT OF CHRONIC HEART AFFECTIONS, AND
AN ATTEMPT TO FORMULATE SOME RULES
THEREFOR.*

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THERE can be no question that the matter of diet is of much importance in the treatment of heart affections, as all the treatises, whether general or special, refer thereto in their respective sections on treatment; but that its importance is not adequately appreciated is also evident from this: that all these works content themselves with a few general phrases, a brief paragraph or two, and the subject is dismissed. So far as my reading goes, no systematic attempt has yet been made to formulate any rule or rules whereby the physician having the responsibility of such cases might guide himself.

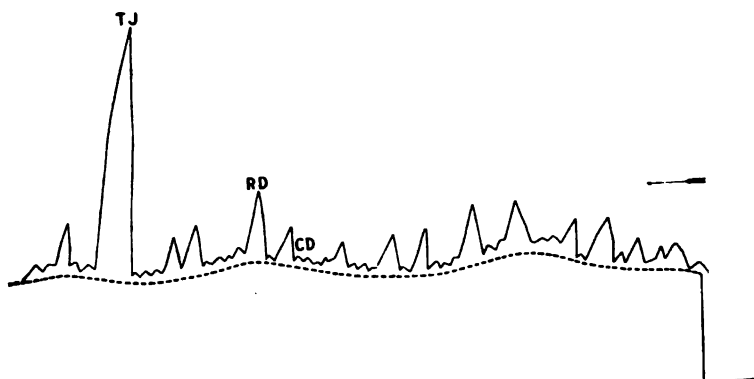
And still, if there is any one special question in the treatment of disease—diseases of the heart in particular—that deserves detailed consideration, it is this of *diet*, for in this the personal equation of the attending practitioner, his general knowledge of dietetics, his personal experience with many dishes, with the various methods of preparing food, and, lastly, a factor not to be overlooked, the state of his own digestive organs, enters more largely than in any other in the whole domain of *morbus curandi*. I remember, while working in a clinic studying the diseases of the digestive tract, asking several gentlemen who were present at the time, whether any one of them had a conception of what it was not to be able to digest well, not to be able to digest this or that article of food, not to be able to digest food cooked after a certain fashion, and all of them in one voice answered “No!” They could all digest well and everything that came along and prepared after any fashion. It is, then, not to be wondered at that very curious dietary regulations are not infrequently prescribed or that even men eminent in the profession pay but little or no attention thereto.

That the stomach is of great influence upon the heart has long since been recognized by eminent clinicians both of past and modern times. Abercrombie¹ already taught that disturbances of cardiac rhythm, even to the producing of murmur, may be caused by the stomach. Stokes² warns his readers not to be too hasty in making diagnoses of organic cardiac affections where symptoms of hepatic derangement or of gastric

* Read before the Medical Society of Greater New York, March 11, 1901.

disturbance are present ; for, he says, " it happens not rarely that with the relief of these latter conditions the heart phenomena, even the evidences of dilatation, may disappear." Huchard² and See⁴ tell us that angina pectoris may depend upon a deranged stomach, and Potain⁵ that even dilatation of the heart may have such origin.

In truth, however, it is not the stomach at all that is at fault. The stomach as an organ *per se* does not influence the heart one way or the other ; it is *what is put into it*, and not infrequently also the condition in which it is at the time to receive this ingested material that is the real factor or factors that must be reckoned with. It was to bring this out more prominently, to show the close relationship between the mode of living and the heart trouble, that in the histories recorded in my paper on " Cardiac Disturbances from Gastric Irritation,"⁶ illustrative of the various disturbances there treated of, I gave in detail the sins in eating and drinking perpetrated by these patients upon their stomachs.



Pressure curve from the fundus of the human stomach (water manometer). Read from right to left. Wavy course of the curve (punctated line) as expression of gastric contraction ; R D, respiratory pressure variations ; C D, cardiac pressure variations ; T J, deepest inspiration. (MORITZ.)

If, now, the wrong done the stomach by forcing upon it an improper diet, improper in quality or quantity, can give rise to cardiac disturbances even of so grave a character as angina pectoris or dilatation, it is not far-fetched to assume—nay, it stands to reason—that where the heart's action has been disturbed by other causes, such improper diet will keep up the disturbance ; and where the heart has been organically damaged it will not alone prevent the *vis medicatrix naturæ* from warding off, as far as possible, the consequences of such damage, but will surely hasten them on.

The heart and the stomach are in close relationship with each other. The great nerve which innervates the stomach is the same one that exerts the controlling influence over the heart, *i. e.*, the *vagus*.

Again, the heart lies in close proximity to the stomach; the former just above and the latter just below the diaphragm, the point of the heart over the great cul-de-sac of the stomach.

In his study of the passive movements of the stomach, Moritz' has found and has demonstrated graphically that certain movements are impressed upon the stomach by the heart—*i. e.*, that with each systole as the heart contracts in width and stretches out in length the point presses down upon the stomach to a degree appreciable to a recording instrument, and thus a wave of movement is created in this latter organ.

With these several points fixed in the mind we can readily understand in what ways the stomach exercises its influence upon the heart. These are:

1. By way of the vagus.
2. By contiguity.

1. BY WAY OF THE VAGUS. It has already been determined long ago by Bidder and Schmidt,⁸ and later by Schiff,⁹ that the ingested aliment is the stimulus that acts upon the intramuscular filaments of the ninth pair, and thus excites the normal gastric movements. When, now, the aliment is of an improper character—improper in quality, quantity, or mode of preparation—and this also has been well determined by long and careful clinical observation, then these nerve filaments are unduly irritated, and this exaggerated stimulus—this abnormal irritation—may spread itself locally or be conveyed upward to the nerve trunks, and along them, and be reflected upon the heart either directly (by way of the vagus and cerebrum or vagus, cerebrum, and sympathetic) or indirectly, in the way that the irritation is primarily reflected upon the pulmonary vessels,¹⁰ and the heart becomes affected secondarily only, in consequence of this. However, all this has been set forth and discussed more fully in the paper "Cardiac Disturbances from Gastric Irritation," already referred to, and requires no further comment here.

The manifestations of such undue irritation may be, in so far as the heart is concerned, (1) disturbances of rhythm, (2) painful sensations about the præcordium, and (3) dilatation of the right half of the heart.

In the article already referred to cases illustrative of various of these forms of cardiac disturbances were given in detail, and though bearing pointedly upon the question under consideration I have not held them as coming strictly within the limits of this paper or as sufficiently demonstrative for our purpose, for the reason that the stomach was there the etiological factor, and have not recalled them here. In the subjoined case the cause lay outside of the stomach, and subsequently an erroneous diet—erroneous in quality and quantity—tended not alone to keep up the trouble, but to aggravate it.

CASE I.—July 7, 1897. Mrs. T., aged fifty-seven years; married; children; woman of large frame; five feet, four inches; weight before her present illness 175 pounds, now 99 pounds. Up to seven years ago she enjoyed excellent health, and, as her economic situation was good, she had neither undue work to fatigue her nor particular cares to worry her. About this period she suffered a severe shock; fire broke out in her home one night, made rapid headway, and ere she knew it her escape was cut off. She made her way to a second story front window, and there she stood with the flames hissing and rolling about her for what seemed to her an eternity. She was finally rescued by the firemen by the aid of ladders.* From this dates the beginning of her ailment—a tendency to nervous paroxysms, with occasional palpitations having suddenly developed. Her nutrition became impaired; she grew abnormally stout, and with this her heart began to functionate badly. She was put on a reduction treatment, and, though she grew thinner, there was no improvement in either the state of her heart or her nerves. Two years ago she had an attack of influenza, and since then she has grown rapidly worse. Her heart trouble became markedly aggravated, the paroxysms becoming more frequent, more severe, and of longer duration. For the last year she has not ventured to come down from her apartment, and had not been out of the house until about a month ago, when she was removed to her summer cottage by the seaside. Here also she remains in her room and will not venture out. Her meals are served in her apartment, and she has an attendant with her day and night. She passes her time between the bed and the lounge, with brief intervals upon the rocking-chair.

She had been long under the care of a most eminent medical gentleman, and latterly a near relative, also a practitioner of prominence, was in constant attendance. At various times in the course of her sickness, particularly in the latter period thereof, she was seen in consultation by other eminent physicians of this city. So far as I could judge, the case was evidently looked upon as a hopeless one, and a "laissez aller" course pursued in general, with merely attempts at palliation when the paroxysms came on.

Status Præsens. Emaciation very marked as already related. The panniculus adiposus has entirely disappeared, and the organs beneath the abdominal walls, which are rather thin and flabby, can be readily felt. When she speaks a light hectic flush comes upon her cheeks, and the same shows upon the cuticle of other parts of her body upon slight pressure. She is very nervous, cries readily, and bemoans her fate continually. The paroxysms referred to come on every third or fourth day, so that she is hardly relieved from one when the other begins to set in. They come in this wise: She becomes restless, her heart begins to beat more quickly; her restlessness grows greater, and the rapidity of her heart's action increases until it palpitates almost tumultuously. These palpitations are, according to her attending physician, very evident to the observer, and the speed of the heart's action proven by the great increase in the number of pulse-beats. With this there is some dyspnoea, though not of marked character. A great fear, a dread of something dire impending, comes over her, her nervous

* This incident of her history I learned only after I had been in attendance on her for some time.

excitation reaches its climax ; she is almost beside herself, and wants a thousand things done at once to relieve her. She says she has noted distinct pauses in her heart's action, and this has been a source of fear to her, lest in one of these pauses the heart may stop permanently. She has uneasy sensations in her stomach, and, with them, what she describes as a "flicking" sensation, which travels from her stomach to her heart, and stands in some, to her indefinable, relation to the pause. She feels easier when she belches up, and to promote eructations takes essence of peppermint, Hoffmann's anodyne, Vichy (carbonated), etc.

She claims that her appetite is good, and she takes three good meals a day (the food being the same as is provided for the other members of the family), with some light things in between. She has been told to eat as much as she can, as it would do her good. Her bowels are constipated, rather obstinately so. She sleeps badly. She is very weak and cannot stand without support, and even then only for a few moments.

Examination. Tongue slightly coated with a white coat, and a few elevated papillæ at tip. Pulse weak, with a distinct intermission here and there. *Heart.* Superficial dulness markedly increased in its transverse extent, reaching on a level of the nipples from $1\frac{1}{2}$ cm. to the right of the right sternal line to within $\frac{1}{2}$ cm. of the left anterior axillary line. The usual evidences of an hypertrophy were not apparent on inspection. The sounds over the various valve regions are weak, but normal in character. The action of the heart is somewhat irregular ; it sounded to the ear as if the heart were turning a somersault upon itself and then righting itself again. *Stomach.* The organ is normally located ; no tenderness to percussion over any part thereof. There is some loss of muscular tone. For obvious reasons, no examination of the gastric chemismus was made. *Liver and spleen* in normal location and apparently normal. *Intestines* in usual position ; no deviation. *Kidneys* in normal position ; no mobility ; functioning normal. (Repeated examinations of her urine were made by the physician in attendance.)

Diagnosis. That the heart is dilated is very evident, but otherwise I hold it to be intact. As to the paroxysms, they are in my judgment not at all dependent upon the condition of the organ. Whether originally they were determined by the general nervousness,* or were merely a part of the general manifestations of the disturbance of nervous equilibrium, I cannot at this late date say. As I see her now, it is very evident to me that the paroxysms are greatly responsible for her nervous condition, as they keep her in a constant state of fear for her life. As regards their causation, they are due, I am certain, to some irritative influence extraneous to the heart, and from the history as given, and the examination, I locate this in the stomach.

Treatment. In accordance with the diagnosis, the treatment was mainly directed to her *stomach*, and the principal point thereof was the diet. This was so arranged that while amply nutritious, it was easily digestible and unirritating. All undue distention of the organ was avoided by proper regulation of quantity, and undue formation of gases

* It must be borne in mind that at this time the special incident recorded in the history, and which was undoubtedly of the greatest etiological importance, was unknown to me, as already noted above.

avoided by the prohibition of various articles that tend thereto. Moreover, it included both roborants and tonics for the intervals between the meals.

Medication. A digestive agent, in the form of a preparation of pancreatine, was prescribed to assist the weakened stomach in its work and to hasten the digestive act; and nux vomica and Fowler's solution were directed, the former for its beneficial effect upon the muscles of the digestive tract, the latter for its inhibitory influence on the development of flatus, and both together as general tonics.

To promote the functioning of the intestinal tract, and at the same time stimulate the general circulation, which appeared to be rather sluggish, I proposed to give her abdominal massage.*

Furthermore, I explained to the patient that it was not so much her heart as it was her stomach that was troubling her; that she was in no danger and would certainly get well; that it would hasten her recovery greatly if she went out and took the air and moved about a little bit as her strength would permit; that she would thereby promote digestion, favor the discharge of flatus, and be invigorated generally. I suggested to her family that she be taken down every day to the beach in a carriage, and be allowed to sit there for a while inhaling the sea air.

Without going into the minutiae of the daily details of the case, which are of no importance here, it suffices to say that the beneficial effects of the recommendations made, and which were fully carried out under the supervision of the attending physician, soon became manifest. For the first four weeks there was absolute freedom from attacks; then they reappeared, but at much longer intervals than previously—once in two to three weeks—and with diminishing severity. Her *morale* was much improved. She went out every day, the weather permitting, down to the beach, and got the benefit of the sea breezes; passed much of her time upon the veranda, and soon began to gain in flesh and strength. I saw her at first twice a week, and latterly at gradually lengthening intervals until September 30th. Though I ceased my attendance then, the treatment was continued on the lines laid down by the physician in charge. The intervals between the paroxysms grew still longer, and finally they disappeared altogether. With increasing general vigor the heart muscle gained in tone and drew together upon itself, as was shown by the greatly diminished area of cardiac dulness, which at the date last named was already very nearly normal.

Late in November I saw her again here in the city, just as she returned from a walk in Central Park, looking rosy and healthy. Her face had become rounder and her body showed decided evidence of filling out. During the period of treatment the diet was very much the same, *i. e.*, as to quality and quantity, though minor changes were made as the patient grew tired of one article and demanded something else instead. Massage was given twice a week for five weeks, and then once a week for a further period of three weeks, and then at irregular intervals as I saw her. As incidents in the case it may be mentioned that for a while she took fluid extract of cactus grandiflora as a heart tonic, but that later on, when the paroxysms reappeared, various other cardiac remedies—*strophanthus*, *digitalis*—were given during the attack.

* As described in my book "Constipation in Adults and Children." The MacMillan Co.

Occasionally some nervine, as tincture of valerian or zinc valerianate, was given.

In March, 1898, we had a blizzard, and on a certain Sunday the weather was particularly severe. While riding down town that day I saw my former patient out promenading on the sunny side of the street, in the vicinity of her residence. In June, 1898, when she left the city for her summer home, she weighed 135 pounds. When I saw her again she had long discarded her attendant. She is in excellent health, stout, and as I saw her some weeks ago she must be fully 165 to 170 pounds. She told me that she feels splendidly, that it does not seem to her as if she had ever been sick. She gets about, up and down flights of stairs, and takes long walks without becoming unduly fatigued; but that she must be careful in her eating, not to overeat or eat anything very heavy, for any infraction of the rules I laid down for her makes itself felt at once about her heart.*

2. BY CONTIGUITY When the stomach is empty it is collapsed upon itself. When food is taken in it distends in all directions, and as the ingestion continues the distention increases. The greater cul-de-sac rises higher and higher and, pushing the diaphragm before it, may reach even to the upper border of the fifth rib, and is then in close proximity to the heart. As the latter contracts and lengthens out its apex is forced into the distended segment of the stomach to a greater or lesser depth, according to the degree of distention.

As indicated by the pressure curve, this is the normal process; and when the heart is healthy, and therefore possessed of normal force, and there is no marked overfilling of the stomach, there is no interference with cardiac function. When, however, the heart has sustained permanent damage, as in chronic valvular disease, and its force is therefore more or less weakened, it cannot drive down the apex into the distended stomach to the extent required by its lengthening out, and the systole becomes imperfect, and the following ones more so. As a result of this the ventricular cavities are not sufficiently emptied, the blood in the auricles is dammed back by that retained in the ventricles, and the whole organ becomes surcharged with it. The consequence is the suffocative paroxysm.

CASE II.—October 22, 1898. M. B., aged thirty-two years; clerk; married; four children. Five feet five inches in height. Weight about 108 pounds. Went through the various infantile ailments—scarlet fever, measles, etc. About seventeen years ago he had an

* A very interesting point in this case and one that attracted my notice quite a number of years ago in the case of another patient, recorded in my paper on "Nervous Dyspepsia," and to which I will merely call attention, without any further discussion thereof here, is this: As seen from the history, the lady before coming under my care ate a fairly large amount of food, a quantity sufficient for a healthy, active person, and though she took no exercise, did not vomit, had no diarrhoea, she grew thinner and thinner. After being placed in my charge, with the quantity of food reduced fully one-half and with more outdoor exercise, and therefore greater oxygenation, she began to gain and to put on flesh as recorded. The question suggests itself—"What became of the surplus energy of the food then taken?"

attack of acute rheumatism. Two years later it was noted that he had cardiac trouble. He was seen in the course of time by various eminent practitioners of the city. Latterly he developed attacks of dyspnoea, which continued to come on with increased frequency. After almost every meal he would have such an attack, and frequently of so grave a character that messengers would be dispatched in all directions to bring in a physician. In the summer of 1898 he went to the mountains, and while there was greatly improved as to the suffocative attacks; but, on the other hand, his bowels became very loose. Almost immediately after eating, and sometimes before the meal was finished, he would have to go to the closet. He had thus four or five large evacuations per day. Since his return home his suffocative attacks have recurred, and he still has a tendency to looseness of the bowels, though not as great as before.

Status Præsens. Very much emaciated, as already indicated. Face has a bluish tint, and is expressive of suffering. His appetite is good, and he eats the usual meals as they are served to the family. His bowels are rather loose, and he has occasional attacks of profuse diarrhoea. He has paroxysms of dyspnoea, mainly after meals; sometimes so severe that he thinks his end has come. Occasionally, uneasy sensations in the stomach. He feels very weak, and though he gets about and goes to the store, it is mainly by force of will; for when there he cannot do much work, and sits about most of the time.

Examination. Tongue fairly clean; teeth good. Pulse feeble, slow, irregular, and with distinct occasional intermissions. *Heart.* No heaving of præcordium. Apex-beat in sixth intercostal space to the right of the left nipple line, and somewhat fluttering in character. *Superficial* dulness of normal extent. Over the left apex a soft, rather blowing murmur, systolic in rhythm; does not travel to the left or upward and is not heard at the back; can be heard to the right as far as the right apex; can be heard with greatest intensity a little higher up, over the mitral valve region. At the base, to the right of the sternum, another murmur, also systolic in rhythm, is noted. This travels upward a little, but seems to be propagated downward, also about half-way to the right apex. The heart's action, on the whole, is rather weak, and the various sounds and murmurs are differentiated with some difficulty. *Stomach* normally located, normal in size. No tenderness over any part thereof. An examination of the chemismus not attempted. *Liver* and *spleen* normally located and apparently normal. *Kidneys*, so far as can be judged by a careful examination of the urine, healthy.

Diagnosis. Aortic obstruction and mitral regurgitation. This was confirmed by subsequent examinations when the patient had gained in strength and the heart in force, and its sounds and murmurs had become more distinct. I also learned subsequently from a physician who had seen him at a much earlier period that the trouble at the aortic valves was the primary one. The paroxysms of dyspnoea (which he thought altogether due to some stomach trouble, and therefore came to me) are due to the overfilling and consequent overdistention of the stomach and mechanical interference thereby with the functioning of the heart. The weakness of the heart as well as the general debility I ascribe to greatly impaired nutrition, and particularly to the looseness of the bowels. The diarrhoea is, as I view it, the result of impaired gastric digestion and the irritation of the intestinal tract by the pouring out

into it of the insufficiently digested and therefore insufficiently prepared food masses. No doubt intestinal digestion is itself impaired by this same irritation, and this also contributes to keeping up this looseness of the bowels.

Treatment. The main feature of the treatment, and the one to which, as I told the patient, I attached the greatest importance, because it meant better assimilation and but little loss, and therefore invigoration, was a proper diet—a diet that would nourish, while at the same time it would give rise to no mechanical interference with the action of the heart, and would not irritate the bowels. A diet list keeping these points in view was arranged for him. *Medication.* To assist the stomach he was directed to take a peptonzyme tablet with each meal (in the midst or immediately at its conclusion). To improve cardiac action, tincture of strophanthus, gtt. v bis die, was prescribed.

Under this treatment he began to improve at once. The suffocative attacks ceased, and did not reappear while he was under my care. The main trouble during that period was with the bowels, which would become costive and then again loose, with a tendency to diarrhoea; and with the loose stool, usually after a constipated one, there would come on some nausea and a feeling of weakness. Two or three times during this period he complained of some oppression in breathing, and each time it was, as he himself admitted, because he had eaten too many crackers at one meal. He excused himself, therefore, by saying that he was more or less hungry all the time and sometimes very hungry. He gained in strength, gained slightly in flesh, and there was improvement in his blood making. On November 7th the note says his pulse was 70, regular, and of much better force, showing that his heart had become stronger. The palpebral mucous membrane had decidedly more color. On December 28th he reported again, and the note says that he remarked that he was feeling better than he had felt for some years. This fair condition of the heart was sustained throughout the time I had him under my care. Occasionally he had rheumatic twinges, which were relieved by salol. In the course of the treatment, as time went on, changes were made in the diet, but the principles thereof were always maintained. The medication likewise underwent changes from time to time. Thus, after a while, strophanthus not appearing sufficiently effective, gave way to digitalis. For some time he took iron. Then there was medication *pro re nata*, tannigen or bismuth and tannic acid for the bowels. In February, 1899, he had a severe attack of influenza, with a tendency to pulmonary congestion. After the fever and cough had subsided a violent pain set in, which he located about the middle of the left clavicle, and it was some days before he was permanently relieved of this. About March 20th he was up in his chair, decidedly anæmic and feeble. He was put upon an iron tonic, and as the weather grew pleasant he gained in strength with fair rapidity. Then my connection with the case ceased.

It is beyond question that the diet chiefly was responsible for the improvement. By it alone the paroxysms were obviated, the great losses through the bowels stoppd, the whole system invigorated, and the heart in particular strengthened and put in a condition to withstand a disease (influenza) that not infrequently has broken down hearts that were in the best form.

CASE III.—October 24, 1899. L. J., aged twenty-six years; pharmacist; married; five feet three inches in height; weight, 133 pounds. He enjoyed good health, as far as he or his elder brother, who called with him, could recollect, up to six years ago. He had been working very strenuously in a down-town pharmacy—long hours, irregular meals, and a badly ventilated place to sleep in—and about the period stated he began to have pains in his chest, about the region of the heart, pains in his left and then again in his right arm. He called in a physician, who prescribed for him, and he got better. He was well for several years and then began to have pains in his stomach. He became very constipated about that time; he also noticed that his feet would swell. He placed himself under treatment, and the symptoms disappeared. For the last few months he has been very ill again, and has had several physicians to attend him. It has been said that his trouble was solely a gastric one, and was treated as such with alkalies, with acids, with the digestive ferments, with purges; and then, again, that his lungs were tuberculous, and the remedies suggested by this condition were prescribed. He has not been benefited by either form of treatment.

Status Præsens He is very sallow, has a sickly cast of countenance, and gives the impression of a tuberculous patient. He has no cough, occasionally gives a short hack, suffers much from oppression in the chest, with shortness of breath. He has pains in his chest. His appetite is now poor, and only light food agrees with him. His bowels are very much constipated, and he must take something to move them. No headache. Sleeps poorly, is awakened frequently with fits and starts. When his bowels have been thoroughly moved he sleeps much better.

Examination. Tongue clean; teeth good. Pulse 80, very shallow (low tension), and readily compressed. Lungs healthy. Jugulars on both sides pulsating strongly. *Heart.* Apex-beat in the fifth intercostal space and very diffuse, extending from 2 cm. to the right of the left nipple line to the left anterior axillary line. *Left apex.* Second sound rather loud and booming. *Right apex.* A blowing murmur, systolic in rhythm, distinctly heard here. Second sound booming as at left apex. At base the same booming in the second sound noted over the pulmonary artery, but not over the aortic region. Heart's action altogether rather weakly. *Stomach* normal, as far as location and size went, and as much as could be determined by palpation. Chemismus, for sufficient reasons, not examined.

Diagnosis. Tricuspid regurgitation. The dyspnoea and the feeling of oppression in the chest I was inclined to believe were dependent in a great measure upon undue distention of the stomach and consequent mechanical interference with the heart's action.

Treatment. He was put upon a diet almost the counterpart of that of Case II.; the very same in principle. As a tonic a glass of Wurtzburger beer was ordered. He was told to take life easy, to make no unusual effort, not to lift or carry, to avoid stairs as much as possible. Advised him to go to a small country town.

November 1st. He came in again to report. He is feeling splendid. Walks with a light and springy gait. He feels stronger, and has no trouble in breathing. Sleeps well. He made inquiry as to changes in diet, but I told him he must keep on as first directed if he wanted to continue to feel well. Re-examination of heart showed apex-beat stronger and much less diffuse than before. Pulse also of a little more force.

April 17, 1900, I was called to see him, and found him in bed in a semi-recumbent position, in bad condition. He was very much oppressed in breathing; could not lie down at all. He is nauseated; there is some œdema of the lower extremities. Pulse very feeble (curve showed but slight ascent). The history of his decadence was this: Following my directions strictly, he improved rapidly and gained in strength. All his symptoms disappeared and, as time passed on and he continued undisturbed, he concluded that he had not been a very sick man at all; that he was well now, and that it was useless to waste his time in idleness. He thereupon opened another large pharmacy in a down-town district, and worked from early morning until late at night—eleven or twelve o'clock. He also began to eat solid food, cautiously at first, and then a little more freely. Still he continued to feel well. Then came the Passover festival, and there was a cooking and a baking in his home, and many dishes of heavy calibre, as are the delight of his countrymen, were prepared for the festival, and when the festive days came around he ate freely, like a man who had starved for many days, and enjoyed all the good things hugely. The consequences were disastrous. The damage done by the overdistended stomach was irreparable this time. Though he improved somewhat, he could not get back to the condition he was in after his first visit to me last year. He remained weak; a walk of a block fatigued him. His breathing became more and more difficult; an œdematous condition with anasarca developed, and sometime in July of the same year he died, with manifestations of cerebral congestion.

It is thus very evident, and the histories here related amply demonstrate the correctness of the contention, that in cardiac affections the question of food is of momentous import to the patient. It is clearly shown, beyond the possibility of question, that he may be made thereby either miserable or suffering, or comfortable and well nourished, and, where this is possible, even permanently cured of his trouble, as it is adapted to his condition or not.

What is proper adaptation?

In the matter of regulating the diet for cardiopaths we must be guided:

1. By the well-established facts as to the nature of the various cardiac maladies, and

2. By the well-established facts in dietetics.

1. The cardiac ailments are classified into two great groups—the *functional* and the *organic*. In the first group the disturbances are mainly of an irritative character—i. e., manifestations of an irritation set up somewhere in the system that has extended to and affected the nerves, innervating the heart. Nevertheless, the organ as a whole is intact, and competent in most instances to do its work normally or at least fairly well. In the second group the heart has sustained damage structurally. It has lost thereby part of its pristine strength, and is therefore more or less readily impaired in its functioning.

Again, while in the first group complete recovery is the natural out-

come, in the second group it is only a question of husbanding the strength remaining to the organ, whether deterioration, to the extent of incapacity to act in the manner requisite for the well-being of the whole organism, shall set in at an earlier or at a later period.

The indications arising to us, therefore, would naturally be that for the first group all *irritation*, in so far as the stomach is concerned therein, must be subdued and avoided; and, for the second group, that it is not alone irritation, but *mechanical interference* also, that we must prevent.

2. The study of dietetics and physiological investigation have taught us: That certain foods are more quickly digested than others. That certain foods give rise to a greater development of flatus in the stomach and intestines, and thus cause greater distention of these parts, than others. That certain foods are digested mainly in the stomach, while others tax this organ but lightly, their preparation for absorption and assimilation taking place elsewhere in the digestive tract. Again, clinical experience has demonstrated to us that the methods of preparation—*i. e.*, cooking—affect the digestibility of the article; that prepared one way it is readily and easily digested, while prepared in another it taxes the stomach to the utmost and not infrequently to the stage of exhaustion. Lastly, it has been shown that fluids leave the stomach much more quickly than do solids.

Having, then, the indications given, we must upon the basis of our knowledge of foods and their digestibility, as stated above, so arrange the nutriment of the patient that it shall fulfil the requirements.

In accord with this I have formulated for myself certain rules, which are as follows:

1. AS TO FOODS.
 - a. All foods that have bulk must be excluded.
 - b. All foods that are flatulent must be excluded.*
 - c. Only foods that are readily and easily digestible are allowed.
 - d. All foods must be so cooked that their digestion is facilitated thereby, not impeded.
2. AS TO MEALS.
 - a. All meals shall be small so that the stomach is not taxed too much nor greatly distended.
 - b. The intervals between the meals must be so regulated that sufficient time is given the stomach to empty itself and to have an interval of rest before the next meal is taken.

Though, as already stated, the conditions are different in the two groups of cardiac diseases, nevertheless the same rules hold good for both, only their application is different. To illustrate this more clearly the following diet lists as directed for the patients of the various groups are subjoined:

* The reason therefor is evident: they distend the bowels greatly, keep the gases in the stomach, and thus cause pressure to be made upon the heart.

(A) FUNCTIONAL DISTURBANCES. Case I.—6 A. M., glass of milk* (sipped slowly or eaten with a spoon). 8 A. M., one wineglassful of Hoff's malt, with iron; a dish of oatmeal, with milk. 10.30 A. M., glass of milk. 1 P. M., a small broiled steak or two small broiled chops (with a little horseradish as a condiment); one-half glass of water; a small slice of white bread; a little fruit-jelly. 3.30 P. M., one and a half tablespoonfuls of dry sherry (or Madeira). 4 P. M., one glass of milk. 6 P. M., wineglassful of Hoff's malt, with iron, one cup of milk, and a small slice of bread (if preferred, a small piece of broiled steak in place of the milk). 9 P. M., one and a half tablespoonfuls of Madeira. At intervals of three hours in the night, milk, one-half cup, and another dose of the malt and iron sometime between the hours of 12 M. and 6 A. M.

Two weeks later she omitted the milk at 6 A. M. and took it with her breakfast at 8 A. M.; also took the milk but once in the night and the malt with iron twice.

Six weeks later, when she was already very much stronger and greatly improved in every way, as she had grown tired of so much milk, she took coffee for breakfast, and in place of the oatmeal mush a soft-boiled egg.

The dietary here, by reason of the great emaciation and great weakness of the patient, was much more liberal (though still fully in accord with the above-stated rules) than I am accustomed to allow. In the cases related in my paper here several times already referred to,* and which in all essentials (except as to etiology) were the analogues of Case I., the diet was much more restricted. It was as follows:

7 A. M., one cup of milk and two small slices of stale white bread. (If the need therefor is felt, one-half cup of milk at 10 A. M.) 12.30 P. M., a broiled steak or two medium-sized chops; a little horseradish as a condiment; one-half glass of water; one slice of stale white bread. 4 P. M., a cup of tea, with milk and sugar (preferably, if patients do not object too strongly, chamomile tea made from German chamomile). 7 P. M., a cup of milk and two slices of stale white bread.

It is, of course, understood that with the disappearance of the cardiac disturbance, and as the stomach shows a ready and easy digestion, the diet is allowed to progress very gradually to that to which the patients have been accustomed, after this fashion: A dish of oatmeal will be allowed for breakfast in addition to what is already taken; then, as it is seen that this is well borne, and the patient complains of hunger, a baked potato with the dinner meal; later still, a little spinach or a little asparagus is allowed in place of the baked potato, and so on.

* It is always directed that a pinch of salt be put into the milk; it takes off the flatness, makes it more palatable, and seems to inhibit in a measure the formation of gases. If it appears indicated two tablespoonfuls of lime-water are also directed to be added.

The last meal to be enlarged is supper. However, the patients are warned to be careful for a long time and a strict observance of the following rules insisted upon:

1. AS TO FOODS. All fried food of whatever character is prohibited.

All vegetables must be prepared after the plain American fashion; that is, they are first boiled in water, and when sufficiently soft are taken out and sliced or hashed up, put into some meat-broth, and allowed to come to a boil. No fats or browned flour allowed therein.

Of soups, only thin broths (without any fat therein), and even then only in small quantity, are allowed.

Of pastries, only the lightest puddings—rice, tapioca (made without any grease)—are allowed. (These must be permitted to get cold after their baking, so that the first heat shall be out; before being served they are warmed just sufficiently to make them tasty.)

2. AS TO EATING. All foods when eaten must be *only moderately warm*.

Never eat until full; always rise from the table feeling that you could have eaten a little more.

(B) ORGANIC (VALVULAR) DISEASE. Case II.—7.30 A. M., one-half of one fresh egg; one-half cup of milk; one cracker. 10 A. M., a cup of milk. 12.30 P. M., a plate of lamb-broth thickened with rice. 4 P. M., milk. 7 P. M., lamb-broth with rice. Before retiring, one and a half tablespoonfuls of dry sherry. When he complained that he could not get home for his lunch (noon), this meal was changed to cocoa and crackers. When he tired of the lamb-broth, after a while, chicken-broth was allowed instead. Likewise, when he tired of rice, the latter was omitted and the soup thickened with very fine noodles. When his bowels showed a tendency to looseness thick rice dressed with sugar and cinnamon was directed.

At one time in the course of my attendance, when he was feeling very good but complaining that he was always hungry, I allowed him to have a tablespoonful of finely chopped beef (which had been previously stewed in its own juice) put into the plate of soup; but it did not agree with him, was not well borne by his stomach and bowels, and had to be stopped after a few days.

Case III.—8 A. M., a cup of cocoa and two crackers. 10.30 A. M., a cup of milk. 12.30 P. M., rice or barley dressed with milk, or lamb-broth or chicken-broth or beef-soup thickened with either of the above cereals. 4 P. M., milk. 7 P. M., same as the noon meal. A glass of imported beer was allowed with his soup either at noon or evening.

The only arbitrariness in the diet in these two cases was as to the articles and the hours for meals; otherwise they were at liberty to substitute one meal for the other, as their tastes and their conveniences made preferable.

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A CONTRIBUTION TO THE STUDY OF PRIMARY SARCOMA OF THE TAIL OF THE PANCREAS.¹

BY M. S. KAKELS, M.D.,
OF NEW YORK.

IN making a diagnosis of an abdominal tumor, two fundamental facts must be considered. First. From what organ does the tumor grow? Second. What is the nature of the growth? *Qui bene diagnosticit bene curat.*

We all know that it is not always easy to detect from what organ in the abdomen a tumor grows, especially if it be very large and occupies a great part of the cavity. Nor can we often discern the pathological nature of the growth. In fact, it is frequently impossible, unless attended with exploratory puncture or incision.

Neither can we by our methods of examination, inspection, palpation, percussion, and auscultation always arrive at a correct topographical diagnosis, because the consistency of neoplasms is frequently misleading.

With no organ of the abdominal cavity is the diagnosis of a tumor attended with greater difficulty than those having their origin in the pancreas. Its deep position, its close proximity and intimate relation with other viscera, such as the liver, stomach, duodenum, spleen, and kidney, make it extremely difficult.

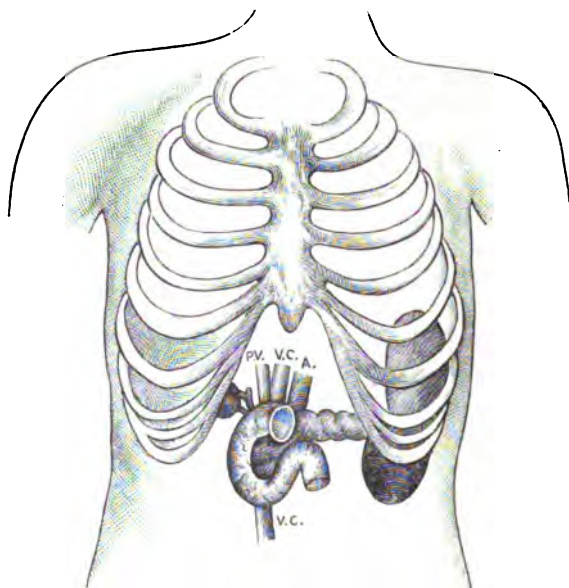
The situation of a tumor in the head, body or tail, if large enough, produces clinical symptoms, which vary according to the pressure exerted upon the underlying or contiguous organs or vessels in these localities. It is therefore clinically important to remember the close relation of this organ to the large vessels posteriorly, such as the aorta, inferior vena cava, portal vein, and the common bile duct; to the

¹ Read before the Metropolitan Medical Society, December 17, 1901.

stomach and duodenum, which lie anteriorly; to the horseshoe portion of the duodenum, which embraces its head to the right, and to the spleen, kidney, and suprarenal gland, which lie to the left.

A tumor affecting its head may press upon the bloodvessels mentioned and give rise to œdema, ascites, or persistent jaundice. A tumor growing from the body may compress the stomach or duodenum and give rise to dyspeptic or stenotic symptoms. A neoplasm springing from the tail, if of large size, may so simulate growths of the kidney or spleen that often erroneous diagnoses are made. This is especially so, as tumors of the spleen and kidney generally grow forward and toward the median line.

FIG. 1.



Of the known tumors which involve the pancreas, cysts, carcinoma, adenoma, sarcoma, and lymphoma have been found in the above order of frequency. Of the solid growths, carcinoma is the most frequent, sarcoma the least. Primary sarcoma is of the rarest occurrence.

There are recorded in the literature but twenty-one cases of sarcoma of the pancreas found either at autopsy or operation. Of this number in five there is an element of uncertainty as to their being true sarcoma, in ten only was the neoplasm noted as occurring positively primarily in this organ, while in the remaining six the histories are not complete and do not indicate whether the origin was primarily in the gland.

Of all these cases, in but three (those of Blind,¹ Routier,¹ and Witzel²) was the tumor found situated in the tail of the gland. To these three must be added the one whose history will be given further on.

Thus, after a careful perusal of the literature on this subject, there are only four authentic cases of sarcoma of the tail of the pancreas. Perhaps the infrequency of this occurrence may make the subject of the paper all the more interesting.

Carcinoma of the pancreas has been most frequently found. As the symptoms of a neoplasm, no matter of what pathological nature, are generally those due to pressure, a few of these that have been observed in cancer will also obtain when due to the presence of other solid growths. The symptoms, though, let me say beforehand, are not distinctive.

If the growth is in the head of the pancreas, pressure on the underlying vessels will produce ascites or oedema from stenosis of the portal vein or inferior vena cava. If the common bile duct is compressed, a slow but persistent jaundice develops, with sometimes enormous distention of the gall-bladder. It is in these cases where Courvoisier's law is exemplified, namely, closure of the ductus choledochus from pressure of a tumor, as a rule, produces enlargement and distention of the gall-bladder, while in stenosis due to the presence of stone the gall-bladder is found small and contracted. This rule, though, is not without exception, yet it occurs so frequently that it has a diagnostic value.

Closure of the pancreatic duct by the neoplasm intercepts the discharge of pancreatic secretion into the intestine, which may be recognized by the peculiar fatty character of the stool, known as steatorrhœa.

By pressing upon the solar plexus, pain in the epigastrium is produced. This may be dull, continuous, and occasionally paroxysmal and radiating. If in conjunction with a palpable tumor, and the above-mentioned symptoms present themselves, a diagnosis of growth is highly suspicious.

There may be evidence of pressure on the stomach and duodenum, characterized by dilatation of these organs and constipation. Or the growth may involve these structures, and by erosion produce intestinal hemorrhage.

Depending upon how much of the organ is destroyed, sugar may or may not appear in the urine.

One of the most characteristic symptoms is rapid emaciation, with cachexia and weakness. Whether this be due to the general condition found in malignant growths as a rule, or whether involvement of this organ produces disturbances in its function, interfering with nutrition and thereby contributing to the rapid emaciation, is a question that has not been solved.

These clinical symptoms are but seldom so very decided as to

justify a positive diagnosis, but if several are evident and a tumor can be felt, we may be reasonably suspicious of malignant neoplasm of the pancreas.

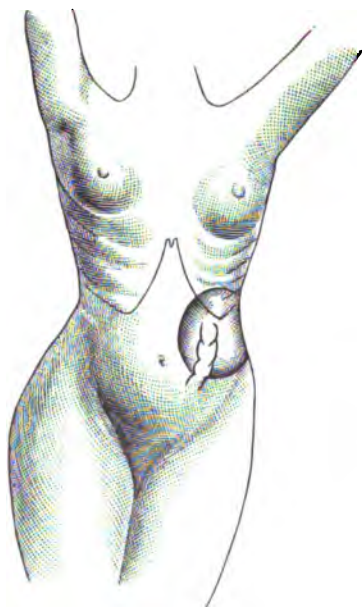
Up to the present time there are no individual pathognomonic symptoms which can with certainty be referred to malignant growth of the pancreas.

In the history of the case I am about to present there was rapid emaciation, pain and tumor, evidences undoubtedly of malignant growth. The peculiar situation of the neoplasm misled suspicion of pancreatic involvement, consequently examination of the stool was overlooked. Perhaps this may have given an important clue. The urine contained no sugar.

One can readily understand how difficult it was to make the proper diagnosis from the following:

Mrs. S., aged fifty-one years, married, four children. Up to six months ago was apparently a healthy woman. At about this time she complained of pain and a feeling of heaviness in the left hypochondriac

FIG. 2.



region, radiating downward. She commenced to lose flesh and strength, and noticed a gradual swelling of her left side. There had been no nausea, vomiting, or dyspeptic symptoms. When I saw her the first time, about the end of September, her condition was as follows: Very cachectic and extremely emaciated, a dry skin but no icterus; her appetite poor; no nausea, pyrosis or vomiting; slightly constipated. She complained of pain in the epigastric and left hypochondriac regions, with great loss of strength and weakness in her limbs. There were no ascites or any oedema. The abdomen on the left was distended by a tumor. The liver was not enlarged, and no protuberances evident. The gall-bladder was not distended nor palpable. The urine was normal in color, no sugar, but contained albumin, epithelial casts, a slight amount of blood, with a number of small round cells. The arteries were sclerosed, pulse frequent

and weak. Organs of respiration normal, heart sounds weak, a slight temperature of 99.5° in the rectum.

On inspection and palpation the tumor was found to extend from below the ribs on the left side down to an imaginary line drawn across the abdomen at a distance of 5 cm. below the umbilicus.

Its breadth extended from the linea semilunaris outward toward the

lumbar region. Percussion elicited flatness over the whole of this mass except along an area corresponding to the descending colon, where tympanitic resonance was marked. The tumor was slightly movable, non-adherent to the abdominal wall. It gave a feeling of semi-fluctuation, and as dulness merged from the tumor on to the spleen and kidney it seemed to be connected with these organs.

Digital examination of the vagina revealed the pelvic cavity free.

On account of the sense of semi-fluctuation, an aspirating needle was inserted, and blood with a few shreds withdrawn; the shreds on microscopical examination proved to be connective tissue and a mass of round cells.

Blood examination drawn from the finger yielded 40 per cent. hæmoglobin. Moderate leucocytosis. No count was made. No plasmodia. The cellular elements of the blood showed no evidences of splenic disease. Tumor of the spleen was therefore easily excluded.

The size and rapidity of its growth inclined me to the belief of its malignancy. It had a sense of semi-fluid consistency, and, being situated in the region where kidney tumors are generally found, I was prompted naturally to attribute its origin from this organ. This belief was in a great measure fortified by careful percussion and palpation in this region. Diagnosis of sarcoma of the kidney was therefore made.

An exploratory incision was advised, with the idea of removal if possible. Accordingly on the 12th of October an incision 15 cm. long was made from the lower border of the ribs along the linea semilunaris. My plan was to attack the tumor transperitoneally. As expected, the growth was found to be retroperitoneal, the size of a child's head, covered by omentum and mesentery. The descending colon lay on its anterior aspect intimately connected by dense adhesions, the stomach was pushed to the right. The surface of the neoplasm was nodular and covered by an immense number of dilated vessels. Its consistence was partly hard, partly soft and fluctuating. In attempting to lift it out of its bed of dense adhesions it partly ruptured, and soft grumous material escaped. It was impossible to shell it out from its adhesions to the neighboring vessels.

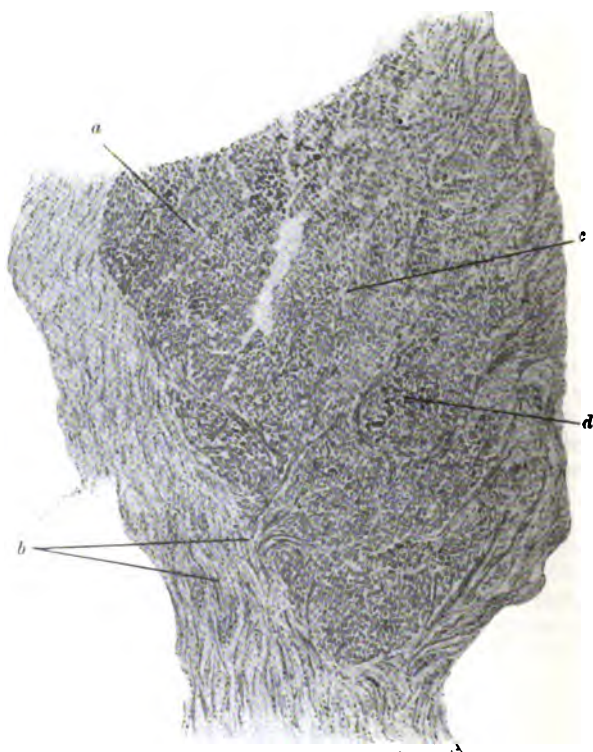
In its depth it was found to have grown from the tail of the pancreas, being merged into the kidney and spleen, both of which seemed to be embedded in its substance.

On account of the enormous difficulties encountered, and recognizing the impossibility of its removal, further operative interference was abstained from. The patient died soon after.

Pathological Report. Autopsy made by Dr. E. Schnaper, pathologist to the Lebanon Hospital. Inspection: Body emaciated; rigor mortis partially established; no œdema. Thorax, lungs: slightly congested and œdematous. Pleura: Both lungs bound down behind by old adhesions. Heart: Pericardium contains about one drachm of clear serum; left ventricle wall thickened; cavity small; muscle pale. Aortic and mitral valve normal; right ventricle wall thin; cavity slightly increased in size. Pulmonary and tricuspid valves normal. Abdominal cavity: Peritoneum, normal. Omentum free and contains only a small amount of fat in its meshes. Mesenteric lymph nodes enlarged, varying in size up to 1½ cm. in diameter. Firm in consistency, the cut surface showing the same consistency throughout. No other lymph nodes of the body were found enlarged. Liver: About

one-half inch below the free border of the ribs; enlarged, firm, yellowish in color. Gall-bladder: Distended with bile; normal. Common bile duct pervious. Spleen: Diminished in size; capsule wrinkled, firm in consistency. Kidneys: Smaller than normal; capsule adherent; surface granular; cortex irregular; markings distinct. Suprarenal capsules, normal. Stomach and intestines, normal. Pancreas: A large, partly hard, and partly soft tumor was found situated in the tail of the gland. The rest of the pancreas was firm throughout; no areas of softening. The pancreas and tumor weighed 2000 grammes.

FIG. 3.



Microscopical structure of tumor. Leitz Obj. 3. Oc. 3.

a. Capillary vessel. b. Connective-tissue stroma enclosing bloodvessels. c. Capillary vessels. d. Mononuclear and polynuclear cells.

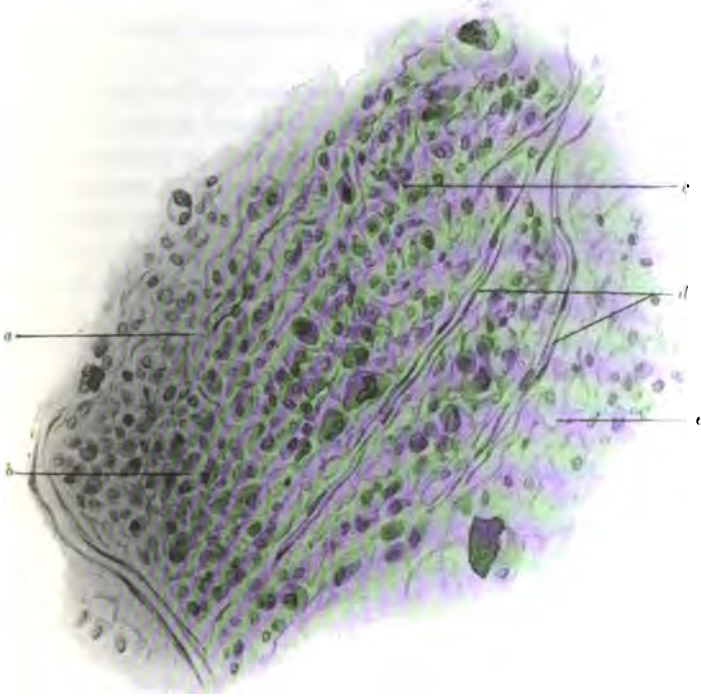
The tumor measures 18 cm. in length, 9 cm. in width, 60 cm. in circumference. Its surface shows irregularities which stand out as firm nodules. These, upon examination, were found to be lymph nodes incorporated in the growth. Firm connective tissue makes the external covering of the tumor. Upon cutting into the same it is seen to send in processes which surround grayish areas which are masses of cells. The length of the pancreas from the head to the border of the tumor is 16 c.m., its width $5\frac{1}{2}$ c.m., its thickness varies from $1\frac{1}{4}$ to 3

c.m. There is the same form of growth distributed throughout the whole of the gland.

Microscopical Examination. Sections were taken from various parts of the tumor, from the pancreas, and from the left kidney and spleen. The structure of the tumor reveals a fine, fibrous, reticulated network, in the meshes of which are a large number of small mononuclear round cells whose nuclei take the stain well. (Fig. 3.)

Very large cells, some mononuclear, some polynuclear, with large oval nuclei rich in chromatin, staining deeply, are also present in large numbers. (Fig. 4.)

FIG. 4.



Detail structure from Fig. 3. Leitz Obj. 6. Oc. 3.

a. Connective-tissue stroma. b. Tumor cells. c. Tumor cells, mononuclear and polynuclear. d. Capillary bloodvessels. e. Connective-tissue stroma.

The fibrous stroma, more prominent in some places than in others, is in direct relationship with the cellular structure. This stroma is highly vascular and shows capillaries with distinct walls. Parts of the tumor have undergone coagulation necrosis. In places small hemorrhages are seen to have taken place in the tumor. Lymph nodes: Increase of the cellular element as well as an increase of the reticulum. Left kidney shows no involvement of the neoplasm. Spleen free from any sarcomatous infiltration. Diagnosis, mixed-cell sarcoma.

Nearly all the latest writers have commented upon the rarity of primary sarcoma of the pancreas.

Senn⁴ states that only a few cases are on record.

Oser⁵ states that the infrequency of primary sarcoma may be inferred from the fact that such recent and well-known text-books on pathological anatomy as Orth's,⁶ Birch-Hirschfeld's,⁷ Ziegler's⁸ and others dismiss the subject with but few remarks by saying that it is exceedingly seldom found.

Körte⁹ says that primary sarcoma of the pancreas is of very rare occurrence.

Lancereux¹⁰ mentions that connective tissue neoplasms of the pancreas are very rare, though several cases of sarcoma are recorded.

In the literature so far as I have been able to gather, primary sarcoma is exceedingly rare.

The first authentic contribution was made by Paulicki,¹¹ who, at an autopsy on a patient dead from phthisis, found a small round-celled sarcoma of the pancreas. This was supposed to be primary.

Segre¹² found that of 132 tumors of the pancreas, 2 were sarcoma, but he does not mention whether these were of primary or secondary growth.

In the pathological institute of Kiel, Rhode¹³ observed only 3 cases of sarcoma of the pancreas with sarcoma in other organs, but whether primary in the gland he does not mention.

Senn, in his *Surgery of the Pancreas*, contributes a case of medullary sarcoma observed by Mayo.

Sidney Martin¹⁴ demonstrated a case of primary sarcoma of the pancreas before the Pathological Society of London.

Litten¹⁵ reported an interesting case of primary sarcoma of the pancreas in a boy aged four years, with extensive metastasis. The growth was confirmed as a primary neoplasm of the pancreas by Virchow.

Blind¹ showed before the Anatomical Society of Paris, 1894, a primary sarcoma of the tail, with metastasis in the liver, in a man aged seventy-four years.

Routier² enucleated a lymphosarcoma which seemed to have developed in the tail of the pancreas.

Neve¹⁶ described a sarcoma of the pancreas which involved the pylorus.

Chvostek¹⁷ observed a case of the pancreas and kidney with many metastases in other organs, but it is not positive whether he was dealing with a case of primary sarcoma of the pancreas or not.

Krönlein¹⁸ operated on a woman, aged sixty-four years, for pyloric carcinoma, which at operation was found to have been a growth involving the stomach, pylorus, and upper portion of the duodenum, and springing from the head of the pancreas. The patient died three days afterward.

Autopsy showed an angiosarcoma of the head. The body and tail of the gland were free from the disease.

Schüler¹⁹ mentions from the Greifswald clinic a case of hemorrhagic sarcoma of the pancreas in which the neoplasm had eroded the pancreatic artery, causing a hæmatoma in the tumor. From this erosion into the artery multiple sarcoma metastasis occurred.

Michaelson²⁰ reported a case from the Würzburger Pathological Institute which he considered a mixed tumor, namely, carcinosarcoma.

Baudach²¹ described a case from the Pathological Institute of Freiburg, in which he was doubtful whether from the histological examination the tumor originated from the glands of the structure or connective tissue. He called the tumor an angioma myxomatosum (cylindroma).

Bonnamy,²² in a thesis entitled *Étude Clinique sur le tumeurs des Pancreas*, records a case of Ingalls'²³ of sarcoma of the pancreas, liver, and gall-bladder, which, from the history and anatomical findings, seemed to have originated from the pancreas.

Briggs²⁴ reports a case in which he states he had to deal with a spindle-celled sarcoma originating from the wall of an hydatid sac of the pancreas. Körte²⁵ doubts this diagnosis of sarcoma from the unsatisfactory microscopical examination, and states that he knows of no instance where sarcomatous degeneration occurred from the walls of an echinococcus cyst.

Witzel²⁶ describes an interesting case of retroperitoneal tumor involving the tail and body. The head was not affected. Microscopical examination proved the neoplasm to be a spindle-celled sarcoma. This was extirpated, but the patient died soon after. As no autopsy was made, it would be difficult to class it as one of positive primary growth.

As was mentioned in the beginning of this paper, and as was gained from the histories, in but three, Routier's, Blind's, and Witzel's, was the growth found in the tail. Blind and Routier's were primary in the tail, Witzel's doubtful. To these two may be added the case which is the subject of this paper.

This, then, makes only three authentic cases of primary sarcoma of the tail of the pancreas that I have been able to collect from the literature on this subject, namely, Blind's, Routier's, and my own. It is probable, since the literature of primary sarcoma of this gland dates back only to 1868, many cases of this rare and interesting disease were not recognized as such before, on account of the imperfect histological knowledge of these growths.

The diagnosis of pancreatic sarcomata, as of all intra-abdominal tumors, is a matter of much uncertainty.

Until further experience and investigations of many more cases from which we can gather some positive symptoms which may point to pancreatic growth have been made, our diagnosis can only savor of probability. I do not think we can be far from wrong in making a diagnosis of retroperitoneal growth, neither do we often fail in recognizing

malignancy from benignancy. But so far as locating the neoplasm or recognizing its nature up to the present time can only be conjectured.

To summarize: The difficulties in the diagnosis of malignant pancreatic tumors are so numerous and great that we rarely find a case in literature where a correct diagnosis has been made before operation or necropsy.

All or several of the symptoms above mentioned, together with the anamnesis, may lead to a probable diagnosis.

Recognition in the living of the nature of the growth is almost impossible.

Tumors of the pancreas are of the greatest interest to surgeons, as it has been only of recent years that operations have been made for their removal.

For surgical intervention the most important symptom is a palpable tumor.

Cysts have been incised and their contents evacuated.

Less frequently, however, have solid growths offered opportunities for surgical interference.

Removal of solid tumors with a portion of the gland itself is exceedingly dangerous and rarely successful. Even if it were possible to extirpate the gland, aside from the insurmountable difficulties on account of the bloodvessels and close proximity of the neighboring organs, the surgeon would not be justified, because, as Minkowski has shown, the entire removal of the gland is followed by rapid and fatal diabetes.

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THE CLINICAL SIGNIFICANCE OF A CHRONIC URETHRAL DISCHARGE.

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THE time-honored custom of designating as "gleet" all chronic urethral discharges has for some time been recognized by genito-urinary surgeons to be a popular error, inasmuch as it does not accurately express the pathological conditions producing such discharge in all cases. As a matter of fact, three different forms of chronic urethral discharge are observed, each dependent upon a distinct pathological condition for its cause, and each requiring for its cure a separate and distinct line of treatment. The one most frequently observed is that to which the name "gleet" is properly given—a term which in its true significance is employed to describe a chronic discharge appearing at the meatus, scanty mucoid or mucopurulent in character, gluing together the lips of the meatus, yellow or yellowish-gray in color, staining the linen, and found upon microscopical examination to be made up of pus, epithelium, and fibrous shreds. This condition is well understood to be a direct result of chronic gonorrhœa, and depends upon the presence in the urethra of one or more localized areas of chronic inflammation with consequent exudation. As demonstrated by Finger, this pathological process may be limited to sharply circumscribed areas, superficial in character, involving merely the mucous membrane; or, in more advanced cases, there may exist involvement of the submucous tissue, with subepithelial thickening, constituting what are commonly described as "strictures of large calibre." Again, in protracted cases, secondary sclerosis occurring at the site of these granular patches, we have a resultant gleety discharge dependent upon a true organic stricture. In the large number of cases where gleet results from a chronic inflammation in the posterior urethra it will usually be found that many of the crypts and acini of the prostate gland are involved in the process, constituting the condition known as chronic follicular prostatitis.

The urine in true gleet is generally clear, and will be found to contain clap shreds in abundance, which fall rapidly to the bottom of the glass, and when stained and examined with the microscope are found to be made up of pus-cells, rarely containing gonococci, epithelium, mucus, and fibrous shreds.

Another form of chronic urethral discharge quite frequently observed is that first described by S. D. Gross, and known as prostatorrhœa.

Considerable confusion still exists among medical writers on the sub-

ject as to just what clinical condition should be embraced under the term prostaticorrhea. Gross, in his original description of the affection (1860), states that it "consists of a colorless, transparent discharge, occasionally containing pus, liable to be confounded with gleet, making a decided stain upon the patient's linen, and occurs as a result of a chronic inflammation of the glandular elements of the prostate gland." Sturgis¹ contends, however, that Gross in the above statement has confounded chronic prostatitis, prostaticorrhea, and urethrorrhea. In support of this opinion, he states that "the secretion from the prostate gland is a thin fluid, cloudy, sometimes milk-white;" again, "the discharge from the prostate in a genuine case of prostaticorrhea is never purulent, and when it is the affection is a prostatitis, acute or chronic, but not a true prostaticorrhea."

These statements are in direct accord with such an authority as Fürbringer, who says that "the secretion of the prostate as obtained by digital pressure from the rectum is never transparent and mucous, but is milky in character;" furthermore, he is of the opinion that "only one form of prostaticorrhea exists—that which consists principally in the discharge of normal prostatic fluid."

On the other hand, Taylor² considers the affection only as an accompaniment of chronic prostatitis, and states that "there is a constant escape from the meatus of a clear mucous fluid or of a mucus mixed with pus and perhaps a little blood."

The writer considers that the term prostaticorrhea, properly applied, is used to designate a discharge of normal prostatic fluid, free from pus, arising from purely functional disorders of the prostate gland, disassociated from inflammatory changes, but consisting for the most part in congestion of the gland, with hyperemia of the acini and hypersecretion of normal prostatic fluid.

The discharge which occasionally appears at the meatus in cases of chronic prostatitis, and which is found to contain pus, does not constitute a true prostaticorrhea, but should more correctly be spoken of as a "gleet," inasmuch as it is found as a secondary result of chronic posterior urethritis. The results obtained from stripping the prostate gland will materially tend to clear up any confusion which may exist in the observer's mind regarding the nature of the affection. Where the discharge into the urethra is the direct result of functional disorder of the gland, constituting a true prostaticorrhea, microscopical examination of the discharge after massage will demonstrate that it is entirely made up of prostatic fluid, consisting of amyloid corpuscles, refractive corpuscles, amorphous phosphates, similar to that described so fully by Finger, Fürbringer, Sturgis, and other authorities. On the other hand,

¹ Prostaticorrhea, etc., *Journal of Cutaneous and Genito-urinary Diseases*, June, 1896.

² *Sexual Disorders of the Male*.

where a given urethral discharge depends upon a chronic follicular prostatitis, examination of the fluid obtained by massage will reveal the presence of pus-corpuscles in abundance, in addition to the elements above mentioned; such a discharge should be regarded as a true gleet.

In the majority of cases the congestion of the gland, serving as the etiological factor in the production of prostaticorrhœa, will be found to be a direct result of sexual abuse, either in the form of frequent masturbation or of excessive sexual indulgence. In addition to these, Sturgis adds as a potent factor in causing the affection the abuse of premature withdrawal at sexual intercourse. Upon this point it is well to note, however, that there is some difference of opinion. Vecki, for instance, asserts that in cases where "frauding" is practised the circumstances are more favorable for the man than for the woman, and that whatever phenomena result from such practice, they are those of neurasthenia, with loss of sexual desire, rather than congestive changes in the prostate gland itself. Sturgis further adds that prolonged riding upon bicycle or on horseback with improper saddle, is a frequent exciting cause of the affection.

In nearly all cases affected with prostaticorrhœa, from whatever cause, rectal examination shows the prostate to be somewhat enlarged, tender upon firm pressure with the finger, and of a soft pulp-like consistency. As regards the appearance of the discharge, it is uniformly described by patients as "white of egg" in character, tenacious and viscid, slightly gluing the lips of the meatus together, rarely staining the linen. It is generally observed by the patient upon rising in the morning; occasionally the fluid escapes after some unusual exertion; in quite a number of cases it appears at the meatus upon straining at stool, much to the alarm of the patient.

The writer has found that, unlike true gleet, the discharge of prostaticorrhœa is not constant through the day, but appears only at such times and under such circumstances as those just mentioned. This escape of prostatic fluid into the anterior urethra is undoubtedly due, as stated by Taylor, to an atonic condition of the compressor urethræ muscle. The urine in all cases is clear, free from clap shreds, but is apt to contain numbers of comma-shaped, fine, hook-like shreds (Fürbringer's hooks), which do not fall to the bottom of the glass, but float in the urine.

A third form of chronic urethral discharge, very apt to be regarded as gleet by the careless or inexperienced observer, is that to which the name urethrorrhœa or chronic urethral moisture has been given.

This term is used to describe a colorless, watery discharge which is constantly present at the meatus, rarely in sufficient amount to glue the lips together. The patient complains solely of a persistent dampness or moisture about the meatus, and upon opening the lips with the thumb

and finger—a manoeuvre at which the patients with this affection get very expert—the meatus and urethra appear to the eye to be congested and to contain more secretion than should normally appear.

This condition is found in all cases to have developed as a sequel to a long-standing gonorrhœa, and is directly due to an atonic, relaxed condition of the capillary bloodvessels of the urethral canal. In White and Martin's work on *Genito-urinary Surgery* the affection is termed urorrhœa or urethral catarrh. It is here described as a "catarrhal discharge dependent upon a weak and leaky mucous membrane, and associated with no more serious pathological lesions than moderate congestion, which must be carefully distinguished from other post-gonorrhœal gleans; the discharge is bland, non-contagious, and local treatment, except by the mildest astringents, is distinctly contraindicated."

Examined microscopically, although it is almost impossible in many cases to secure sufficient discharge to obtain a "smear," it will be found to consist almost entirely of cylindrical epithelium and mucus. Urine in all such cases is invariably clear.

This condition will often be noted as one result of the vicious practice which patients who have suffered from chronic gonorrhœa fall into of "stripping the penis," for the purpose of noting the presence or absence of any urethral discharge.

The affection just described is not to be confounded with that spoken of by Fürbringer and Sturgis and termed "urethrorrhœa ex libidine," which is not constant in character, has no direct etiological relation to gonorrhœa, but is a direct result of sexual excitement.

| | History of case. | Macroscopical appearance of discharge. | Microscopical appearance. | Results upon the discharge of sexual intercourse or use of alcohol. | Appearance of urine. |
|---------------|---|---|---|--|--|
| Gleet | Chronic anterior urethritis. Chronic posterior urethritis. Epididymitis. Prostatitis. | Yellow or yellowish-gray. Forms a crust gluing lips of meatus together in the morning. Linen distinctly stained. | Pus. Epithelium. Fibrous shreds. Occasionally gonococci. | Discharge increased. | Clear or cloudy, containing claps and pus. |
| Prostatorrhœa | Not necessarily any venereal disease. History of sexual abuse. Frequent masturbation. Onanism. | Milk white, appearing during sleep, or on heavy physical exertion, or at stool; does not glue the meatus, linen slightly stained. | No pus. Characteristic prostatic fluid. Amyloid corpuscles. Refractive corpuscles. | No effect. | Clear, containing small comma-shaped bodies. |
| Urethrorrhœa | Chronic gonorrhœa. Practice of "stripping the penis." | Appearance simply of moisture in excess at the meatus. | No pus. Cylindrical epithelium mucus. | No effect after sexual intercourse. Very often improved by occasional use of alcohol. | Clear. |

As the treatment in each of these three varieties of chronic urethral discharge differs very materially, it is obviously essential that the physician should be able to arrive at a correct decision as to what particular form he is called upon to treat. To determine this point investigation should be made into: First, the history of the case; secondly, the macroscopical and microscopical appearance of the discharge itself; third, the effect upon the discharge of indulgence in sexual intercourse or alcohol; fourth, the character of the urine. The accompanying table is given as showing at a glance these various points in the differential diagnosis.

THE SANITARY MEASURES TO BE ADOPTED AFTER FLOODS.

BY GEORGE A. SOPER, PH.D.,
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THE following paper is designed to set forth a few suggestions for the guidance of those who may be called upon to assist in safeguarding the health of communities which may be thought to be exposed to epidemic diseases by reason of destructive floods.

It is evident that information which may be useful in averting the consequences of flood has special significance to the people of the United States. Possessing an enormous territory, with a wide range of climatic and geographical features, it is not strange that such catastrophies should occasionally occur. The low, flat valleys of the Mississippi and its tributaries afford an imperfect opportunity for the discharge of the waters sometimes collected upon their immense drainage areas. On such occasions thousands of square miles are inundated. Villages are submerged, and great destruction of property results. The fact that these floods are anticipated has not been capable of reducing their hardship, as witness the almost annual overflows, which caused a loss in 1874 of \$13,000,000, and the destitution in 1882 of 60,000 inhabitants of Mississippi.

Occasionally some of the streams of the Northern and Eastern States cause destructive floods, but their shorter, straighter courses and smaller flood-plains render such wide-spread damage as is common in the South and West impossible. Nevertheless, there have been destructive floods from such rivers, particularly in connection with the giving way of dams. Of this sort was the Mill River, Mass., disaster of 1874, and the Johnstown, Pa., flood of 1889.

Inundations from the sea have occurred on the low-lying coasts of the United States on several well-remembered occasions. Notable among coast floods have been those at Galveston in 1886, with a loss of thirty-eight lives and \$5,000,000; the storm of 1893 on the Louisiana coast,

during which practically all the settlements between New Orleans and the Gulf were destroyed; the Sea Island flood, wherein 2000 people were killed, 20,000 rendered homeless, \$5,000,000 worth of property destroyed; and the Galveston calamity of 1900, in which 8000 people were killed and \$20,000,000 damage was done to property.

After the first shock of such disasters as those referred to the world is generally appalled by the news that pestilence is liable to break out at the scene of destruction. At Johnstown and Galveston great apprehension was felt that one or more of the acutely infectious diseases would appear among the survivors and rescuers. Rumors that pestilence had actually broken out found their way to the public press, where they threatened to do serious injury to the commercial interests which were already so seriously crippled.

It may not be unprofitable to inquire for a moment what reason existed for the popular apprehension, and to determine if possible wherein lay the danger, if any existed.

There is but brief information available concerning the sanitary aspects of the great floods of antiquity and the middle ages. They are obviously not connected in point of time with the great epidemics which visited the civilized world in those periods; nor do the facts at hand suggest that floods have ever been considered important agents in the generation or spread of infectious diseases. In point of fact, a belief in the cleansing power of floods is strongly implanted in the popular mind. The Bible story of the deluge is not inaptly held as the symbol of a remedy for the evils of corruption rather than a precipitant of disease. Why, then, should pestilence have been so greatly feared at Johnstown and Galveston?

It is probable that the fear of disease grew from a contemplation of the disorder and great destruction of life and property, the demoralization which was everywhere apparent, and particularly the want of sanitary control, which very soon manifested itself. Had an earthquake, for example, produced similar conditions of wreck and confusion, it is not improbable that the same fears of pestilence would have been experienced.

Undoubtedly the active source of infection was supposed to lie in the lifeless and decomposing matters which were associated with the wreckage. To the senses of the survivors and rescuers the odors of decomposition were a constant menace and active source of alarm.

As to the reality of the danger which is popularly supposed to exist in the presence of dead bodies of men and animals, little need be said to those who are familiar with modern views of the etiology of the communicable diseases. By them it is known that a foul odor is only rarely in itself a source of danger, while it is a most unreliable indication that disease-producing conditions are close at hand. According to

accepted theory, diseases which are capable of becoming epidemic are incapable of originating in foul or decomposing matters. Every form of infectious disease is produced by a specific agent peculiar to it. This agent or its progenitors has existed for an indefinite period, and is only developed in large proportions and with dangerous virulence within the body of a living host. Thus yellow fever, bubonic plague, typhus, cholera, etc., are bacterial diseases which occur only when the micro-organisms associated with them are carried by some means from one subject to another. Familiar vehicles for the transmission of infectious material in such cases as those which we are considering are water- and milk-supplies, flies, dust, and impure food.

It follows, if these views be accepted, that those who are compelled to handle the bodies of previously healthy men and animals killed by flood or other violence need have little fear of contracting any of the so-called filth diseases, no matter how far decomposition of the body may have proceeded. Nausea is the worst form of illness to be anticipated. And what is true of the bodies of men and animals is true of vegetable matter as well.

The conditions are different, however, when we have to deal with the remains of persons ill of, or killed by, disease. During illness and after death the excretions and surfaces of the person are frequently highly dangerous as sources of infection. Here are to be found the bacteria or special agents which are capable of reproducing the disease with which they have been associated. It is obviously important that contact with infectious material be avoided, that it be prevented from disseminating, and that its source should be as soon as possible removed. These ends are accomplished by isolating each case of sickness, disinfecting contaminated objects, and destroying infectious material as near its origin as possible. In the conditions which obtain after floods probably very few people will have been killed excepting those who were in health, while among the large number of persons who remain there may be some sickness. Therefore, as a general rule, it is safe to say that danger of pestilence lies not among those lifeless matters destroyed by the flood, but rather among the living.

After the destruction of their homes many of the flood sufferers of Johnstown and Galveston found shelter in camps. Under such circumstances it was inevitable that overcrowding should now and then have occurred, and that the attention given to such subjects as drainage, the disposal of waste, the procurement of pure food and water, the avoidance or destruction of insects, and careful supervision of the health of the survivors should have been for a time neglected. Nor is it to be wondered at that in the excitements of the hour the usual care for individual safety should have been relaxed. Physical and mental fatigue marked the condition of nearly every individual. We cannot

fail to recognize in these conditions important predisposing causes of disease. They were insufficient to produce an epidemic, but were well calculated to spread a pestilence should it break out. There was needed but a spark to kindle the flame of a greater disaster than had been wrought by the elements.

The spark was looked for among the large number of strangers who flocked to the scenes directly after the news of the calamities was spread abroad. The people who visited the wrecks were almost exclusively men of the meanest class—curiosity seekers, adventurers, thieves, and worse. They were drawn from all points of the compass, and generally came on freight trains or on foot. Their number was very large. They lived from hand to mouth, subsisting on supplies sent to the flood sufferers, without working and without responsibility of any kind. In view of the circumstances, it is extremely curious that none of the acutely infectious diseases appeared.

From the foregoing considerations the steps which should be taken to safeguard public health in thickly settled flood-stricken communities are fairly indicated. There is need of a strong sanitary organization, with a responsible head, to avoid epidemic, by removing the conditions which favor the spread of infectious diseases and to watch for cases of such sicknesses in their incipency.

Who should be chosen to occupy the position of chief sanitary authority? By all means, the proper person is the health officer of the town, city, or county, if he is present and capable of exercising the onerous and exacting duties of the office. In disasters of so great magnitude as those cited it may be that the city and county health officers are unable to cope with the situation. It then becomes a question as to what other authority should be summoned to the scene. It is essential that a sanitary bureau be established promptly.

In the United States no help is more suitable in this emergency than that which the State health organization should be capable of furnishing, and it is appropriate that the Commonwealth should contribute its services in the event of such an emergency. It is to be regretted that all of our States have not such excellently conducted boards of health as to be capable of affording such prompt and competent services as are demanded under the circumstances which we have under consideration.

When the State is unable to accept control the United States Government may be appealed to. There is a fund placed at the disposal of the President and Secretary of the Treasury for use on such occasions as demand unusual protection to prevent the spread of epidemics. In the absence of a national board of health, the United States Marine Hospital Service is prepared to take the field at short notice to avert or put an end to any epidemic which threatens to invade the country. It should be remembered in this connection that the resources of the Gen-

eral Government are not available except when the danger is very real and of such a nature that the country at large is liable to be affected through the inability of local authorities to control the situation.

Having settled the question of authority, it will be desirable to establish headquarters for the direction of the sanitary organization. The confidence and co-operation of the flood sufferers should be encouraged from the outset. Much importance is attached to this point and to the desirability of distributing immediately and as widely as possible a knowledge of the purposes and methods as well as the powers of the sanitary bureau. Secrecy with regard to the real condition of the public health, such as has been sometimes practised with regard to the identity or presence of communicable diseases, is not considered a wise policy; cases of illness should be reported by their proper names and announced in an accurate manner. For this purpose it will be advantageous to issue bulletins.

A set of sanitary regulations for the guidance of the people may be issued to advantage, and should contain instructions as to the cleaning of premises, the repair of injured household effects, the disposal of garbage and other wastes, the methods of using deodorants and disinfectants properly, and the need for proper precautions in the matter of diet. With the idea of reducing the number of people to be cared for, as many as possible of those who are not usefully employed in the work of rescue should be advised to leave town for a few weeks' rest.

The scene of the wreck in the districts wherein camps have been established may be advantageously divided into sections, the limits of which, for convenience of reference, should be marked on a map. It is suggested that each one of these sanitary districts be made a unit for the operation of a small force under the direction of a minor sanitary officer, it being an advantage to encourage a spirit of emulation and amiable rivalry in the interest of greater efficiency among the various corps at work. It will be found desirable to keep a business-like record of the operations of the bureau, and for this purpose an office force of clerks will be useful. The operations which it will be necessary to carry on outside of the office can be accomplished through the aid of local help. Physicians and others whose former residence was in the locality will have the advantage of familiarity with the place and people.

One of the most important divisions of the work of the sanitary bureau will be the observation and direction of the flood sufferers in their efforts to look after their own health. A corps of responsible inspectors should be organized to move about among the people in a systematic way, to see that the regulations of the sanitary authority are properly carried out, and to supplement and interpret his directions by sensible counsel and advice. Cases of disease which are capable of becoming epidemic should be watched for by these men with the greatest

care, particularly in the cheap lodging-houses, camps, and other places where the evils of overcrowding and uncleanness are most liable to occur. When a case of infectious disease is discovered the patient should be isolated and the premises thoroughly disinfected in accordance with the practices commonly observed in large cities with regard to the more dangerous diseases.

The water-supply and food provided for the sufferers and helpers should be inspected systematically. Wells believed to be contaminated should be pumped out, and either thoroughly cleaned or closed with an official seal. Unless the water-supply is wholly beyond suspicion, that part of it which is to be used for drinking and cooking should be required to be boiled.

In cleaning damaged houses attention should be given to the removal of slime, mould, or silt from floors, walls, and furniture. Carpets should be taken up and dried in the sun and beaten, or else destroyed. Upholstery, especially mattresses and cushions, to be preserved should be opened and sun-dried. Cellars should be pumped out, ventilated, and whitewashed. Fumigation will not be necessary in these operations unless the presence of disease germs is strongly suspected, in which event the use of formaldehyde is recommended.

Of the great variety of so-called disinfectants which will probably be sent to the scene of the wreck the most useful will be quicklime, chloride of lime, and carbolic acid. Although the public is tolerably familiar with the use of these chemicals, instruction should be given in the ways in which they may be employed to the greatest advantage, particular attention being given to the necessity of bringing about an intimate contact between the material to be treated and the agent, the desirability of economy, and the advantages to be gained by avoiding the necessity of using deodorizing and disinfecting materials.

In making a permanent disposition of decomposing or decomposable organic matters as much use as possible should be made of the opportunities afforded for incineration. The bodies of horses, cows, and other animals, garbage and excreta may be easily burned if first sprinkled with rosin. Bonfires may then be made over such material with fuel taken from the wreck. Condemned beds, carpets, furniture, etc., may be soaked with tar or oil before burning, to ensure complete combustion. In disposing of human remains burial is much to be preferred to burning, provided the graves or trenches intended for interment can be decently made and of a depth of at least six feet. All bodies disposed of in this manner should be placed in one locality, which should be at least one hundred yards from any habitation or well. Where insufficient depth of soil is found for burial in the usual way, quicklime may be used to advantage in the bottom and covering of the trenches, a few pailfuls of water being thrown over the top to ensure immediate action.

With regard to the removal of débris and cleaning about the scene of the wreck, it will be well to remember that the removal of infectious material or matters which are either actually offensive or liable to become so, does not constitute the whole duty to be fulfilled. An appearance of order and neatness is a condition greatly to be desired, since it makes for cleanliness and wholesomeness, and is in itself an outward evidence of that control and moral support which it is perhaps, after all, one of the highest functions of the sanitary bureau to maintain.

REPORT OF A CASE OF DERMOID CYST OF THE MOUTH;
CRITICAL REVIEW OF THE LITERATURE.

BY CHARLES GREENE CUMSTON, M.D.,
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CYSTIC productions in the tongue or the floor of the mouth have been divided into four groups by Streckeisen, according to whether they are subcutaneous—that is to say, more superficial than the mylohyoid muscle; between the geniohyoid muscles, or in their tissue; between the genioglossus above the geniohyoid muscles; and, lastly, in the concavity of the os hyoid. Out of all the cases that I have been able to collect I can find only one that appeared to be distinctly subcutaneous, while in those instances where the cystic production appeared superficial it will be found that they had not only pushed aside the mylohyoid muscles, but extended between the geniohyoid muscles, and even between the genioglossus. A more precise classification may be made if their points of origin are taken into consideration, and they may develop in any of the various tissue layers from the skin of the suprahoid region up to the mucous membrane of the buccal cavity. Cases have also been reported of cysts situated in the tissues at the apex of the tongue or on the dorsum of that organ. In other instances the neoformation was so large that it distended the tongue, transforming it into a large cystic cavity. These cysts develop in size in indirect relation to the amount of resistance offered them by the surrounding structures, and consequently their precise anatomical site is frequently difficult to assign.

Gerard-Marchant divides dermoid cysts of the floor of the mouth into adgenien and adhyoidien, according to their adhesion to the lower jaw or to the os hyoid, and in fact these cysts oftentimes contract adhesions with these bones.

Streckeisen's classification would lead one to suppose that these growths are always attached to the os hyoid; but this is not the case in each and every instance, as will be seen by reading the report of cases appended to this paper. In one case the adhesions connected the growth to the bony insertions of the genioglossus. The classification

of Gerard-Marchant is faulty in that it only includes a restricted number of cases, because in a large majority these cystic productions are free of adhesions. When adhesions do exist they are usually composed of a very short fibrous band of varying resistance, while in other cases the growth is attached to the bone by a sort of thin, fibrous pedicle, which may contain a duct having a diameter of two or three millimetres; or, on the other hand, the adhesions may be of the consistency of fibro-cartilage containing a duct. In these cases the primary origin of the adhesions was due to the formation of the cystic process. Similar examples are found in congenital cysts of the neck, but they are most infrequent in those developing in the floor of the mouth.

When adhesions have been noted connecting the cyst with neighboring structures other than the bone, in most cases they were secondary and followed a suppurative process arising in the cyst cavity. The diagnosis of adhesions is hardly possible before operation is undertaken, either because these attachments have a weak resisting power or because their development is so feeble that they cannot give rise to any signs on palpation. Should the cyst and the os hyoid move together during deglutition it is a fair proof that adhesions exist between them. In some instances the tumor adhered to the lower jaw, but nevertheless moved with the os hyoid.

As to cysts of the tongue, they may appear to be superficial, but nevertheless they are lying beneath the muscle, and in this organ they are always separated from the mucosa by a muscular layer which may be reduced to an extreme thinness, but which is never wanting. When they develop toward the under surface of the tongue they invade the floor of the mouth, and can only become submucous by penetrating the space separating the genioglossus muscles.

These cysts may be divided into two groups, according to the structure of their walls and the nature of their contents, the mucoid and dermoid cysts. Mucoid cysts are of extreme rarity, and I have only been able to find three recorded cases. It is believed that they are derived from the canal of Bochdaleck. Their walls are composed essentially of two layers, the external one being a connective-tissue structure. The internal aspect is lined by a mucous membrane composed of cylindrical epithelium or of pavement epithelium. The epithelium in two cases was provided with cilia; they were absent in the third case, but this may have been due to a suppurative process which had invaded the cavity of the cyst before its extirpation had been undertaken. The contents of these cysts was a viscous fluid.

The histology of dermoid formations in the floor of the mouth is usually that of the skin. The lining membrane of the cyst has a basement layer of connective tissue on which rests the layer of pavement epithelium, arranged in several strata; this inner membrane

varies in thickness in different parts of the cystic cavity. The upper strata of epithelial cells are quite flattened, and as they have undergone a complete horny transformation their nuclei are wanting. They are very loosely connected—a fact which is particularly evident on the surface, where the single scales and flattened layers are on the point of desquamation, and they are very loosely connected with the remainder of the epithelium.

Next to the horny stratum will be found several layers of nucleated cells which correspond to the rete of Malpighi, the uppermost of which are somewhat flattened, but gradually become cubic and polygonal in shape, the lower layer of which rests on the connective tissue, and here the cells have almost a cylindrical form. I am not aware that sickle-shape cells have ever been demonstrated to exist.

The corium is composed of densely packed bundles of connective tissue, running parallel or at right angles to each other, and very tightly intermingled. Elastic fibres are also present. The lower strata includes large and small sebaceous glands as well as a large number of unstriped muscular fibres, either single or in bundles, and interspersed throughout the corium.

The general direction of the muscular fibres varies, but the majority run parallel to the surface of the epithelium, while others are more or less at acute angles with it. Here and there we find the muscular fibres running singly across the entire corium, and arranged very similarly to the erector muscles of the hair of the skin. Hair follicles are not usually found; but they must, in all probability, be present in the cyst walls, because this is demonstrated by the presence of hair in the contents of the cyst. The sudoriparous glands and papillæ are not to be found. In both cases the cyst contains a whitish-yellow, cheesy mass, and being the product of the cyst walls, is composed of horny epithelium and epithelium which has undergone a fatty degeneration as well as the products of decomposition and the secretion of the sebaceous glands.

According to our present views, dermoid cysts are congenital formations; that is to say, they are derived from epithelial germs which during foetal life have become segmented off in some way or another, or which have reached a spot where, in extra-uterine life, epithelium is normally absent. This germ aberration or retention does not take place at any one particular spot of the ectoderm, but only at spots where a special process of development favors it, such as the fetal sulci lined with the ectoderm. Now, when the ectoderm closes in, or when it enters into relation with other tissues for the purpose of forming certain organs, the epithelial débris may remain; and it is only this explanation that can be scientifically employed as the reason for the typical localization of dermoid cysts. According to this conception,

the point of origin of dermoid cysts in general must be marked out when the oral cavity and the limitrophous parts of the throat are developing. This formation commences somewhere about the end of the second week of foetal life.

According to Hertwig, the formation of the blastodermata and the first process of segmentation are completed, and the human embryo essentially represents two long tubes, one of which is enclosed within the other, the inner tube being composed by the trophic germinal plate and the middle visceral dermic plate; while the outer, which is only separated from the inner layer by the abdominal cavity, is entirely composed of the ectoderm.

The oral cavity first becomes differentiated, and below the projection formed by the frontal part of the brain an inflection of the epithelium takes place on the ventral aspect of the head, which gradually becomes deeper and deeper toward the closed end of the cephalo-intestinal cavity, so that it is finally only separated from it by a thin partition—the outer epithelium and the trophic germinal plate.

Very shortly afterward this partition gives way and its fragments are turned back, and consequently an external communication has been effected, namely, the oral cavity. At the summit the latter is bordered by the frontal protuberance, laterally by the two maxillæ, and inferiorly by the mandibular projections, which also represent the upper pair of branchial arches.

Simultaneously with these changes occurring at both sides of the region of the throat the four branchial arches make their appearance. Parallel with the mandibular arches small indentations arise, which, by pushing aside the middle blastodermic layer, reach the surface and become connected with the epidermis. The epidermis, at the point of contact, dips down and forms a cleft so that a deep and a superficial branchial cleft are formed. They are only separated from each other by a thin epidermic membrane which runs between the clefts. The first branchial closes and forms the oral cavity, and below this is the first pharyngeal cleft, and below this are three other branchial arches and clefts.

Such being the embryonal formation of the throat and oral cavity, two possibilities suggest themselves to one's mind for explaining defects in the development of these organs. The first may take place when the lateral branchial clefts close up from above downward, or later on, at the time when union takes place between the branchial arches and clefts in the median line of the throat. The first process may give rise to congenital fistula of the neck; and, according to Roser, we can have four fistulæ resulting from an imperfect development of the branchial ducts, their orifices being found respectively in the anterior part of the region of the ear, the region of the os linguæ, the region of the larynx,

and the sternomastoid region. We also may trace back to this formation those cysts which are rather frequently observed under the mandibula, the *os linguæ*, and below and along the side of the sternocleidomastoid muscle; they are probably the débris of the branchial duct.

The clefts situated in those parts which face the intestinal cavity are coated with cylinder epithelium, while externally pavement epithelium is found; consequently the cysts contain either pavement or cylinder epithelium, or both may be present, according to whether the segmentation is a deep or a superficial one. Most frequently these cysts occur between the angle of the mandibula and the thyroid cartilage, especially those that are lined with pavement epithelium and which are commonly called atheroma, and also between the horizontal branch of the mandibula and the *os linguæ*. It is in this latter region that their origin can be more easily recognized. Pathological formations of a very similar character are also met with in the median aspect of the neck, although they are far more infrequent than those found on the sides. We also meet with certain fistulæ and cysts whose origin can only be explained by assuming a defective closing of the branchial arches, resulting in a retention of the epithelial germs. The fistulæ extend in front of the larynx and also in front of the *os linguæ*. The cysts are partly situated in the incisura jugularis, but the larger number are present under the tongue, in the floor of the mouth.

Hoffmeister divides these cysts into those that have their origin in the entoderma and those arising from the ectoderma. The former are said to be characterized by the thinness of their walls and the absence of hair. Dermoid cysts, however, are those most frequently met with. It is not easy to understand why dermoid cysts situated in the median line are less frequent than those found on the side of the neck, and why, if they do arise in the former situation, they do not occur in the floor of the mouth. For the present, at least, we can only assume that the conditions necessary for the formation of a congenital defect are more favorable on the side of the throat than at the median line, and that in the median surface the region of the second branchial duct offers the greatest chance for the rudimentary formation of dermoid cysts.

Although dermoid cysts in the floor of the mouth are rather rare pathological findings, and comparatively few cases have been reported, there still have probably been quite a number operated on whose histories have not been published. Undoubtedly they have often been mistaken for a ranula, and then, too, the symptoms produced by dermoid cysts naturally depend largely upon their size and the rate of growth. Directly after birth and in infancy, when the cyst has not developed, or, if so, only to a small degree, it gives rise to little or no trouble, and very often when they remain very small they are hardly ever noticed by the patient. It is only after the period of puberty is

reached that these cysts commence to develop in size, and it is then that they increase sufficiently in volume to cause a certain amount of inconvenience.

At the bottom of the buccal cavity the cyst, as it increases in circumference, frequently pushes the tongue upward and backward, so that it becomes pressed against the roof of the mouth, and, owing to this, all the functions of the mouth and tongue are rendered difficult. Speech becomes more and more indistinct, and when the cyst arrives at a certain size the articulation of words and mastication become practically impossible. Respiration may also be difficult, though no instance of complete obstruction to breathing has ever been recorded, although this might be quite possible if the growth pressed sufficiently on the larynx, or if it closed up the posterior pharynx by pushing the tongue sufficiently backward. These dermoid cysts never give rise to pain, and their prognosis is naturally favorable, because a radical operation can be performed with a perfect result.

The pathology of other cystic neoplasms which are occasionally met with in the mouth, more especially those which develop from the superficial mucous glands of the mucous membrane, I shall leave aside as not having any direct bearing on the subject of this paper, because these productions never attain any size, and their prognosis is perfectly simple.

From the differential diagnostic point of view we have, however, a pathological process which has for its site of development the under aspect of the tongue and floor of the mouth, and which presents almost identically the same clinical symptoms as dermoid cysts, or at any rate in many instances they simulate the symptoms of dermoids very closely. The cystic tumor to which I refer is ranula, and I will devote a few words here to its pathology, not only because it has given rise to a great deal of confusion, but also, and more especially, because up to the present time the true character of this disease has not been clearly defined, and many tumors of the tongue have been given this name, which really did not belong to them.

The first attempt to explain the cause of ranula was made by Diemerbroek, who considered this affection as a cystic production due to the retention of saliva in the duct of Wharton. According to this authority, the retention is produced by stenosis of the duct or by its complete occlusion. Soon afterward the ducts of Bartholini and Rivini were also considered the starting-points of ranula, so much so that even Dupuytren believed that the small mucous glands situated in the bottom of the mouth could give rise to the formation of this affection if retention of their secretions occurred. Fleischmann believed that ranula was a hygroma of a certain mucous follicle which he discovered in the mucous membrane of the bottom of the mouth, and is to be found

on the anterior border of the genioglossus muscle. But all these theories have been proven to be untenable; Fleischmann's follicle is not constantly present, and the small retention cysts occurring in the mucous glands of the mucous membrane lining the bottom of the mouth never attain the size that ranula does; and, still more, they do not lie beneath the mucosa, but are embedded in it. As to Diemerbrock's theory, it may be refuted from the fact that Wharton's duct is found in a perfectly normal condition when ranula exists.

Other attempts to explain the pathology of ranula were made by Neumann and von Recklinghausen, but this time with the aid of the microscope. Since Neumann was able to demonstrate the presence of ciliated epithelium in the anterior of the cyst he believed that ranula might originate in the gland tubules of Bochdaleck, for the simple reason that they are lined with a ciliated epithelium. Von Recklinghausen, on the other hand, considered that ranula arose from the main ducts of Blandin-Nuhn's gland, which is situated at the tip of the tongue. This theory was considered as the only correct one for some years, until Hippel's paper was published a year or two ago, dealing with the structure and pathogenesis of ranula, and it may be said that this essay has pretty thoroughly cleared up the question regarding the mode of origin of this neoplastic cystic formation.

Hippel considers ranula as a retention cyst of the sublingual gland, and, in some rare cases, of the glandula incisiva. The causative factor is to be found in a chronic interstitial inflammation of the ducts which occludes the smaller tributary ducts. From the commencement the cystic formation arises from the continued glandular secretion, whose emunctories are occluded, and later on by a proliferation, degeneration, and desquamation of the epithelium lining of the ducts as well as a transudation occurring from the new-formed capillary vessels. As a secondary effect the occluded portions of the glandular tissue undergo a pressure atrophy and disappear.

Now, according to this mode of origin, the wall of ranula shows no epithelial structures whatever. It is simply a thin sac composed of connective tissue, embedded in which are found atrophied glandular lobes here and there, which are oftentimes difficult to recognize as such. Externally the walls of this cystic production are connected with the sublingual gland at its lower and posterior aspect, and after a while the process may extend somewhat to the front and toward the upper part of the gland. The ranula gradually extends over into the intracinous connective tissue of the sublingual gland. On the anterior aspect, however, it comes directly into contact with the submucous strata of the mucous membrane of the mouth, and at this point the cyst is much thinner than at other places, which is due to the quantity of the contents present in the cyst and to the degree of distention of

its walls. The contents of ranula are composed of saliva secreted from the gland, which is mixed with blood-serum transudated from the numerous new-formed capillaries of the cyst wall. Microscopically we find small epithelial masses which represent the last remnants of the epithelium structures.

The position of ranula is naturally not always the same, and varies according to the position of the sublingual gland as well as the site of the glandular lobe which is the seat of chronic interstitial inflammation. Generally speaking, the cystic production is seated beside the frænum of the tongue, and then as it increases in size it extends outwardly and inwardly, so that it appears to have an almost median position. In other cases a glandular lobe that is situated rather laterally may have been affected, so that the cyst is also situated to one side and does not reach the median line. It is rather unusual to find a case of ranula where the cyst is in the median line near the dental arcade, and, according to Hippel, when this does occur the affection has originated in the glandula incisiva, which histologically has the same structure as the sublingual gland, and consequently the former may be the seat of the same pathological process as the latter. The development of these cysts is, as a rule, not very considerable, and only in rare instances do we ever find a case of ranula that has developed to any great size. The submental type of ranula will produce a pressure on the soft parts composing the floor of the mouth, in order to find space enough to develop.

The commencement of dermoid cysts in most instances passes by unnoticed by the patient, but in a large number of cases the presence of the cyst was noted at birth or shortly after; but at this early period the tumor is usually only discovered accidentally. In a few cases its size was large, considering the age of the child, and was a hinderance to nursing—a fact which drew their attention to the condition present.

In adolescence and adults the growth, which had been discovered and been known to exist for a varying length of time, has been estimated at a size varying from a walnut to an apple. In one instance it attained the size of the fist. The growth may project both into the mouth and from under the chin in some cases, but there are some reported where these growths project simply under the chin and showed no prominence in the floor of the mouth. In other cases where the tumor only develops upward under the mucous membrane of the mouth—and this is by far the most frequent condition—no projection can be noticed in the suprahyoid region.

In those cases where the cyst is sufficiently large to fill the buccal cavity more or less completely, the tumefaction that it produces in the suprahyoid region may become accentuated or make its appearance if it were absent in the beginning. The growth projecting into the mouth

pushes the floor of the cavity toward the suprahyoid region. The cyst situated in the floor of the mouth is more or less near to the lower jaw, whose concavity it occupies and fills. In some instances it lies directly against the posterior surface of the symphysis and pushes up the frænum of the tongue, while in other cases it recedes from the jawbone, and by examination a sort of transversal furrow will be found separating it from the bone. In other instances the cyst may develop in the tongue itself, which gives the organ the appearance of being filled with liquid, as the new-formation usually involves the entire thickness of the organ and extending backward nearly to its base.

In all cases the cyst is located in the median line, and the frænum of the tongue, which is more or less tense, divides the cyst into two symmetrical parts. The integuments which cover the cyst, whether skin or mucous membrane, are healthy in appearance and preserve their natural color. The mucosa in particular has a rosy color, and only in a few exceptional cases has it been known to take on a grayish hue, and this only occurred in mucoid cysts—a fact which can be easily understood. In a few cases the exterior aspect had a yellow color, which was due to the transparency of the mucous membrane and the walls of the cyst, which by transmitted light gave the yellow tint of the fluid contents to the coverings of the tumor. But in most cases the mucosa is opaque, and what is the most essential point to be remembered in the diagnosis is that in no case has the bluish color of the mucous membrane been met with, which is so characteristic in the greater number of cases of ranula. Wharton's ducts always remain patent, although they are sometimes deviated from their normal course, but saliva will always be seen flowing from their orifices.

By palpation the mucous membrane of the mouth will be found smooth and even, and the same may be said of the skin, which can be moved easily over the underlying organs. This condition is what should be expected, since the very loose cellular tissue forms a complete sheath for the cyst, and if inflammation has not taken place within the growth no adhesions will exist between it and the fat. Palpation will also show that the limits of the cyst are more or less distinctly circumscribed, and it has an oval shape, without any elevations or irregularities in its contour. Intracystic tension may be marked, but extracystic tension is in most instances only slightly pronounced. The cysts may be elastic, but in a large majority of cases the growth was soft, sometimes simply renitent, but usually fluctuation could be elicited.

To demonstrate the presence of fluctuation bimanual palpation is perhaps the best way of proceeding so that the pressure may be transmitted from one projection to the other. A symptom which has been described by Linhart is that when pressure is made on the culminating

point of the cyst its sides expand not rapidly, as in cases of liquid tumors having a distinct fluctuation, but with a certain tardiness. The swelling at the sides of the cyst will disappear as soon as the pressure has been removed.

Puncture of the cyst with an exploring needle may be useful in ascertaining the nature of the contents of the growth, but I would limit the practice to doubtful cases, although in several instances it was done where the surgeon thought he was dealing with a case of ranula, and was thus able to rectify his diagnosis. With the exception of mucoid cysts, whose liquid contents might flow off, although with difficulty, nothing will come away through the exploring needle if the growth is a dermoid, but the needle when withdrawn may contain particles of caseous matter. There is, however, no reason why a certain amount of fluid should not be present in these cysts, just as there is occasionally found in dermoids of the ovary, and in one case reported by Caye some liquid was present. The dermoid cysts of the mouth develop without pain, and this is perhaps why in most cases their exact beginning is unknown, and although they may have been present for some years they are not recognized by the patient until late in life.

A dermoid will develop in the direction of the least resistance, and when it grows toward the suprahoid region the patient will not be much hindered by its presence; but it is not the same when the growth projects up under the tongue. If the cyst is large it may produce a difficulty in the articulation of words or an alteration in the tone of the voice. This symptom is frequent, but it is common to all intra-buccal growths, and particularly to ranula.

As the tongue is pushed upward and backward, deglutition is sometimes embarrassed, while mastication is often difficult on account of the obliteration of the buccal cavity by the growth. Even when this may have arrived at an extreme degree respiration does not appear to be interfered with excepting when the patient makes an effort.

Deformities of the skeleton have been noted in a few cases. In one the alveolar border was projected forward almost horizontally, so that the lower incisors were very prominent. In another case the lower incisors and canines were pushed forward obliquely. The lower jaw was likewise projecting. I would say, however, that in these rare instances the cysts had reached a very large size and had an exclusively intrabuccal development.

In no case have I been able to find that the cystic production opened spontaneously, as is the case in dermoid cysts in other regions; but there is no reason why this should not occur in the cysts under consideration. Incomplete operations, such as puncture or incision and drainage of the cyst, have always been followed by a recurrence of the growth, and usually have increased the difficulties of a radical opera-

tion, as these interventions have generally given rise to inflammation resulting from septic infection of the cyst.

The prognosis of dermoid cysts of the floor of the mouth is quite serious, inasmuch as in the newly born it prevents nourishment being taken; in the adult the relative gravity of these cysts lies in the fact that they have no tendency to retrocede.

The tumor increases in size, and may, if allowed to continue, become so voluminous that movements of the tongue become impossible. On the other hand, it may become spontaneously infected and give rise to a severe inflammatory process, as occurs in dermoid cysts of the ovary; and this may also arise, as I have already said, after an incomplete surgical operation for its relief. In these cases also an incomplete operation may result in the formation of a sinus, which will not close until complete extirpation of the growth has been done; but when complete extirpation has been performed the patients promptly get well, and all the disturbances in mastication, deglutition, and phonation soon disappear.

The diagnosis is most important on account of the treatment, and there are cases which offer no difficulty. If, for example, we are dealing with a congenital tumor situated exactly in the median line—indolent, with a slow development, without inflammatory symptoms, having a special pasty fluctuation, the mucous surface being opaque or of a whitish or yellowish color—and if at the same time Wharton's ducts are patent, there is no occasion to hesitate, and an exploratory puncture will be quite superfluous.

But in most instances these cysts do not present themselves with all these signs, and occasionally it is only by eliminating each one of those tumors which may occur in this region that a diagnosis can be surely arrived at; and what proves the difficulty of the diagnosis is the relatively large number of mistakes that have been committed and have only been rectified after an exploratory puncture or during the operation.

Among those tumors which might be mistaken for a dermoid cyst I would only mention tubercular abscess and solid neoplasms which have undergone an ulcerative process, because the mistake that is most likely to occur is confounding a ranula with a dermoid cyst, and *vice versa*. Since dermoid cysts are congenital formations in those cases where the presence of the growth has been noted early in the patient's life, the possibility of ranula will at once be excluded.

Now in a case of ranula the condition of affairs is entirely different; they may develop at any time and at any age, provided a chronic inflammatory process develops in a small duct of the sublingual gland; and this can bring about a cystic formation in a much shorter time than a dermoid would need for its development. It is not at all

unusual for a patient to say that the tumor developed in from one to two weeks, and that the large cystic growth present commenced as a small elevation under the tongue. Those cases of dermoid where the commencement of the growth does not begin in infancy or during puberty are very rare indeed. They may possibly be explained by assuming that the epithelial germs which did not disappear during fetal life, for some reason or other later on began to develop; but even then it is probable that a very small tumor had existed under the tongue, but had not been observed by the patient until it began to increase in size and caused some trouble.

Patients will also occasionally say that a small tumor situated under the tongue has burst on one or more occasions on account of some injury inflicted, and that after having discharged its contents had again reached its original size, and this speaks rather more in favor of the diagnosis of ranula than of dermoid. The thick walls of a dermoid cyst offer much more resistance to external violence, but when circumstances are very unfavorable it may happen that the walls become lacerated, and then the dermoid will discharge its contents; but in this case, on account of the slowness of the secretive process, the sac of the cyst is refilled after a much longer time than would be the case in ranula. Other distinctive features between ranula and dermoid cyst may be had by inspection, and, as may be seen by the drawing illustrating my case reported in this paper, the surface of the dermoid growth is smooth; they are rounded tumors which extend out across the floor of the mouth and pretty well fill up the bottom of the buccal cavity, and push out the mucous membrane overlying them in a very uniform manner. The growth reaches equally outward on both sides of the median line and uniformly presses on the submental region. The mucous membrane covering the growth, being elastic, has become stretched with ease, due to the slow growth of the tumor, and no tension or pathological change can be observed. The mucous membrane has retained its normal thickness and color, although the tumor walls considerably differ in hue from the surrounding structures on account of their whitish color.

The aspect of ranula is quite different. Owing to the above-mentioned reasons, excepting those rare instances where the cystic formation originates in the glandula incisiva, the tumor is never found in the median line, but is always situated more or less to one side; in its first stage of development, at the lower border of the free part of the tongue beside the frænum. Ranula causes the mucosa to project, but as it increases in size it does not retain its original rounded shape, but yields on account of the thinness of the cyst walls and of the liquid contents which exercise pressure on it, more especially by the tongue, so that in the end it takes on a rather elongated shape, one end

of which appears usually as a rounded projection under the mandibula on the side of the throat; but what is most characteristic of ranula is its bluish color, which is due to the transparency of the clear liquid contents of the cyst through the thin layer of connective tissue and the red mucosa lining the floor of the mouth; the latter, on account of the rapid growth of the cyst, becomes stretched, although to a limited degree, but has, nevertheless, become somewhat thinner.

Lastly, there is a difference in these two types of cysts which is made evident by palpation. Although the surface of both is smooth to the touch, and the mucous membrane is freely movable over the growth, and although both these cystic productions are distinctly limited from the surrounding tissues, it will be found that if with two fingers we try to define the presence of fluctuation this symptom can be obtained distinctly in cases of ranula, except, perhaps, in those instances where the cyst is less plainly defined, owing to its large size and very tense walls, while dermoid cysts will show pitting left on its surface by the exploring finger in many cases, which demonstrates the putty-like consistency of the contents of the cyst. If, however, the contents have become partially liquefied, an occurrence which may take place occasionally when the dermoid is of long standing or a very large one, we are naturally liable to get a sensation of fluctuation. In doubtful cases an exploratory puncture will immediately inform us of the consistency and quality of the contents, and the diagnosis is made. If it is a cheesy, lumpy mass, or in those instances where liquefaction has taken place, when it is a thick, oily, brown fluid, we have a dermoid cyst, while in cases of ranula the contents are a thin, clear, light-colored liquid; but, for surgical reasons, puncture should never be resorted to if it is possible to avoid it.

The sanguineous ranula of Dolbeau, or, in other words, erectile tumors of the floor of the mouth, have, in common with dermoids, only their slow progress and congenital origin. They are violet in color, they are reducible, and they do not change in size during the cries of the child.

To finish with the fluid tumors of the floor of the mouth, I would mention hydatid cysts, which have twice been met with in this region, in which case the diagnosis could hardly be made otherwise than by an exploratory puncture if the cyst is situated in the median line.

Lipoma of the floor of the mouth have a few symptoms in common with dermoids, among others a false fluctuation. These neoplasms are extremely rare; they are not congenital; their progress is slow and progressive; they often project into the suprahyoid region; they are more or less irregular in shape; their thin encapsulating membrane occasionally gives rise to the yellowish color by transparency. By palpation they have a peculiar feeling, devoid of that soft, pasty, and often fluctuating consistency of dermoids.

As to the treatment of dermoid cysts of the floor of the mouth, it may be said that it is a simple surgical procedure. Not long ago these productions were treated by simple puncture followed by the evacuation of the contents; but it is evident that no permanent result could be obtained in this way, for the walls of the cysts will continue to secrete, and after the little wound left by the needle has closed up the contents would again collect. At the present time the only proper treatment is extirpation of the cyst.

The buccal route was preferred for a long time, because it appeared a more simple matter to operate in the mouth, as the growth projected under the tongue, and consequently was more directly reached. Besides this, a cutaneous cicatrix was avoided, and still more, the extirpation of the cyst through the mouth was never followed by any serious symptoms. At the present time the consensus of opinion is that these growths are better removed through an incision in the submental region. The soft parts are separated by blunt dissection, and it may be necessary to divide the platysma and the mylohyoides muscles. The entire cyst can be removed with greater facility if care is taken not to puncture it, and thus allow it to discharge its contents, because the walls will then collapse, and dissection will be rendered far more difficult. After the cyst has been removed the resulting cavity had better be packed with wicking and drained for a few days. I believe that removal of these cysts through the mouth, by making an incision directly through the mucous membrane should only exceptionally be employed, because very unfavorable conditions for the wound are naturally present. Retention of the secretions may occur because the field of operation is limited, and dissection of the cyst by way of the buccal cavity is often very difficult, and quite frequently the contents must be allowed to escape, at least partially, in order to effect its removal.

I will now in a few words outline the technique of the operation. An incision is made in the median line, extending over the entire convexity of the growth from the lower border of the jaw to the os hyoid. The geniohyoid muscles are exposed, and the muscular mass composed by the mylohyoides muscles are separated with a Kocher dissector and held aside by retractors; the cyst is then exposed to view, and with the finger the adhesions binding it to the surrounding structures are broken down, care being taken to free the growth from the adhesions binding it to the os hyoid or the genian apophyses.

If by any chance the contents of the cyst should escape during the operation, the wound should be carefully cleansed before the sutures are inserted, because occasionally the contents are septic and might infect the wound.

CASE I. (Cumston.)—Male, aged twenty-one years, was referred to me in October, 1900, for a large tumor situated in the floor of the

mouth. The patient said that the growth had been present since the age of seven years. It was then a small, painless tumor situated in the floor of the mouth, in the median line. For the last three years the tumor has been growing until it has reached its present size, which is that of a large lemon. (See illustration.)

By examination the growth was found to be oval in shape and reached above the level of the lower teeth. The tongue was thrown upward and backward, but it could be pushed out of the mouth a little. The posterior limits of the growth were difficult to make out on account of its size, but it certainly extended backward to the angle of the lower jaw. It did not project in the submental region, but by palpation here it could be felt. The buccal mucous membrane was perfectly normal



CASE I. Dermoid cyst of the floor of the mouth. (Dr. Cumston's case.)

in color and moved freely over the surface of the growth. Both Wharton's ducts could be seen giving issue to saliva when they were stimulated.

By palpation the tumor gave rise to the sensation of a doughy fluctuation, as if the cavity might contain a thick, oily substance, but no pain could be elicited.

Speech was very difficult for the patient and deglutition was becoming so, and it was particularly for this latter symptom that the patient was advised to seek surgical aid. From the history of the case, and the data obtained from the patient and by examination, I had no hesitancy in making a diagnosis of a dermoid cyst of the floor of the mouth. Extirpation of the cyst was performed through an incision in the submental region.

Pathological examination of the growth after removal gave the following data: The cyst was situated in the submucous cellular tissue of the floor of the mouth, between the genioglossus muscles. The contents of the cyst were a grayish, pasty mass, which microscopically was found to contain cholesterolin, hair, and elements which probably represented altered epithelial cells. Microscopical section of the cyst wall showed that the internal aspect of the cyst was lined by an epidermic layer in every way similar to the normal epidermic layer, with rows of horny cells, and under these a granular layer very distinctly visible, and finally the rete of Malpighi. The second layer was composed of a dermis in every way identical to that of the skin, excepting that perhaps the elastic tissue was a little more developed, and the papillæ were less distinct than in normal skin, appearing larger but less elevated. The hairs present had bulbs, and were surrounded by a distinct sheath with sebaceous glands. From these pathological findings we were evidently dealing with a perfectly typical dermoid cyst.

CASE II. (Caye).—Female, aged sixteen years. Came to hospital for a tumor in the mouth which hindered the movements of the tongue. The growth had been present since infancy, had always been distinctly in the median line, and increased in size very slowly until about three years ago, since which time it has developed progressively. Examination showed a tumor situated exactly in the median line in the floor of the mouth under the tongue, extending a little more to the left than to the right. It was the size of a walnut and was perfectly smooth. The growth reached as high as the dental arch, but did not project into the submental region.

Mucous membrane of the buccal cavity normal. Palpation showed the tumor soft; when pressure was relaxed it took its primary shape. The presence of adhesions binding the growth to the deeper structures could not be ascertained; the orifices of Wharton's ducts were seen on the anterior aspect of the growth. Diagnosis, dermoid cyst of the floor of the mouth.

Extirpation of growth by the way of the buccal cavity. The growth did not adhere to the muscular tissue, being isolated from the surrounding structures by an enveloping capsule of loose cellular tissue. Anatomical diagnosis, dermoid cyst.

CASE III. (Rocha).—Male, aged twenty-two years. For the last two years had noticed a tumor under his tongue which had slightly interfered with speech. As the growth was increasing in size, speech was becoming more difficult as well as the mastication of food. On inspection, the entire floor of the mouth was filled by a tumor which projected well up into the buccal cavity, pushing the tongue upward. The growth was situated in the median line. The surface of the tumor was smooth, and the mucous membrane covering it normal. The growth, on palpation, was found to be soft, but no distinct fluctuation could be elicited. Diagnosis of dermoid cyst of the floor of the mouth was made.

After puncture of the cyst through the mouth, which gave exit to a certain amount of caseous substance, an incision was made in the submental region transversely, about 10 cm. in length. After cutting through the platysma and mylohyoideus muscles the dermoid was laid free and was dissected out. The wound was plugged with gauze and sutured. Recovery uneventful.

CASE IV. (Rocha).—Male, aged thirty-eight years. Ever since childhood he had noticed the presence of a tumor under his tongue. In the beginning it was about the size of a cherry, but later it increased in size so that in the last twenty years it had grown to the volume of a walnut, and had then appeared to stop growing. The patient did not complain of any pain in the growth and it did not trouble him much to eat or speak. During the last two years the growth had reached the size of an apple.

Under the tongue the entire floor of the mouth was pushed up by a tumor, the mucous membrane of the buccal cavity covering it being perfectly normal. The growth did not appear to be connected with the tongue. The sublingual glands appeared to be somewhat atrophied. By palpation the tumor appeared to give rise to a pasty fluctuation, and when the finger was pressed upon its surface the imprint made remained for a few seconds and then disappeared. Diagnosis, dermoid cyst of the floor of the mouth.

The mucous membrane of the mouth was incised to the extent of about 6 cm. and, by blunt dissection, was freed from the surface of the tumor. During extirpation the walls of the cyst broke and gave issue to a certain amount of caseous matter containing hair. Gauze packing and drainage of the wound, with suture of the mucous membrane. Recovery uneventful.

CASE V. (Syme).—Female, aged twenty-four years; presented a tumor in the floor of the mouth sufficiently large to completely hide the tongue, and projected in the submental region as well. It had been present since infancy, but had only begun to increase considerably in size in the last few months. An incision was made which exposed a cyst of a yellowish color, its contents being a grayish, caseous substance. The cyst was about the size of a large hen's egg.

CASE VI. (Linhart).—Female, aged twenty-one years. Had noticed for the last two years a flattened tumor under the tongue, which in the beginning had not interfered with speech, but during the last six months had notably increased in size. The floor of the buccal cavity was depressed, and in the submental region a fluctuating distended tumor could be felt. By inspection the mucous membrane of the floor of the mouth was seen to be pushed up by a growth which occupied the entire floor of the mouth, and whose borders touched the teeth of the lower jaw. At the upper aspect of the growth the point of the tongue was hardly to be recognized, situated as it was in a sulcus, and consequently could not be projected forward. The mucous membrane covering the cyst was movable over the growth, and on the median line a fine white cord drawn over the middle of the cyst, and which represented the frænum, could be seen. The orifices of the ducts of the sublingual glands were found on the lower lateral part of the growth very near the floor of the mouth. Fluctuation was especially evident when bimanual palpation was practised. Deglutition was so difficult that the patient could swallow hardly any solid food.

A curved incision following the border of the lower jaw was made in the mucous membrane, and the flap thus formed was dissected off and raised toward the tongue. Another incision was made in the median line, care being taken to avoid the orifices of the salivary ducts which separated the flap of the mucous membrane into halves. On each side of the median line the genioglossal muscles could be seen, and

they were divided the entire extent of the wound. After a rather difficult dissection the cyst was freed, but on account of its size it was impossible to deliver it through the mouth; but, by puncture, a certain amount of caseous material was allowed to escape, which reduced the cyst sufficiently to allow of its easy delivery.

CASE VII. (Linhart).—Male, aged twenty-eight years; presented a rounded tumor on the floor of the mouth, the convex surface of which touched the roof of the mouth, preventing the jaws from coming completely in contact, and interfered with mastication and speech. The growth was covered by the buccal mucosa, under which the oblique course of the excretory ducts of the sublingual glands could be seen.

Digital pressure could flatten out the tumor, and produced depressions which only disappeared when movements of the tongue were made. The cyst was of a pasty consistency and painless. The exact situation in the median line, between two longitudinal projections formed by the border of the genioglossal muscles, settled the exact location of the growth.

A longitudinal incision in the median line was made in the mucosa, carefully avoiding the salivary ducts, and the tumor was enucleated. It was only slightly adherent, and was easily detached by the finger.

The cyst, after removal, was larger than a goose egg, its contents being composed of a thick white mass containing numerous short hairs. The patient was discharged, well, a week after the operation.

CASE VIII. (Patel).—Female, aged forty-nine years, with a tumor situated on the right side of the floor of the mouth. The growth was first noticed about eighteen months ago. It was about the size of an almond at that time, and, according to the patient's story, it had not increased in size since. There were no functional disturbances produced by the growth.

Examination showed on right side of the floor of the mouth an elongated tumor lying against the external aspect of the lower jaw and projecting slightly above it. The tongue was projected upward. The tumor occupied the position of the sublingual gland. Its inner limits stopped at the median line, while outwardly they were not distinct, and appeared to extend beyond the sublingual cavity. The mucosa covering the tumor was somewhat vascular and granular. The orifice of Wharton's duct was indistinctly seen.

By palpation the tumor was found to be soft; it could easily be reduced, and pulsation was absent. On the left side a small tumor having the same characters was noted. Both of these tumors, which were situated on each side of the median line, lie on a tumefaction which appears to occupy the entire floor of the buccal cavity. Fluctuation could be distinctly made out, but there was entire absence of pain. Examination of the median suprahyoid region showed an increase in size, but the patient says that it had always been there. By palpation a tumor, the size of a turkey's egg, was found, situated above the os hyoid, and apparently extending more to the right than to the left. The skin was movable over the surface of this growth, and the tumor did not move during the act of deglutition.

A median suprahyoid incision was made, and after going through the muscles the growth was exposed. It was then incised and gave exit to quite a large amount of semi-solid coffee-colored contents, composed of sebaceous material. The cyst was then dissected out with difficulty,

on account of the tough adhesions binding it down on the right side. Drainage. Cure.

CASE IX. (Delore).—Male, aged forty-five years. Only about six or seven years ago he began to have a slight difficulty in speaking and eating. The mass continued to increase in size, so that after the lapse of two or three years the disturbances in deglutition and speech became very marked, and attacks of suffocation would occur during the night on account of the weight of the tumor forcing back the tongue.

The growth, which was about the size of a large orange, and covered by a smooth and normal mucosa, projected almost from the mouth. It was situated almost in the median line, and pushed the tongue against the roof of the mouth and slightly to the right. About two-thirds of the tumor was on the left, the other third on the right of the median line. The suprahypoid region was elongated, flatter than normal, and was pushed backward by the tumor.

Palpation revealed a peculiar softness, and distinct fluctuation could be elicited. Below, the tumor was in contact with the os hyoid and the posterior aspect of the lower jaw. It moved with the tongue and os hyoid, with which it was manifestly adherent. It did not appear to adhere to the jaw.

A puncture was made, and some semi-solid contents, reddish in color, oily in consistency, and containing cholesterin, were evacuated. The pocket of the cyst was then enucleated through an incision in the mucosa. The cyst was found very adherent to the os hyoid and apophysis geni. Complete extirpation was, however, accomplished after much difficulty. A drainage-tube around which gauze was packed was inserted into the cavity. Cure. Histological examination showed the cyst to be a dermoid.

CASE X. (Richet).—A child a few days old was brought because for the last few days he would only take the breast with difficulty, and as time went on this appeared to be increasing. On examination the tongue was found pushed up by a tumor the size of a small walnut, situated in the floor of the buccal cavity, which it appeared to almost completely fill. Fluctuation was present, its surface was smooth, and extended from the front of the mouth backward.

A few weeks later, after other treatments had failed, excision of a portion of the walls of the cyst, with cauterization of its interior, was performed, and as the improvement was great the child was not again seen for two months. At the end of this time the patient was brought back, and a small, rounded projection could be felt in the floor of the mouth, but an operation having been refused, the child was lost sight of.

CASE XI. (Denonvilliers and Verneuil).—Male, aged twenty-eight years, entered the hospital for a large tumor situated in the floor of the mouth. At about the age of six he began to notice a small indolent tumor, situated in the floor of the mouth below the tongue, in the median line. In the beginning the tumor was about the size of a walnut, and has little by little increased in size. For the last two years it has apparently remained stationary, and has never given rise to any pain.

Examination shows that the growth occupies the totality of the floor of the mouth. It is about the size of a small orange, globular, and rises considerably above the level of the teeth of the lower jaw. The

tongue is completely pushed up and backward, and is hidden by the tumor. Forward projection of the tongue is absolutely impossible. There is no projection in the suprahyoid region. The mucosa covering the tumor is normal and is perfectly movable over the surface of the growth. Wharton's ducts can be seen. Palpation shows fluctuation, and does not produce pain.

Puncture of the cyst gave issue to whitish-yellow sebaceous matter. A transversal incision was immediately made into the cyst, and by pressure a large quantity of this substance was expressed, amounting to about 100 drachms.

After various treatments, which were without result, an incision was made in the mucosa and the cyst enucleated. Extirpation was easy excepting at the lower part of the cyst, where the adhesions were so tough that they had to be cut with scissors.

Histological examination showed the growth to be a dermoid cyst.

CASE XII. (Cruveilhier.)—Male, aged sixty-two years. On examination of the sublingual region a rounded tumor occupying the median line was found. The growth was about the size of a small russet apple and pushed the tongue upward against the roof of the mouth. The tumor could not be felt in the suprahyoid region. Fluctuation was distinct. Transparency of the growth was absent. The patient died shortly afterward from bronchitis, and the examination at autopsy showed that the cyst was a dermoid.

CASE XIII. (Gallard.)—Female, aged twenty years, presented in the floor of the mouth below the tongue a tumor the size of a hen's egg. The patient was quite sure that she had not noticed it before the age of twelve years.

The tumor appeared thin, elastic, and by palpation gave rise to the sensation of false fluctuation, but by pressure with the finger the tumor kept its imprint for a few minutes. The growth appeared to interfere with phonation and deglutition, and pushed the tongue toward the roof of the mouth, and at the same time formed a slight projection in the suprahyoid region.

The cyst was punctured and gave issue to a white, sebaceous matter, and in order to avoid the cyst a strip of the walls was excised from the anterior wall, after which the contents were easily removed.

Histological examination by Prof. Cornil showed that the growth was a dermoid.

CASE XIV. (Bryant.)—Male, aged twenty-five years, was sent to the hospital with an inflamed tumor, situated under the tongue, which the patient had noticed for the first time about six years previously. A seton was inserted, but the tumor had never disappeared. It had never caused any trouble until six months ago, when it began to increase in size, so that it became troublesome. A surgeon opened the growth and gave exit to a hard matter, and the entire growth began to be inflamed from this time.

When Bryant saw the patient the latter could hardly speak, the tongue being pushed against the roof of the mouth by an inflamed tumor situated beneath it, and completely occupied the concavity of the lower jaw. On the left side was an opening through which a sound could be easily passed. By pressure a peculiar semi-solid, sebaceous mass with a fetid odor could be removed. The opening which already existed was increased in size, and about three ounces of the contents

were evacuated, after which the walls of the cyst became visible and were found smooth and inflamed.

The patient was ordered to frequently wash out the mouth with hot water, and when he returned some time later the condition of affairs was relatively good. The cyst finally became completely empty and the inflammation subsided.

CASE XV. (Bryant.)—Male, aged nineteen years, with a tumor situated underneath the tongue, in the median line, which was first noticed about six months ago. It had gradually increased in size, and latterly had become so large that the patient could hardly speak. The tongue was pushed toward the roof of the mouth by a large cyst, which projected equally on both sides of the frænum. Wharton's ducts could be seen passing over the growth.

The tumor was incised, its contents evacuated, and the cavity plugged with charpy. Inflammation took place, and an apparent cure was obtained.

CASE XVI. (Neumann.)—Male, aged fifty-two years, had had since birth a growth which had only taken on a notable increase in size during the last few years, and finally had obtained such a volume that the tongue was pushed back toward the pharynx, producing great disturbances in deglutition and speech.

By inspection of the mouth the tongue could no longer be seen, and in its place there was found a fluctuating tumor projecting out of the mouth. A small piece of the anterior wall of the growth was excised, and microscopically a dermoid cyst was diagnosed.

CASE XVII. (Verneuil.)—Female, aged eighteen years. The time at which the cyst first made its appearance was uncertain, but it certainly had been noticed three years before the operation. The cyst was the size of a hen's egg, indolent, non-transparent. It did not disturb either speech or mastication, and it projected in the suprahoid region.

Puncture gave issue to about 30 grammes of a grayish, thick liquid. Examination of the buccal cavity showed that the cyst was situated under the tongue, in the median line. It was partly enucleated with the finger through an incision in the median line of the floor of the mouth, but it was quite adherent to the anterior aspect of the os hyoid.

Histologically the growth was a dermoid cyst.

CASE XVIII. (Meunier.)—Male, aged fifty-two years. Examination showed a large cyst occupying the floor of the mouth, but at no time had the growth given rise to any trouble in speech or deglutition.

At the operation the cyst was found to occupy the median line, immediately behind the lower jaw, below the tongue, above the mylohyoid muscle, between the genioglossal muscles, which could be seen sliding over the sides of the growth. The tongue was movable in every direction, was pushed up over the level of the teeth, and when it was pushed backward it caused the tumor to project upward and forward, producing a projection under the chin when the tongue was carried forward. The mucosa was movable over the growth. The tumor was about the size of a large mandarin orange; it was smooth, soft, rounded, fluctuating, and painless. Wharton's ducts could be seen, and maintained their normal relation to the tongue.

The cyst was removed by a longitudinal incision of the mucous membrane.

A careful histological examination showed the growth to be a typical dermoid production.

CASE XIX. (Nicaise).—Female, aged three years. Presented a dermoid cyst, situated on the under surface of the point of the tongue and somewhat to the left of the median line. It was about the size of a walnut. The growth hindered the movements of the tongue.

An incision was made on the under aspect of the tongue, and the cyst was dissected out. The growth was found to be connected to the median raphe of the organ by a very resistant hard cord, which was inserted perpendicularly to the posterior aspect of the lower jaw in the median line. This fibrous cord was also dissected out, and was found after removal to have a fibrous consistency, while its axis contained a narrow canal whose walls were very similar to the skin.

CASE XX. (Combalat).—Male, aged thirty-five years. Had had as long as he could remember a tumor the size of a cherry in the floor of the mouth, situated in the median line. From the age of six years the patient had noticed a very slow though steady increase in the size of the growth, but it was only latterly that mastication and speech had been difficult. Examination showed that the growth occupied the floor of the mouth and completely filled the concavity of the lower jaw. It extended back as far as the last molar teeth. When examined with the mouth closed a small tumefaction was noticed in the suprahyoid region. With the mouth open the end of the tongue was pushed back and upward, so that it came in contact with the roof of the mouth. The mucosa covering the growth was pale, dry, and looked like parchment. By palpation the tumor was soft and pasty to the feel, while an indistinct fluctuation could be made out. The orifices of Wharton's ducts could be seen.

Extirpation was performed by an incision through the mucous membrane. Microscopically the growth proved to be a typical dermoid cyst.

CASE XXI. (Bazy).—Male, aged thirty-one years. Had noticed a tumor in the suprahyoid region which had developed during the last year. On examination a growth the size of a small egg was found, being about six centimetres long in its longest diameter and from three to four centimetres in its transversal diameter. It was oval in shape and flattened antero-posteriorly. It appeared to be bound down to the suprahyoid region, and extended as far as the os hyoid, to which it was adherent, while its upper extremity was less adherent to the surrounding tissue. The skin was not adherent to the tumor. The growth projected above the floor of the mouth, and by palpation its surface was found smooth and rounded, without any adhesions to the mucous membrane. All functional signs were absent. The diagnosis was dermoid cyst of the suprahyoid region.

A vertical incision in the median line was made, extending from the lower jaw to within three centimetres above the os hyoid. The growth was easily dissected out, and only adhered to the os hyoid. Its dissection at this point was difficult.

Histologically the growth was a dermoid cyst.

CASE XXII. (Bazy).—Male, aged nineteen years. Had noticed for the first time eight months previously a small tumor in the region of the os hyoid. It was painless and rolled under the skin. The growth had developed slowly, and in eight months had acquired the size of a walnut. Its surface was regular, it was situated in the median line,

while its consistency was somewhat firm and gave a sensation as if the walls of the growth were distended by soft contents. The skin was movable over the growth, and appeared normal. The tumor was adherent to the deeper structures, particularly to the os hyoid. It did not form any appreciable projection into the inside of the mouth.

An incision was made in the median line from the jaw to the thyroid cartilage. The growth was dissected out with difficulty, on account of numerous adhesions to the muscles and to the os hyoid. Suture. No drainage. Excellent recovery.

CASE XXIII. (Gerard-Marchant.)—Female, aged nineteen years. Had had for many years a tumor the size of a pigeon's egg in the median line, just above the os hyoid. The growth adhered so intimately to the latter that it was impossible to insert the finger between it and the cyst.

The growth was removed by a suprahyoid incision, and was found so adherent to the os hyoid that it was necessary to draw the latter out of the incision in order to accomplish this end.

The walls of the cyst contained air, and histological examination showed that it was undoubtedly a dermoid cyst.

CASE XXIV. (Duplay.)—Female, aged seventeen years. Had noticed for the first time a few months previously a tumor in the mouth which hindered the movements of the tongue. No pain had preceded the development of this growth. It was about the size of a hen's egg, and pushed up the floor of the mouth from the lower jaw to the insertion of the tongue. It was situated exactly in the median line. The mucosa covering it was normal, and perfectly movable. The growth projected downward between the muscles and formed an elevation in the suprahyoid region. It was very movable, and in no way connected with the os hyoid or the jaw. Fluctuation could be elicited. Operation showed the growth to be a dermoid cyst.

CASE XXV. (Lediard.)—The cyst was removed from the tongue of a child, aged six years; it had been present since birth, but had only lately increased in size, so that the lips could only with difficulty be brought over the tongue. The mouth was kept open and the end of the tongue projected between the lips. Speech was very indistinct, and some of the incisor teeth had fallen out on account of pressure. The alveolar borders of the lower jaw were pushed forward for the same reason, and the frænum of the tongue was ulcerated. The cyst occupied the centre of the tongue and extended backward into the neighborhood of the os hyoid. It was easily enucleated, and the child rapidly got well.

Microscopical examination showed that the contents consisted of mucous corpuscles, crystals of phosphates, and cholesterin. The growth was a dermoid cyst.

CASE XXVI. (Schmit.)—A young man noticed the presence of a tumor under the tongue. There was no pain and no disturbances of speech or swallowing. With the mouth closed a rounded tumor was seen in the median line, in the suprahyoid region, extending slightly over to the left. The skin covering the growth was normal. With the mouth open and the tongue raised up the growth was found to disappear from the suprahyoid region and projected under the tongue. The mucous membrane covering it was normal and did not adhere to the growth.

The growth was removed through an incision in the mucous membrane, and did not adhere either to the os hyoid or the lower jaw.

CASE XXVII. (Lannelongue).—Male, aged ten years. Presented a tumor in the median line, in the suprahyoid region, which extended over to the right. The growth was about the size of a small mandarin orange, and raised up the cervical region behind the chin. It was perfectly regular in shape, and was situated under the aponeurosis, without any adhesion to the skin. It appeared to be adherent to the os hyoid. Fluctuation was difficult to elicit.

A median vertical incision was made, and it was necessary to incise the muscular layer in order to expose the growth. It was easily dissected out, as it was nowhere adherent.

Histologically the walls of the cyst were covered with epidermis, but no air glands or papillæ were discovered.

CASE XXVIII. (Guinard).—Male, aged seventeen. Had noticed two years ago for the first time a tumor the size of a small walnut in the suprahyoid region. In a few months it had increased so in size that it projected up in the floor of the mouth. When seen the growth had attained the size of an egg and pushed the tongue upward and backward. It was situated in the median line, and appeared to be bound to the symphysis of the lower jaw. The mucous membrane covering the growth was normal and did not adhere to it. Wharton's ducts were normal. Palpation showed that the tumor was manifestly fluctuating, with a round, smooth surface.

The growth was removed through a transversal incision of the mucous membrane; and was found to be solidly adherent to the apophyses geni and to the intergeniohyoid raphe. It was removed with difficulty.

Microscopically the growth proved to be a dermoid cyst.

CASE XXIX. (Barker).—Female, aged twenty-eight years. Had difficulty in swallowing for six years. Two months before coming under observation she noticed a tumor, which had increased in size steadily. It was situated in the median line, behind the symphysis of the jaw, and pushed the mucosa forward so that there was a space between the dorsum of the tongue and the lower part. It also projected under the jaw. The skin covering the growth was normal and did not adhere to it. The tongue was pushed up toward the roof of the mouth, and from this great difficulty in speech resulted. The mucosa covering the growth was thin, and large veins were scattered through it. The growth was fluctuating and indolent.

An incision was made in the median line from the symphysis of the jaw to the extent of about two and a half inches, and included the skin and raphe of the mylohyoid muscles. It was easily enucleated without opening into it, but was found solidly adherent to the os hyoid. Microscopically the growth was a dermoid cyst.

CASE XXX. (Barker).—The author has seen another case similar to the preceding one, which was also situated between the genioglossal and the hypoglossal muscles. It was not quite so large as the preceding case, and was easily removed through an incision in the mucous membrane.

CASE XXXI. (Lannelongue).—Male child, aged one year. Presented a tumor of the tongue seated in the under aspect of the organ, and occupied the two posterior thirds of this surface. The frænum was pushed to the right from the development of the growth. The

tumor was whitish-gray in color, and a few small vessels were seen on its surface. It was ascertained that the child was born with the growth, which at the time of birth was about the size of a pea and was seated more to the left than to the right. It had never interfered with nursing.

The growth was easily enucleated.

Histological examination of the cyst showed it to be a dermoid structure.

CASE XXXII. (Dardignac.)—In this case the growth was congenital in origin, and had rapidly increased in size when the subject was about twenty years of age. It was located exactly in the median line, and had contracted intimate adhesions with the periosteum of the lower jaw at the level of the apophyses geni. It was soft in consistency and absolutely indolent.

The growth was easily enucleated excepting at the point of its implantation, where it contracted the adhesions already spoken of.

Histologically it was a dermoid cyst.

CASE XXXIII. (Hofmokl.)—Female, aged twenty-eight years. During the last six months she had noticed a small movable tumor in the suprahyoid region. The growth was indolent and produced no functional disturbances. The tumor increased slowly in size, and finally projected into the mouth and underneath the tongue, and then interfered with mastication and speech. Examination with the mouth closed showed that the respiration was normal, and in the median line of the throat, just above the os hyoid, a tumor the size of a hen's egg was seen. The skin was perfectly normal, and by palpation the growth was found soft and very elastic.

Inspection of the interior of the mouth showed a tumor underneath the tongue, covered by a normal mucosa, and extending equally out on each side of the frænum. By pressing on the suprahyoid tumor the intrabuccal growth became more accentuated and tense. Fluctuation could be made out. Wharton's canals were intact.

The growth was opened through an incision in the mucous membrane, and after the sebaceous contents had been evacuated the cyst was easily enucleated excepting at a point where it was adherent to the jawbone.

Histological examination showed the growth to be a dermoid structure.

CASE XXXIV. (Gross.)—A young woman consulted the author for a tumor which had developed in the median line, in the suprahyoid region, the development of which had begun in early infancy. The growth had progressively increased in size, but had never given rise to pain or other symptoms. Examination showed a tumor projecting in the median line of the throat, just above the os hyoid. It was rounded in shape and about the size of a small apple. By palpation it was perfectly movable, and no adhesions with the surrounding structures could be made out. It was elastic, but did not fluctuate and did not project up in the floor of the mouth.

The growth was enucleated through an incision in the submental region. During the operation the cyst broke, which rendered its extirpation somewhat difficult.

Histologically the growth was a dermoid cyst.

CASE XXXV. (Gosselin.)—Female, aged twenty-one years. Shortly after birth there was noticed a small tumor under the tongue. For

fifteen years the growth remained about the size of a horse-chestnut, but for the last five years it had gradually increased, without causing pain or any change in speech.

On examination a growth the size of a hen's egg was seen situated in the median line, underneath the tongue. Its projection into the mouth was more noticeable in the left, and pushed the tongue slightly backward. The mucous membrane of the mouth did not appear to be thickened, and presented a grayish hue, with rosy areas. The growth was smooth, rounded, and soft, but was not pasty to the feel, and almost gave rise to the sense of fluctuation. No finger-print remained when pressure was made.

Curved incision of the mucous membrane, which was dissected off the anterior aspect of the cyst, which was easily enucleated excepting at the lower part, where the growth had contracted solid adhesions of a fibrous nature.

A careful histological examination showed that the growth was a dermoid cyst.

CASE XXXVI. (Reclus).—Male, aged thirty-one years. Had noticed, three years previously, a rounded, movable tumor, the size of a walnut, which had commenced to push the tongue upward. It had increased slowly without producing any pain or functional disturbances. In a year's time it was the size of an egg, and in two years about the size of an orange. At the end of three years it had reached the size of an adult fist. It projected both under the floor of the mouth and in the suprahypoid region.

The surface was smooth and regularly rounded, and was situated under the muscles and skin, which was movable over the growth, and normal. The tumor projected markedly behind the dental arch, pushing back the tongue against the roof of the mouth. The mucous membrane was in a hyperæmic condition and thickened, but did not adhere to the growth. Fluctuation was evident, respiration easy, deglutition somewhat interfered with, mastication was possible, and the voice had only undergone a slight change in tone.

An incision was made in the suprahypoid region, in the median line, over the most prominent point of the growth. This was punctured and gave issue to 400 drachms of sebaceous material characteristic of the contents of a dermoid cyst. The opening thus made soon closed and the cyst began to distend again.

Three months after the latter operation the growth suddenly took on a rapid increase in size, accompanied by a sharp pain and an inflammatory reaction, so that the mouth was completely obliterated and the patient could not even swallow liquids.

When we saw the patient, two days later, the condition was alarming, although the respiratory functions were practically intact; but the man could only be nourished through a stomach-tube passed by the nose. The cyst appeared larger than when first seen, so much so that it filled the mouth and the patient could not close the jaw.

As extirpation was urgent, it was immediately done.

A careful histological examination showed the growth to be a dermoid cyst.

CASE XXXVII. (Reclus).—Male, aged twenty-five years. Had always noticed that his throat was a little large at its upper part. The suprahypoid region was tense and slightly prominent. Little by

little the tumefaction increased, and when the patient was seen a rounded tumor in the suprahyoid region was found. The growth was limited by the concavity of the lower jaw and by the os hyoid. When the patient opened his mouth the tongue was seen pushed up and pressed against the roof of the mouth. Pressure made on the growth within the mouth caused the growth to bulge in the suprahyoid region.

Enucleation was easy excepting in the region of the os hyoid, to the posterior aspect of which it was adherent.

Histologically the growth was a dermoid cyst.

CASE XXXVIII. (Sutton.)—Male, aged twenty-four years. Presented himself with a tumor occupying the floor of the mouth and situated in the median line. During the lapse of nine years he had undergone seven operations, because the cyst had been wrongly diagnosed as ranula.

The cyst was dissected out, but was so intimately connected with the os hyoid that a periosteal elevator was necessary to peel it off.

Histological examination of the cyst showed that it was a dermoid structure.

CASE XXXIX. (Ferron.)—A young soldier complained of difficulty in breathing, which had been present for several years. During the examination of the nose and throat the tongue was found to be pushed against the roof of the mouth by a tumor the size of an egg, occupying the floor of the mouth and projecting more in the left. The growth did not make any appreciable projection in the suprahyoid region. It was regular in form, smooth, and not transparent. It distended the mucosa, whose color was normal. The tumor was soft, and gave rise to an indefinite fluctuation. It hindered the movements of the tongue, which were otherwise normal.

After puncture sebaceous matter made its exit, and the cavity of the cyst was swabbed out with a solution of nitrate of silver, and a drainage-tube was inserted. The result of this was that an acute inflammatory process was set up, which finally resulted in the spontaneous elimination of the emptied cyst.

CASE XL. (Paget.)—A cyst was situated under the tongue of a small girl, occupying the median line of the floor of the mouth, and had the appearance of an abscess. It had been incised twice, which had given exit to a thick liquid similar to pus. As the trouble had recurred, the growth was dissected out and was found to be a cyst measuring about three-quarters of an inch in diameter, with an internal surface composed of an epidermis from which grew a few fine hairs. The papillæ of the chorion and that part of the chorion situated below the rete Malpighi were impregnated with a black granular pigment, but the rete Malpighi itself was not pigmented.

CASE XLI. (Farwell.)—Female, aged seventeen years, with tumor, situated under the tongue, about the size of a small orange. It filled the mouth, and pushed the tongue toward the roof in such a way that the patient could not swallow solid food. In every other way she was well, but stated that she had always had a small enlargement under the tongue as far back as her memory could go, and that the growth had increased in size only during the last three months. A physician had incised the tumor, but the opening closed up after this operation, and the tumor had increased in size so that it was larger than before this operation.

The anterior wall of the growth was seized with forceps and, after excision of a large portion of the anterior wall of the cyst, a large quantity of thick, sebaceous matter containing hair was evacuated. The wound rapidly healed, and nine months after there was no recurrence.

CASE XLII. (Gerard-Marchant.)—Female, aged twenty-four years; had had from birth a tumor which projected into the mouth and under the chin. The growth had little by little increased in size, and had now attained such a volume as to functionally disturb the patient. Local examination showed a tumefaction in the suprahyoid and sublingual region. A median projection existed, which pushed forward the skin of the submaxillary region and projected under the sublingual mucosa. Both mucous membrane and skin were perfectly normal and in no way adherent to the growth.

By palpation the cyst appeared freely movable, and could be pushed up and down from the suprahyoid region into the buccal cavity, and vice versa. It was rounded and about the size of an egg, but the entire outline and growth were difficult to ascertain because part of it was deeply situated. Fluctuation could not be elicited, but the growth seemed elastic. Pressure with the finger on the growth left no imprint. The tumor moved with the os hyoid during deglutition, and if at this time an endeavor was made to separate the growth from the bone it was found adherent to the latter.

The cyst was dissected out by an incision in the suprahyoid region and was found situated between the geniohyoid and genioglossal muscles, surrounded by a loose cellular tissue and easily dissected out with the finger, excepting in the region of the os hyoid, to which it intimately adhered by bands of fibrous tissue.

A careful and detailed histological examination showed the growth to be a dermoid cyst.

In looking over the literature of dermoid cysts of the floor of the mouth I have been able to find a number of other instances, but as no histological examination had been made, and as they were recorded superficially, I have thought best not to include them in the list of cases here reported, fearing that they might not be true dermoid cysts. Cysts of the tongue of a dermoid nature are extremely rare, and I have only been able to find two cases where I think, from the microscopical examination, that the growth was a true dermoid: one was reported by Verchère and Denucé, the other by Lediard. Other cases of congenital cysts of the tongue have been reported by Gerster, Hadden, and others; but, from the careful perusal of the published reports of these cases, I am more inclined to believe that they were, without any doubt, mucoid and not dermoid productions.

REVIEWS.

PROGRESSIVE MEDICINE—A QUARTERLY DIGEST OF ADVANCES, DISCOVERIES, AND IMPROVEMENTS IN THE MEDICAL AND SURGICAL SCIENCES. Edited by HOBART A. HARE, M.D., assisted by H. R. M. LANDIS, M.D. Vol. IV., December, 1901. Philadelphia and New York: Lea Brothers & Co., 1901.

THE discontinuance during late years of the *Index Medicus* has put upon such reviews of medical and surgical progress as *Progressive Medicine* an increased responsibility and value, since upon them the busy man must depend not only for his information regarding current literature, but to them he must also look for reference to articles which he may wish to read in full.

The first portion of this last volume of 1901 deals with Diseases of the Digestive Tract, and presents much that is of interest both to the medical man and the surgeon, since under this head is presented the late literature relating to gastric ulcer, intestinal perforation, appendicitis, etc. The interest which the profession has lately taken in diseases of the pancreas, and the progress which has been made in the treatment of these, is shown by the number of writers quoted and the results obtained by surgical treatment. We regret to note the omission of J. Collins Warren's very important article on the subject of "The Surgery of the Spleen" (*Annals of Surgery*, May, 1901) in the brief page allotted to this organ.

The chapters devoted to Genito-urinary Diseases impress us as being of the greatest value, presenting, as they do, numerous references to recent literature, and the author has shown most excellent judgment in his choice of articles worthy of review. Certainly few papers pertaining to the subject have escaped his notice. The small amount of space, however, which is given to the subject of syphilis is noticeable.

Following this part of the book is that which deals with Anæsthetics, Operative Technique, and Wound Treatment, Infection, Diabetic Gangrene, and Examination of the Blood in Surgical Cases. Although only the subjects above mentioned are dealt with, this division of the book has the heading "Anæsthetics, Fractures, Dislocations, Amputations, Surgery of the Extremities, and Orthopedics." This, we feel, is a mistake, and is apt to be misleading, and perhaps disappointing to the reader. The chapter on Anæsthetics is highly to be commended, as it presents in readable and comprehensive style practically all the recent and good literature on the subject. The portion relating to local anæsthesia is rightly given considerable space, in which the technique of the procedure is carefully detailed. The chapter on Examination of the Blood in Surgical Cases is extensive, and contains much that is of interest from a diagnostic point of view.

The next division of the book relates to Diseases of the Kidneys, and under this heading are discussed Oxaluria, Functional Albuminuria,

Urinary Changes in Gastro-enteritis, Bright's Disease in the Young, the Varieties of Uræmia, etc.

The three last divisions of the book deal with Physiology, Hygiene, and Practical Therapeutics. Nothing strikingly new appears under the head of Physiology, but much that will be found of interest in regard to Metabolism, Absorption, the Nervous System, etc. The portion on Hygiene presents the literature of the year relating to Tuberculosis, Yellow Fever, the Restriction and Prevention of Typhoid Fever, etc. The closing chapter discusses in a clear way the recent advances made in Practical Therapeutics, and will be found of value to those seeking new knowledge in this department of medicine.

J. H. G.

A SYSTEM OF PHYSIOLOGIC THERAPEUTICS. Edited by SOLOMON SOLIS COHEN, A.M., M.D. Climatology, Health Resorts, Mineral Springs, by F. PARKES WEBER, M.A., M.D., F.R.C.P. (Lond). Physician to the German Hospital, Dalston; Assistant Physician to North London Hospital for Consumption; author of "The Mineral Waters and Health Resorts of Europe," with the Collaboration for America, by GUY HINSDALE, A.M., M.D., Secretary of the American Climatological Association; President of the Pennsylvania Society for the Prevention of Tuberculosis; formerly Lecturer on Medical Climatology in the University of Pennsylvania. Vol. III., pp. xiii., 336. Vol. IV., pp. xiii., 420. Illustrated with maps. Philadelphia: P. Blakiston's Son & Co., 1901.

THERAPEUTICS implies the application of remedial measures in the treatment of disease; it is not limited to drugs. These volumes, forming a part of a series, deal with climatology to form one phase of what the editor designates as a practical exposition of the methods, other than drug-giving, useful in the prevention of disease and in the treatment of the sick. Thus far the subject discussed falls under the title of "Therapeutics;" but to assume that these non-drug methods cover all that is conveyed by the term "Physiologic Therapeutics" is to ignore the plain teachings and accepted literature of pharmacology. We like the idea of grouping together mechanical and gross physical agencies, for with the gathering together of *ex parte* arguments by various eager and presumably qualified persons we can more readily ascertain the basis of their claims and test the soundness of the logic by which they reach their conclusions. We approve of making use of every aid upon which we may levy for the lessening of human misery, and in the present enlightened condition of the profession we are not over-particular as to its source, even if a given method has been exploited by charlatans. Thus, electricity, mechanotherapy, and pneumatotherapy have already come, and hydrotherapy is now coming, into legitimate use. But with their use by intelligent and discriminating practitioners the glamour of their results becomes dimmed, and the logic of established facts soon determines their true worth. Each and every one ceases to be a panacea, but each and every method is capable of rendering more good to the world at large because of its more intelligent application. As a matter of fact, volumes

like these are of twofold service—they demonstrate to the physician that outside of his particular environment are agencies capable of accomplishing that for which he may possibly be striving in vain, and that no one place is sole proprietor of all the favorable auspices under which patients may live. Further than this, the very conscientiously gathered information and the transparent frankness of the authors impress the reader with the variety of health resorts and the good which their judicious election may entail. The first part consists of fifty pages devoted to Physics, Physiology, and General Therapeutics of Climate; next comes the bulk of the work, covering a detailed description of health resorts in every quarter of the globe, some presented on suspicion; and, finally, the third part of 140 pages, indicating how particular diseases and conditions may be benefited by a sojourn at various localities. Anyone who considers that this is a compilation of undigested and indigestible material gives little credit to the patience, discrimination, and learning of the authors. Whoever expects to find extravagant claims or unwarranted laudation will look in vain. One who searches for evidences of credulity on the part of the authors will be disappointed. The volumes are conscientious and adequate, full and yet compact. If we might yield to national pride, we would say that the work done by the American Climatological Society has borne mature fruit, and young as we are we have more information of practical bearing than has accumulated during centuries of old-world observation. Among the best of good work we would put in a prominent place "The Hawaiian Islands," by Coan, written quite likely *con amore*, a model of terse statements and clean cut, idiomatic English. We find but little in the book for adverse comment: "Everyone nowadays admits that tabes dorsalis and general paralysis of the insane seldom occur in persons who have not had syphilis" (vol. iv., p. 280) hardly correctly states the facts, and the classing of hydrotherapy (vol. iv., p. 313) as preventive treatment of pulmonary tuberculosis scarcely reaches the exaggerated height of our water-curists. In many places we note the belief of the authors that drugs are not without effect even in health resorts. In conclusion, we know of no better source than these volumes which the physician may consult in the interest of his patients, or the average reader peruse, that he may add to his store of information. R. W. W.

THE HYGIENE OF TRANSMISSIBLE DISEASES: THEIR CAUSATION, MODES OF DISSEMINATION AND METHODS OF PREVENTION. BY A. C. ABBOTT, M.D., Professor of Hygiene and Bacteriology, and Director of the Laboratory of Hygiene, University of Pennsylvania. Pp. 350. Philadelphia and London: W. B. Saunders & Co., 1901.

THE second edition of this most valuable and interesting book omits nothing of importance which the advances in medical research have brought out on the subject of transmissible diseases since the appearance of the first edition two years ago. The chapter on Typhoid Fever is enlarged and contains some new and instructive charts. That on Dysentery is revised and enlarged, and includes a discussion as to the propriety of the ordinary usage of the term. In the consideration of

prophylaxis in tuberculosis statistics are given which show the very large percentage of deaths from this disease in a half-dozen of the large cities of the United States—six and more times as great as that from four of the leading so-called zymotic diseases combined. The author advocates the education of the patient, so that he may not become a public menace, the establishment of public sanatoria for such as cannot look after themselves, and the registration of all cases, whatever their clinical manifestations. The chapter on Bubonic Plague is largely rewritten, and includes new sections on protective inoculations, special quarantine measures, and the rôle of rats in its dissemination. Especially interesting in the chapter on Malaria is the discussion of the rôle of the mosquito as the disseminating agent. The chapter on Yellow Fever covers the advances in our knowledge of the etiology of this disease due to the researches of medical officers of the United States Army and Marine Hospital Service. In the consideration of other of the transmissible diseases whatever is new has been incorporated, and the whole work is quite abreast of the times. The book is commendable in the highest degree in all respects, and should be in the hands of every progressive practitioner and public health authority. C. H.

ANÆSTHETICS AND THEIR ADMINISTRATION: A TEXT-BOOK FOR MEDICAL AND DENTAL PRACTITIONERS AND STUDENTS. By FREDERICK HEWITT, M.A., M.D. Cantab., Anæsthetist to His Majesty the King; Anæsthetist and Instructor in Anæsthetics at the London Hospital; Late Anæsthetist at the Charing Cross Hospital, and at the Dental Hospital of London. Second edition. New York: The Macmillan Co.

THE second edition of Hewitt's work is substantially a new one, as it has been thoroughly revised and its scope extended so as to include the history and experimental physiology of general anæsthetics, and we may here suggest that the title be changed to "General Anæsthetics and their Administration," as they alone are discussed. As the author states, a student's opportunities for becoming a proficient anæsthetist are lamentably inadequate. Experience is a vivid and impressive teacher, but it is hard on the patients. Hewitt's book will supply the want felt by a lack of experience as well as any book can. While purporting to be a work for the advanced student, we believe it will be of greater value to the casual anæsthetist in private practice and the evanescent one of our hospitals. With great detail, with many references to literature, and with the citation of illustrative cases from an enormous experience, to carry conviction, the chemical, physical, physiological, and clinical aspects of this important subject are dealt with. This prolixity on some of the pages may be adversely criticised. Frequent repetitions and cross-references, however, add to our understanding, even though they sacrifice our time. There are 500 clear-typed pages, containing 59 illustrations, mostly of apparatus. The index is above the average, and will help to balance the arrangement of the book, which is below the average.

The writer points out the analogies between simple anoxæmia and general anæsthesia, and thinks that future experimentation may lead

us to conclude that general anæsthesia is produced by a limitation of oxidation upon which the nerve centres depend for the execution of their functions. The much-disputed point whether chloroform has or has not a depressing effect on the heart is settled in the affirmative. Nitrous oxide and ether are regarded as the routine anæsthetics, the former for very short, the latter for longer operations; but routine is set aside in certain cases, owing to the condition of the patient or nature of the operation, both of which are elaborately considered in that portion of the treatise dealing with the selection of anæsthetics. The fact that children are as susceptible as adults to the toxic effects of chloroform is pointed out. Concerning nitrous oxide and oxygen the writer says the anæsthesia is comparatively light, inconvenient reflexes are prone to arise, and that it may be recommended in carefully selected cases, but not for general use, although it is the safest method known, no fatalities having been recorded. Ethyl chloride is said to be less suitable than other anæsthetics, owing to the fact that the muscular system does not, as a rule, become relaxed. The management and treatment of the difficulties, accidents, and dangers of general anæsthesia are gone into with as much thoroughness as possible, sixty pages being thus consumed. We fear too much stress has been laid on vigorous traction of the tongue, which, in itself, may inhibit cardiac and respiratory action, and not enough on the value of atropine.

Americans will object to the late Dr. Evans being spoken of as an English dentist.

To crystallize our opinion of this edition, we may say that it is an exceedingly valuable work, which may be studied with profit by surgeons, specialists in anæsthetics, hospital residents, and all others who may be called upon to administer general anæsthetics. F. T. S.

DISEASES OF THE HEART: A CLINICAL TEXT-BOOK FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE. By EDMUND HENRY COLBECK, M.D., Physician to Out-patients at the City of London Hospital for Diseases of the Chest; Late House Physician at St. Mary's Hospital, etc. London: Methuen & Co., 1901.

THE diseases of the heart and circulatory system must always be a subject presenting serious difficulties to the student of medicine, and even to the practitioner whose early training has not given him the advantages of an extensive hospital service under competent clinical teachers.

The author of the present volume, who has served his apprenticeship at the right hand of so brilliant a clinician as Sir William Broadbent, has thus approached his task with an adequate conception of the inherent difficulties that present themselves to the student. As a result he has succeeded in producing a very concise and comprehensive study, which may be taken to exemplify the latest teaching of the English school.

It is no disparagement of his work, however, to express the doubt that the average undergraduate student of medicine is sufficiently prepared

to make the best use of a text-book that so exhaustively covers the subject as the volume before us. On the other hand, it is a pleasure to note that it is admirably suited to the needs of the practitioner and the clinical worker who has already mastered the broad principles of the subject and looks for a comprehensive guide in his bedside studies of the heart and its diseases.

The treatise covers 336 pages of closely printed text. Controversial matter has been to a large extent omitted, the most generally accepted theories being given their due prominence, and the confusion attaching to the discussion of opposing views being largely avoided. The style is clear and forcible, and the division and classification of the subject-matter is made more striking by skilful management of headings in boldface type.

The chapters on Methods of Diagnosis, the Pulse, and Diseases of the Myocardium are especially noteworthy for breadth of grasp and exhaustiveness of treatment.

Altogether, this is one of the best books on the subject that has recently appeared, and it deserves, as it will doubtless command, a goodly measure of success.

T. S. W.

A TEXT-BOOK OF GYNECOLOGY. Edited by CHARLES A. L. REED, A. M., M.D., President of the American Medical Association; Gynecologist and Clinical Lecturer on the Surgical Diseases of Women at the Cincinnati Hospital; Fellow of the American Association of Obstetricians and Gynecologists, etc. New York: D. Appleton & Co., 1901.

IN the preparation of this work the author, while aiming to produce a volume which should be entirely up to date, did not intend to simply collect a number of papers by various authorities. He therefore, while inviting the co-operation of a large number of men in its preparation, has carefully arranged their contributions with the idea of producing a unity of style and a consecutiveness of statement. In the present state of rapid advancement in the subject there seems to be no other way to accomplish his aim. Of course, a certain amount of individuality is wanting, but his work has been done so well that this is hardly noticeable, and the advantages gained far outweigh, in our opinion, any possible disadvantages.

The scope of the volume is a large one, as the author departs from the narrow limits of the older books on gynecology, and, in accordance with the recent works on the subject, extends his boundaries to include the majority of surgical conditions found in woman between the diaphragm and the pelvic outlet. He thus includes a very complete chapter upon the urinary affections, and gives considerable space to a discussion of the kidney. Regarding this organ, the treatment when its mobility demands an operation is limited, by the preference of the author, to an actual stitching to the fascia—a narrowness of view which seems rather unfortunate when we consider the success following the use of the gauze pack and the rubber drainage-tube, methods which do not seem to have the subsequent dangers of the stitch.

A chapter on the History of Gynecology is of interest as showing how little is really new under the sun; while the chapter on the Gen-

eral Pathology of the Reproductive Organs as compared with general changes occurring in other parts of the body will well repay careful perusal. In his chapter on the General Medicinal and Local Treatment of Disease he gives an account of pelvic massage, and sums up the dangers of this treatment, which promised so much at the first, but which, as he well points out, has but a limited scope. The difficulties in determining the proper cases for its employment are mentioned, and a much-needed caution is given as to the dangers incident to pelvic examinations under ether in a case in which the type of pain would indicate the possibility of the presence of pus.

The close relationship existing between the subjects of obstetrics and gynecology is acknowledged by the inclusion of a chapter on Cæsarean Section, which adds considerably to the value of the book.

A chapter on Tubal Infections, in which, among other methods of treatment, a description of the cases suitable for vaginal puncture is included, is of great interest and value.

Any review of this work would be incomplete if the last chapter were passed over without reference. There is probably no one part of the whole subject which has given rise to more acrimonious discussion, to say nothing of the unnecessary suffering entailed upon a very unfortunate class of women, than the question of the interdependence of nervous conditions and gynecological disease. In the last chapter the subject is considered from the stand-point of the gynecologist, hysteria and neurasthenia being the two phases considered, and the conclusions deduced as the result of the large experience of the author will appeal to the thoughtful physician as sound.

W. R. N.

A TREATISE ON PLAGUE: THE CONDITIONS FOR ITS CAUSATION, PREVALENCE, INCIDENCE, IMMUNITY, PREVENTION, AND TREATMENT. By MAJOR GEORGE S. THOMSON, M.B., M.Ch., M.O.A. (Roy. Univ. Irel.), I.M.S. (Bombay); and DR. JOHN THOMSON, M.R.C.S.I., L.R.C.P.I., Special Plague Officer under the Government of Bombay. Pp. 299. London: Swan Sonnenschein & Co., Ltd., 1901.

THIS work is essentially a plea for improved sanitation as a defence against plague; for cleanliness, ventilation, and avoidance of overcrowding in place of routine disinfection. It is very largely an elaboration of Pettenkofer's argument that for the raising of crops two factors are essential, namely, the seed and the proper soil, neither of which alone can produce results. It contains much that is clinically, historically, and otherwise interesting, but not a great deal that is new. No topic germane to the subject has been neglected, but the arrangement is exceedingly bad, and the whole work lacks connection. Many subjects which might well be considered by themselves in separate chapters are treated piecemeal here and there, so that much of the book is a hopeless jumble; and, to crown all, there is no index. The first part of the book is especially remarkable for its unusually bad and difficult style, and the whole is marred by an offensive intolerance in discussion. Of both of these faults the following may serve as a fair example: "That destruction of rats is a useful palliative measure of a purely temporary benefit

no one denies—*e. g.*, Kitasato reports on the limitations of outbreaks at Kobe and Osaka, which he attributed to the measures adopted, and where it is believed the epidemic would have attained much larger proportions had not so many rats been destroyed. First it was the use of the whitewash brush; now the abuse of the rat. Imbecility could hardly go further or show greater impotency; but an imbecile's reasoning faculties were always *non est* over those of many guides, and seem only in abeyance, we can charitably suppose."

Among the most interesting portions of the book may be mentioned the account of the epidemic in Satara City, which gives a graphic description of the mode of life of the natives, and the chapter on Haffkine's Prophylactic Inoculation, which measure, the authors believe, should be encouraged in spite of the fact that it can never be the sole remedy against the disease, and that, by leading to a false sense of security, it postpones the day of deliverance from its inroads.

C. H.

A REFERENCE HAND-BOOK OF THE MEDICAL SCIENCES, EMBRACING THE ENTIRE RANGE OF SCIENTIFIC AND PRACTICAL MEDICINE AND ALLIED SCIENCE. By Various Writers. A new edition, completely revised and rewritten. Edited by ALBERT H. BUCK, M.D., of New York City. Vol. III. Illustrated by chromo-lithographs and 676 half-tone and wood engravings. New York: William Wood & Co, 1901.

THIS volume of the *Reference Hand-Book*, beginning with about the middle of the letter C and ending with about the middle of the letter E, includes in its pages many articles on most important subjects. Among the sections especially to be noticed are those upon the Circulation of the Blood, which is ably considered by Dr. Dreyer, and the Cranial Nerves, by Dr. C. Judson Herrick. These two authorities consider these physiological topics in a way which makes their articles of great value to the physician who desires to look up points in the physiology of the subjects under discussion. To the specialist in otology the present volume will be of the greatest interest, nearly 100 of its large pages being devoted to the consideration of topics connected with the ear and its diseases, each subject being written of by an authority upon it. The illustrations of the various pathological conditions of the ear are most valuable, and, taken altogether, the section on the Ear and its Diseases may be regarded in the light of an excellent epitome or small manual on the subject. The Pathology and Treatment of Dislocations are given lengthy discussion. Buller's discussion of the Diseases of the Cornea, though somewhat brief, will prove of decided value to the general practitioner, into whose hands these cases usually fall before they are seen by the oculist. A topic of interest to all physicians is Deaf-mutism, which is ably written of by Dr. Gallaudet. It is impossible in reviewing so large and varied a collection of articles upon so many different subjects to pick out all of those worthy of particular mention. A few of the other articles which possess particular interest are those on the Decidua, by A. S. Warthin; Epilepsy, by Allan McLane Hamilton; Dietetics, by B. Franklin Stahl, and especially that on Dysentery, by W. W. Johnson.

A work of this character, so fully up to date, so complete in each individual article, so thorough in the range which it covers, possesses the very greatest value to all physicians, no matter in what branch of work they may be occupied. It constitutes a most complete encyclopedia of the medical sciences.

F. R. P.

LECTURES ON THE SUPPURATIONS OF THE MIDDLE EAR AND THE ACCESSORY CAVITIES OF THE NOSE AND THEIR INTRACRANIAL COMPLICATIONS. By DR. HENRY LUC. 8vo. pp. 500. 26 illustrations. Bal-lière, Paris, 1901.

Luc's excellent treatise adopts a broader point of view than has often been taken, and deals with all forms of pericranial suppuration, with their intracranial extension, and this not merely from the position of the surgeon executing operations within the cranium for relief of these consequences, still less of the pathologist studying their extensions and etiology only, but of the clinician actively engaged in forestalling them. Therefore, while his book appeals most to the specialist in aural and nasal diseases, it is a most valuable study for the operative surgeon and the general practitioner, however much in its specialized parts it goes beyond the limits which they would set themselves as to following his procedures.

In his treatment of acute tympanic suppuration, Luc strongly favors free incision of the drumhead with inflation by Politzer or catheter, and the instillation of carbolyzed glycerin. He then tampons lightly for drainage, using a wick of gauze strip or a series of cotton pellets, and between the daily dressings covers with a light bandage. Where this is impracticable the hot douche is to be employed. After relief of pain, at the end of one or two weeks, he substitutes peroxide, cleansing and boric insufflations.

For the chronic suppurations he differentiates the various more common forms, according as the perforation is small or large, is in the Shrapnell membrane or below, and complicated or not with polypi or cholesteatoma. He makes good use of attic irrigation and the bent probe, yet might claim more success if still more thorough in their use. While this is not to be often expected of any but the specialist, it is really well within the powers of any careful practitioner, and deserves far more vigorous commendation.

Failing success within a few weeks or months in which good local treatment has been conscientiously employed, he advises operation; first, the simple excision of the ossicles, and, if after six weeks this remains unsuccessful, then the exenteration of the tympanic cavities. This he does after Stacke's method, cauterizing the field with a $\frac{5}{16}$ zinc chloride solution, and packing with iodoform gauze, in later operations with suturing of the mastoid wound. He dresses only after eight days, as a rule, then every two to four days, and looks for a cure in two to six months.

For chronic mastoid suppuration he counsels the radical tympano-exenteration, and his descriptions of the operative technique in all these respects is almost too minutely full. In dealing with the accessory

cavities of the nose he is similarly thorough in theory, and perhaps in practice, and his technical descriptions are generally excellent. As usual, he credits to Heryng (International Congress, 1889) the priority for transillumination of the maxillary sinus which Addinell Hewson had well demonstrated four years earlier (Pennsylvania State Society, 1885); and in dealing with the acute phases of sinus inflammation has only hot fomentations and steam inhalations in his therapy, without mention of the nearly specific effect of atropia and the freeing of the natural passages by cocaine and adrenal locally.

The outcome of these various conditions in inflammation, thrombosis, or abscess-formation within the cranial cavity are excellently set forth. Rather striking at first glance are his employment of the galvano-cautery not solely for searing the infected dural surfaces of sinus or cranial fossæ before incising them, but in enlarging the opening of brain abscess and in removing the cerebral hernia; and where many operators cautiously irrigate within the dura only with boric or salt solution, he employs 1 : 2000 bichloride.

B. A. R.

THE PHYSICIAN'S VISITING LIST FOR 1902. Philadelphia: P. Blakiston's Son & Co.

THIS useful little publication will be welcomed as usual by its friends in the profession as it makes its yearly appearance. It is small, neat, and compact, and the fact that it is entering its fifty-first year speaks volumes for its popularity. Its arrangement is similar to most other visiting lists, having sets of tables for reference in the front, followed by pages for the daily record of cases (arranged for twenty-five names weekly), births, deaths, vaccinations, cash accounts, etc. Bound attractively in soft black leather, it fits the pocket easily. A visiting list is a necessity to the physician, as it is to it that he must turn for his data if the legality or justice of his charges is questioned.

G. M. C.

NERVOUS AND MENTAL DISEASES. By ARCHIBALD CHURCH, M.D., Professor of Nervous and Mental Diseases and Head of Neurological Department, Northwestern University Medical School; and FREDERICK PETERSON, M.D., Chief of Clinic, Department of Nervous and Mental Diseases, and Clinical Lecturer on Psychiatry, College of Physicians and Surgeons, New York. Third edition, revised and enlarged. Pp. 870, with 322 illustrations. Philadelphia and London: W. B. Saunders & Co., 1901.

A BOOK on nervous and mental diseases which has reached a third edition in so short a time needs no review, and has proven its right to be. This edition has been revised, new illustrations added, and some new matter, and really is two books; the part on Nervous Diseases having been written by Dr. Church, while Dr. Peterson is responsible for the section on Mental Diseases. The descriptions of disease are clear, directions as to treatment definite, and disputed matters and theories are omitted. Altogether it is a most useful text-book.

C. W. B.

CRAZES, CREDULITIES, AND CHRISTIAN SCIENCE. By CHARLES M. OUGHTON, M.D. Chicago: E. H. Colegrove, 1901.

In writing his little book on the various kinds of delusions by which the human race has been deceived from time to time, concluding with the form of imbecility which is now prevalent under the name of Christian Science, Dr. Oughton states that he has endeavored to avoid controversial discussion. It is, however, impossible to write a book of this kind in which the blasphemous and unscientific claims of Christian Science are exploited without exciting the spirit of controversy in its benighted disciples. It seems to us that it is a pity that any notice should be taken of a propaganda which exists solely for the purpose of making money for its priesthood, and thrives upon the ignorance of its victims.

The best way to effectually suppress a thing like Christian Science is to ignore it absolutely, as it deserves. It is unworthy of men of science or religion to attempt an argument on a subject which presents but one side. Dr. Oughton's book is well written, and for those who have not looked into the subject it brings forward many of the salient points of Christian Science which are merely the claims of its founder to a sort of Godhead.

Of course, the arguments of Dr. Oughton are unanswerable, but whether they will succeed in convincing the persons who would yield to such a delusion of the foolishness of their ways is open to question.

F. R. P.

THE MEDICAL NEWS VISITING LIST FOR 1902. Weekly (dated, for 30 patients; Monthly (undated, for 120 patients per month); Perpetual (undated, for 30 patients weekly per year); and Perpetual (undated, for 60 patients weekly per year). The first three styles contain 32 pages of data and 160 pages of blanks. The 60-patient Perpetual consists of 256 pages of blanks. Philadelphia and New York: Lea Brothers & Co.

THIS *Visiting List* has been before the profession for so many years and has established itself so firmly as an indispensable adjunct to the physician's outfit that it is almost superfluous to again direct attention to its merits. Its convenient size and its very practical arrangement (the pages being arranged for special data, such as obstetrical engagements, vaccinations, patients' addresses, and cash accounts) combine to form a visiting list which has fully justified its great success.

When the importance of a visiting list in its medico-legal aspect is recognized the value of as perfect a one as the present must be apparent. It should be remembered that the visiting list constitutes the physician's legal record, which alone he can present as testimony in court in cases in which he sues for the collection of money due. This renders it all important that such record should be kept with simplicity and in an easily explainable manner.

Thirty-two pages of tables of various kinds, doses, arithmetical conversion tables, etc., accompany the book, and, being well chosen and such as are of real value, add greatly to its usefulness. J. H. G.

THE MENTAL FUNCTIONS OF THE BRAIN: AN INVESTIGATION INTO THEIR LOCALIZATION AND THEIR MANIFESTATIONS IN HEALTH AND DISEASE. By BERNARD HOLLANDER, M.D. (Freiburg, B.), M.R.C.S., L.R.C.P. (London). New York and London: G. P. Putnam's Sons, 1901.

THERE is a type of mind that always takes the view contrary to that which is commonly accepted, and on the one hand makes heroes of the dead who have been relegated to low rank; or, on the other hand, tears off the mask of greatness from the world's favorites. Such persons may sometimes be necessary, but, as a rule, they get little thanks for their labor. The author seems to belong to the former class. His book is a defence of Gall, whom he ranks among the greatest of students and geniuses. He states that his book "is the first work on the subject [clearing up the mystery of the fundamental psychical functions and their localization in the brain] since the dawn of modern scientific research." The gentleman thus shows himself self-reliant if not a little egotistical. He bases his opinion on 800 cases, and has studied medical literature with much attention. The book is the product of fifteen years of investigation. May we say frankly we think those years have been largely wasted. The book is written in good faith, but lacks convincing power.

C. W. B.

THE ACCESSORY SINUSES OF THE NOSE: THEIR SURGICAL ANATOMY AND THE DIAGNOSIS AND TREATMENT OF THEIR INFLAMMATORY AFFECTIONS. By A. LOGAN TURNER, M.D. (Edin.), F.R.C.S. (Edin.), Surgeon for Diseases of the Ear and Throat, Deaconess' Hospital, Edinburgh. With forty plates and eighty-one figures. Edinburgh: William Green & Sons, 1901.

IN this handsomely got-up volume of 204 pages of text there are inserted forty plates and eighty-one figures illustrative of the subject-matter. The book may be properly described as an atlas of the anatomy and surgical pathology of the accessory nasal cavities. The illustrations are most of them photographs of frozen sections or of sawed skulls. They are particularly well executed and chosen to illustrate the points which the author presents in the text. The maxillary, frontal, ethmoidal, and sphenoidal sinuses are taken up in turn and their normal and pathological anatomy carefully considered. The particular value to be attached to Dr. Turner's work is its practical usefulness to those who are engaged in the operative treatment of diseases of the nose and throat. We know of no work of this character that so thoroughly elucidates the anatomical relations in these structures.

The portion of the book devoted to diagnosis and treatment is presented in such a concise manner that it has not been possible for the author to elaborate the subject as it deserves; but these matters have been so fully considered by Grünwald and others in their monographs that we think Dr. Turner has done wisely in sacrificing the consideration of this portion of his subject to the much more important matter that precedes it. The work should be in the hands of every specialist in laryngology and rhinology.

F. R. P.

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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On Tuberculous Strictures of the Small Intestine.—FIBIGER (*Nord. med. Archiv*, 1901, Afd. ii., xxxiv., h. 2, No. 8) reports four cases of tuberculous strictures of the small intestine, two of multiple cicatricial strictures and two of multiple hypertrophic strictures. A careful anatomical study of the first two cases leads the author to the following conclusions:

1. That the process of healing in tuberculous strictures may be so far advanced that all characteristic tuberculous tissue changes and tubercle bacilli may have disappeared. That the strictures caused by tuberculosis may be impossible to recognize in these cases on microscopical investigation.

2. That when the process of healing is less advanced, and the strictures still show ulceration, the diagnosis may still be remarkably difficult and only possible by the most minute microscopical investigation. Indeed, the tuberculous nature of intestinal strictures can be excluded only where the microscopical examination of the intestine itself, of the mesenteric glands, and of the liver has shown that these organs contain no tubercle bacilli and no tuberculous tissue changes, and where, also, inoculations of guinea-pigs have a negative result.

The author then analyzes fourteen instances of syphilitic strictures of the small intestine reported in the literature. In a large proportion of these instances the pathological investigation of the case is shown to have been insufficient to justify a positive diagnosis of syphilis, and in many the possibility of tuberculosis must be acknowledged. In two cases of hypertrophic strictures of the small intestine which have been carefully studied by the author, and were unquestionably of tuberculous origin, there was found a proliferative obliterating endophlebitis of certain veins in the mesentery. These changes were entirely analogous to those which have been described in syphilis changes, the discovery of which formed the main argument for

classing one, at least, of the best studied cases of so-called syphilitic stricture of the intestine under that heading. The author concludes: "There are in the literature only very occasional descriptions of affections of the small intestine with fibrous, retracting processes and tumor-like infiltrations of the intestinal wall. It seems natural to interpret these phenomena as scar formation in gumous nodes in the intestinal wall, and there is no reason to doubt the syphilitic nature of these cases."

This, however, does not hold for the far commoner cases of so-called syphilitic strictures of the small intestine. That some of these are of syphilitic origin is, naturally, possible; but it must be emphasized that the changes described may be ascribed to another etiology. No positive proofs have been offered that these cases were caused by tuberculosis, and it is probable that the majority were, as a matter of fact, tuberculous strictures.

The Relation Between Accidents and Diabetes.—HIRSCHFELD (*Deutsche med. Wochenschrift*, 1901, xxvii., 571), in an address before the Berliner Verein für innere Medizin, concludes that a definitely dependent relation of diabetes upon trauma is in some cases distinctly recognizable. These are instances of nervous diabetes where we must suspect that there has been a direct action upon the central nervous system. In these cases recovery or extensive improvement seems to be relatively common.

It is also possible that an accident may bring about diabetes in an individual who has previously suffered from some pancreatic disease. Finally, definite pancreatic disease may arise as a result of injury, cysts, hemorrhages, and perhaps also chronic inflammation.

An accident may also have an unfavorable influence upon an already existing diabetes, the most important being its action in determining the onset of coma. In some of these cases coma is so readily excited that there may be question as to its relation to the injury, but in minor cases the relation of the accident to the onset of the coma seems unquestioned.

It is also probable that in some instances an injury may be the determining cause of gangrene where otherwise the existing vascular changes might have pursued their course without evil result.

The capability of the majority of diabetics for heavy muscular exercise is materially diminished.

Mixed Infection of Typhoid Fever and Malaria.—FIOCCA (*Il Policlinico*, 1901, viii., M. 477) reports the interesting case of a patient who, after having suffered with unquestioned malarial fever, returned three weeks later with a continued fever which proved to be typhoid. This ran a typical course, lasting thirty-nine days. During defervescence quinine was given on account of the markedly intermittent character of the fever, but without effect. The nature of the fever was unquestioned both on account of the clinical symptoms and owing to the positive Widal test. On the eighth day after the return of the temperature to normal there occurred a chill; examination of the blood showed characteristic *estivo-autumnal* parasites.

Vigorous treatment with quinine was immediately instituted, the temperature remaining normal thereafter for seven days, following which there was a relapse of the typhoid fever of nine days' duration. During all this

period no parasites were found in the blood. On the tenth day after this there was a second characteristic malarial paroxysm, which yielded again to quinine. Parasites were again found in the blood.

The author in the beginning emphasizes the fact that the conception of a clinical picture characteristic of combined typhoid and malarial infections is an entirely false one, and in commenting upon this case he justly observes : "This case, besides offering convincing evidence of the existence in the same subject of typhoid and malarial infection, serves further to demonstrate, if, indeed, there be need of such a demonstration, that the combination of the two infectious processes in such a manner as to produce a separate clinical entity (typhomalarial) is a hypothetical conception which is not met with in reality."

Pancreatic Necrosis following Trauma.—SELBERG (*Berlin. klin. Wochenschrift*, 1901, xxxviii., 923) reports an extremely interesting case of typical acute hemorrhagic and gangrenous pancreatitis following a kick by a horse. The patient, a man, aged thirty-nine years, was kicked in the epigastrium. He lost consciousness for a time, and on recovering suffered from intense abdominal pain. This was followed by a swelling in the epigastric region and dulness in the left lower chest. Eighteen days later he was admitted to the hospital, at which time he was suffering from marked epigastric pain. There was a doughy swelling along the free border of the ribs on the left; dulness in the lower half of the left chest and over the region of the stomach; evidences of fluid in the left pleural cavity; abdomen soft, but swollen; tympany in the median line; dulness in the flanks; when lying on the left side the elevated points were tympanitic; not so, however, when he lay on the right.

A diagnosis of perforative peritonitis was made, but no operation was performed owing to the hopeless condition of the patient, who died two days later. On autopsy, besides the pleural effusion, numerous fat necroses were found in the omentum; the head of the pancreas was wholly necrotic, the rest infiltrated with blood. The microscopical investigation of sections of the pancreas showed that the entire organ was necrotic, as well the glandular as the fatty tissue; the lymph nodules were well preserved.

The author ascribes the necrosis either to the escape of pancreatic juice—in support of which is the fact that the necroses were more numerous in the neighborhood of the wound—or to the possibility that necroses arose at a point or points as a direct result of the wound, into which bacteria afterward entered.

The Acid Intoxication of Diabetes in Its Relation to Prognosis.—HERTER (*The Journal of Experimental Medicine*, October 1, 1901, vol. v., No. 6, p. 617), in calling attention to the fact that it has been firmly established that diabetic coma is regularly associated with the excretion of large quantities of organic acids, states that comparatively little attention has been paid to the excretion of these acids during the period preceding the onset of coma. He reports an interesting series of cases in which the oxybutyric acid has been estimated, with especial reference to its prognostic importance.

The conclusions drawn from the study of these cases are as follows :

1. A careful balancing of the normal acids and bases of the urine makes it possible not merely to detect the presence of organic acids in the urine, but also to determine approximately the amount of such acid. The method recently described by Herter and Wakeman can be recommended as securing a greater degree of accuracy, for the amount of labor involved, than any other procedure.

2. The determination of the N of NH_3 is a useful procedure for clinical purposes, since it is probably true that a considerable excretion of organic acid (say 15 grammes oxybutyric, or more, in twenty-four hours) is always attended by an increased excretion of NH_3 . As much organic acid as corresponds to 10 grammes oxybutyric acid may be excreted in twenty-four hours without causing an increased excretion of NH_3 . We cannot rely on the ammonia output to detect moderate quantities of organic acid.

3. Where organic acids are removed in considerable amount without increasing the excretion of NH_3 , the acid takes out other alkalies, probably in some instances chiefly K.

4. In cases of diabetic coma the urine always contains a large excess of organic acids, and the N of NH_3 is usually increased to 18 to 25 per cent. of the total N.

5. Crotonic acid can regularly be obtained from the urine of patients in diabetic coma.

6. The condition of diabetic coma is preceded by a period of days, weeks, or months, in which there is a large excretion of β -oxybutyric acid (20 grammes or more in twenty-four hours), and in which the N of NH_3 is largely increased.

7. Patients whose urines show or have shown a large excretion of organic acids are in danger of developing diabetic coma, but the N of NH_3 may temporarily rise as high as 16 per cent., and yet coma may be delayed for more than seven months. The persistent excretion of more than 25 grammes of β -oxybutyric acid indicates impending coma.

8. A patient passing 30 grammes of β -oxybutyric acid in twenty-four hours may still have enough energy and strength to be about all day and perform considerable muscular work.

9. A patient who has been excreting very little organic acid and has gained weight may, within a few months, show the presence of considerable quantities of organic acid, and die in typical diabetic coma.

10. When the urine contains little or no organic acid there is no immediate prospect of diabetic coma, but patients with such urine are probably liable to most of the other dangers that threaten diabetic patients. The relation between the degree of acid intoxication and the susceptibility to infection seems worthy of special experimental study.

11. Where the urine regularly contains more than 200 grammes of sugar per day there is usually considerable organic acid in the urine, and large amounts of acid, indicative of coma, are invariably accompanied by considerable or great glycosuria.

12. Sometimes there is much sugar and little or no acid in the urine, and sometimes there is considerable acid and little sugar. These facts render it desirable to examine the urine of diabetic patients at least once a month, with reference to the amount of acid excreted, for the element of acid

intoxication must be clearly separated from the element of glycosuria in our study of the progress of a case. In other words, we must recognize the acid intoxication as an important and sometimes as a dominant factor in the prognosis, and this element should be regarded even in those cases of diabetes which have the clinical indications of a mild type of the disease. We may thus hope to prolong life in many instances by taking precautions as to diet and out-of-door life which might not otherwise be deemed necessary.

13. The withdrawal of carbohydrate food frequently leads to a considerable reduction in the quantity of organic acids excreted. The reason for this is not yet clear, and the phenomenon deserves careful study.

On Albuminuria after Cold Baths.—REM-PICCI (*Policlinico*, 1901, viii., M. 389) reports observations upon thirty-five individuals who had altogether 115 baths. More than 350 analyses of the urine were made. The author concludes that albuminuria may be considered a constant phenomenon after cold baths. Different subjects react differently to cold baths taken under the same conditions. Those which show albuminuria more readily are, as a rule, the less robust and thinner individuals, such as are more sensitive to cold. The limits of temperature necessary to produce the phenomenon are from 12° to 13° C., when the immersion lasts no more than three minutes. If the temperature be from 15° to 20° C. the albumin appears only after fifteen minutes' immersion. Above this temperature albuminuria does not occur, even if the bath lasts much longer. The colder the bath the more rapid the appearance of albumin. Distinct albuminuria may occur eleven minutes after the end of a bath at 10.5° C. lasting ten minutes.

This albuminuria is of short duration, lasting several hours only. The milder the phenomenon the more transient it is. The cold-bath habit does not affect the appearance of albumin in the urine. The degree of albuminuria (serum albumin) is very slight; in the more marked cases the quantity barely reaches 0.25 gramme per mille. The sediment occasionally shows a few hyaline casts and often crystals of oxalate of lime. The baths are often followed by an increased flow of urine, which is commoner after shorter baths and in patients who have suffered especially from the sensation of cold. While the flow of urine is usually increased in association with albuminuria, there appears to be no constant relation between the two; either may occur without the other. There is an increase in the total elimination of solids after a cold bath, whether or not albuminuria is present, the total nitrogen and urea as well as the chlorides being increased. There is never urobilinuria.

The arterial pressure increases after baths of short duration, while following baths of longer duration there is a diminution in pressure which may last for several hours. After very prolonged baths in which the action of cold was intense there was an increase and irregularity in cardiac action, more or less diffuse cyanosis, and sometimes evident dilatation of the right heart.

Most authors have attributed the albuminuria occurring after cold baths to a stasis in the renal circulation. This view, however, is not held by the author, who believes that the phenomenon is due to the cutaneous irritation.

That albuminuria may be due to the action of the nervous system upon the renal elements without concurrent vascular changes is suggested by Berkeley's demonstration of the direct relation between the terminations of the nerves of the kidney and the renal elements, as well as by the observations of Arthaud, Butten, Masius, Vanni, and Boeri, who have demonstrated the trophic action of the vagus upon the kidney.

Rem-Picci believes that the albuminuria is functional, but emphasizes the fact that while the phenomenon may have but little importance, occurring upon a single occasion after too cold or too prolonged a bath, yet if it be a daily occurrence it can scarcely fail to have, in the end, a deleterious effect upon the kidneys. Too cold baths are, therefore, inadvisable in general, and should be absolutely proscribed when there is any suspicion of latent renal lesions.

Some Rare Complications of Dysentery.—REMLINGER (*Revue de Médecine*, October 10, 1901, p. 873) states that dysentery is quite frequent among the soldiers stationed at Tunis. He reports a number of interesting and unusual complications of this disease observed among the invalided soldiers admitted to the Belvédère Hospital. Regarding the bacteriology of the cases of dysentery, the writer states that the only organisms they succeeded in isolating from the stools were the common varieties of the colon bacillus. In none of the cases was the amœba coli found.

The first case reported developed definite signs of an acute nephritis. There was marked œdema, and the urine contained considerable albumin. In addition, both of the knee-joints and ankle-joints became very painful and swollen, and there was also marked muscular pain in the lower extremities. The autopsy showed that there was an acute parenchymatous nephritis, and the writer claims that the muscular and articular pains were of dysenteric origin. The patient had no fever.

Two other cases are reported in which there was marked general anasarca without clinical evidences of any cardiac or renal involvement. There was no albuminuria. Injection of methylene blue beneath the skin indicated a deficient permeability of the renal epithelium to solids. This remarkable condition usually develops during convalescence. Notwithstanding that the cases failed to show the usual urinary changes of nephritis, the writer is inclined to the view that the anasarca is due to some inherent changes in the kidneys, owing to its yielding to the usual treatment of nephritis.

In another case the patient developed an arthritis of the right knee-joint, which became markedly distended with fluid. The whole right lower extremity subsequently became swollen, and a diagnosis of phlebitis was made clinically, although no reference is made to the condition of the veins at the autopsy. There were no marked gross changes in and about the knee-joint, and the exudate was evidently not purulent.

The occurrence of an epididymitis is noted in two cases during convalescence. In both cases gonorrhœa was excluded as a cause.

The last case recorded is an interesting one of dysentery complicated by an abscess of the spleen, which eventually ruptured, causing a general peritonitis and death. At the autopsy the spleen was found to have been almost completely disintegrated by the abscess before it ruptured.

SURGERY.

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Omentofixation.—MONGOUR (*Journal de Médecine de Bordeaux*, August 25, 1901) states that fixation of the omentum between the posterior abdominal wall and the parietal peritoneum is a most simple operation; vascular adhesions form, and in this way the blood of the portal vein is conveyed to the vena cava more freely than if it has to pass through the permanently obstructed blood-channels of the liver. This obstruction presents itself in venous cirrhosis of the liver, generally of an atrophic type, with endophlebitis and periphlebitis of the intrahepatic ramifications of the portal vein. When this ascites by its volume becomes an obstacle to the free movement of the lungs and heart, it is removed by abdominal puncture, but if the vascular lesion be not relieved the ascites re-forms, and punctures at frequent intervals become necessary. The operation of omentofixation was performed concurrently by Talma and Van der Meulen in 1889. Since that time the technique has been modified several times, but the original principles of the operation remain the same. As the result of the vascular adhesions the collateral circulation is formed by the anastomosis of the epigastric, abdominal subcutaneous, thoracic, and internal mammary veins with the original branches of the portal trunk. The experiments on animals with this operation have not been a success. The author has collected from the literature eighteen cases of this operation, with the following results: Death some hours or days after operation in 6 cases; not improved in 3 cases; improvement doubtful in 4 cases. Recovery from the ascites in 5 cases. This operation is a grave one, for these statistics show a mortality of 35 per cent., and it is also important to consider the five cases which have been reported as cured. In one of these cases the patient had simply an ascites of cardiac origin, while in another case it is very doubtful whether the cirrhosis was of the atrophic type. There can be no doubt that in those cases of hypertrophic cirrhosis the ascites is occasionally spontaneously absorbed. The hypertrophy is for the liver a process of defence, while in those cases of bi-venous cirrhosis it is a surgical *noli me tangere*. Clinical experience has shown that those patients with hypertrophic cirrhosis who have been operated upon have recovered, notwithstanding the operative interference, and not by reason of it. In some cases it is absolutely impossible to determine the remaining functional value of the diseased liver, and, therefore, impossible to separate the cases that are operable from those that are not. In cases of doubt an exploratory operation is indicated, and

then to proceed to omentofixation in all those cases of atrophic cirrhosis with recurring ascites, except where the patient is cachectic. It is certain that the future of those cases of cirrhosis in which all treatment is stopped is not a brilliant one, but with the operation of omentofixation it becomes even more dark, and as the least one may do for these patients is to respect the few days that remain for them to live, the author does not believe in this method of treatment.

Gangrene of the Extremity of the Left Leg during Convalescence from Pneumonia; Amputation; Recovery.—DOUILLET (*Archives de Méd. et de Pharm. Militaires*, September, 1901) reports the interesting case of a man, aged twenty-one years, who had a faint pneumonia of the right lung, and who, on the second day of the defervescence, complained of much pain in the left leg, which was soon followed by gangrene, and eventually the leg was amputated at the point of election for its relief. In order to determine the cause of the gangrene, a dissection was made of the amputated portion of the leg. This showed that the anterior tibial and peroneal arteries were not only permeable but apparently unaltered, while the posterior tibial artery was completely obliterated. The deeper veins presented no pathological changes. Without doubt the gangrene was the result of the obliteration of the posterior tibial artery, for it commenced in the portion of the leg whose blood-supply was derived from that artery. This obliteration was not due to an embolus, but was the result of an arterial thrombosis. Failure to obtain a bacteriological and microscopical examination made it impossible to determine the exact condition of the arterial tunics and the alteration of the blood by the pneumococcus or its toxins. These are factors which are competent to have played an important part in the formation of the thrombus, especially as the circulation in this patient was extremely poor not only from the onset of the pneumonia but throughout convalescence.

Notes on a Case of Acute Urethritis Due to the Staphylococcus.—MALHERBE (*Annales des Mal. des Organes Génito-Urinaires*, November, 1901) states in conclusion: 1. The urethral canal may occasionally be the seat of acute inflammation due to the staphylococcus albus. It may be obtained in pure culture on either serum or gelatin. 2. The contact of the meatus with a buccal mucous membrane affected with aphthous stomatitis may, perhaps, be a possible mode of infection. 3. This urethritis due to the staphylococcus is shown by a very slight sero-sanguineous discharge that differs in every way from the discharge of gonorrhœa. 4. There is pain on urination, and it may be more marked than in gonorrhœa. 5. The pain is severe along all the canal during urination, and there may be neuralgia and cutaneous hyperæsthesia in all the genital region. 6. It may have a duration of five weeks. Notwithstanding the violence of the local inflammation there is no effect on the general health. 7. It ceases spontaneously without treatment, and is not followed by complications. 8. It may or may not be easily contagious to women. 9. Acute staphylococcus urethritis is probably rarely seen in a marked form, but it is probable that those cases where a marked inflammation remains limited to the fossa navicularis and does not produce discharge are of this nature or due to some germ other than the gonococcus.

A Case of Traumatic Intraperitoneal Rupture of the Bladder; Laparotomy; Recovery.—DOHRN (*Deutsche Zeitschrift für Chirurgie*, July, 1901) states that up to the year 1878 out of 131 reported cases of intraperitoneal rupture of the bladder only one case recovered, while in 1896 out of 38 cases of traumatic intraperitoneal rupture of the bladder that were treated by operation 21 died. It is most important that operation be performed as soon after the accident as possible, although later an operation may be successful, as in the author's case. A man, aged forty-one years, was run over by a heavily laden wagon, which passed over the right hip and the lower portion of his abdomen. He was at once removed to his home, and complained of severe pain in the lower part of the abdominal cavity and an inability to void either urine or feces. The same evening there were nausea and vomiting. Operation was performed forty-six hours after the accident. A median incision above the pubes was made, and the extraperitoneal portion of the bladder was found uninjured. The peritoneum, which bulged, was then incised and the peritoneal cavity was found to be filled with a reddish-yellow fluid with a markedly urinous odor; flushed out; recovery.

Auto-intoxication from Intestinal Obstruction.—KUKULA (*Archiv für klin. Chirurgie*, vol. lxiii, p. 773) reports on his numerous and laborious experiments, which he instituted with the intention of determining the substance or substances which produce the large number of symptoms of prostration and collapse, in the course of intestinal obstruction whose origin cannot be explained by the mechanical impediments to the passage of the intestinal contents, and which he, in conjunction with many others, believes to be due to an auto-intoxication. In the first part of his paper he reviews the work which has been done and the literature which has been published on the subject. He divides the substances which have been isolated from the intestinal contents, and which have been claimed as causes of auto-intoxication, into two groups: Those arising during the normal process of digestion, which may, however, exist in abnormal quantities; they may be products of carbonic-acid fermentation, as acetic acid, lactic acid, butyric acid, etc., or of the putrefaction of the albumins, as NH_3 , N , CO_2 , H_2S , leucin, phenol, cresol, etc. To the second group belong only those chemical substances which are formed in stagnant or fermented fecal matter, for example, pyridin, chinolin, the toxalbumins, and ptomaines; of the latter, the diamins, as tetramethylene and pentamethylene diamins, and ethylene diamin, cholin, neurin, interest him especially. He deplores the fact that we know very little more than the names of these substances. The substances of the first group, when existing in normal amount only, preserve the peristaltic movements of the intestines; when in abnormal amounts peristalsis is increased, gastritis and enteritis are produced. Lactic and succinic acids are the worst of these offenders. The products of albuminous decomposition are much more toxic, however, and only rarely do they exist in the normal intestine; even small quantities of H_2S , NH_3 , and phenol are eminently toxic to lower animals. The second group is much more important, however, to the question of auto-intoxication, but after reviewing everything written about it carefully, he concludes that regarding some we are

not even certain that they exist, regarding others we know no more than that they exist.

In the second part of his paper he describes his experiments, of which there were three series: In the first group, believing the symptoms to be due to toxins, he used the contents of the intestines from five cases of occlusion, injecting the filtrate into rabbits, rats, dogs, mice, and guinea-pigs. He experimented with thirty-nine animals, but by far the greater number did not react at all. Six of them died within a few days, but only one of these had typical obstruction. He believes his ill-success to have been due to too great a dilution of the material. Of nine animals in whom he produced artificial obstruction of the intestines, but one died with characteristic symptoms. Of five animals in whom he produced strangulated hernia and volvulus respectively, but one of the latter died as the result. No toxicity of the intestinal contents of these animals was found when they were injected into cats. In the second group of his experiments he attempted the production of intestinal occlusion in twelve dogs; in seven he succeeded, three others recovered, and two died of purulent peritonitis. Of the seven cases four died with characteristic symptoms within a few days, and at the autopsy the typical picture of intestinal obstruction was found. The other three were killed at the time when presenting none of the symptoms, but showing much phenol and indican in their urine. In all the cases it was noticed that these two substances increased progressively in the urine until the day of death, or, in those dogs which recovered, until the day of restitution of the intestinal function. He does not believe, however, that these substances are of great value in solving the question of autointoxication, as in no case did they exist in sufficiently large quantities to be considered toxic.

The intestinal contents from the obstruction cases were injected into dogs and cats, and produced, whether injected pure or as filtrate, characteristic symptoms of intoxication. By means of alcohol nearly all of the toxic substances could be extracted from the fecal matter, and animals injected with this extract showed the symptoms even more markedly than the others, but intraperitoneal injections were even more potent than subcutaneous. Pentamethylene diamine, sulphuretted hydrogen, and methylmercaptan are believed by Kukula to play an important part in producing the symptoms, the first, however, more than the other two, especially as in his experiments with the gases he was not very successful. Control tests with normal feces were constantly made, but were found only slightly toxic, and then only for a short time. In the third series he used the intestinal contents of four cases of obstruction, and of one normal individual (the latter for control purposes), injecting them into thirty animals, with the same results as in series two. The symptoms thus produced are considered by him to be identical with the symptoms of intestinal obstruction in man, and as the same substances produce them in both instances, he considers them due to an absorption of the septic materials through the coats of the intestine, the epithelium of which is affected very early in the course of the obstruction. He believes these septic substances to be, in part, gases, in part products insoluble in water (pentamethylene diamine), but he is not able to definitely state the exact causes of auto-intoxication.

THERAPEUTICS.

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Therapeutic Value of Adrenalin Chloride.—DR. D. S. REYNOLDS, in a series of observations on diseases of the eye, concludes that: 1. It is a powerful hemostatic and acts promptly, generally within one minute from the time it is applied to mucous surfaces. 2. Its effects persist from twenty minutes to four hours. 3. It promptly relieves ciliary pain in all forms of keratitis, iritis, and even cyclitis of glaucoma. 4. It reduces ocular tension in glaucoma, and apparently prevents hemorrhage in iridectomy. 5. It promptly clears up interstitial opacities of the cornea following contusions, and seems to modify favorably the opacities of punctate keratitis in cases of syphilitic iritis. 6. It will, in many cases, so reduce the swelling in the tear passage as to allow a stream of fluid to pass from an Anel syringe through the duct without the aid of a probe. In a great variety of tinnitus aurium prompt and sometimes lasting benefit follows the introduction of a drop of adrenalin solution through the Eustachian catheter, blown into the tympanic cavity. 7. In all forms of swelling in the lining of the nose prompt relief follows the application of four or five minims of adrenalin solution sprayed into the passage. Operations in nasal sinuses are rendered bloodless. One to one thousand solution of adrenalin in sodium chloride may be relied on to check epistaxis.—*American Medicine*, 1901, vol. ii., p. 32.

Action of Caffeine on the Mammalian Heart.—DRS. ARTHUR R. CUSHNY and BERT K. VAN NATEN have made a careful study of this subject, being led to make the investigation by reason of the few direct observations which had been recorded of the action of this alkaloid on the heart of mammals, and because of the frequent use made of caffeine in the therapeutics of heart disease. They find that the effect of caffeine on the dog's heart appears to consist in (1) an acceleration of the rhythm without further change; (2) a shortening of the movements, commencing in the auricle and spreading to the ventricle, and, in large doses, (3) auriculo-ventricular arrhythmia terminating in the fibrillary contractions of the auricle and finally of the ventricle. The first phenomenon—the acceleration—appears to be due to stimulation of the most irritable part of the heart, the so-called excito-motor apparatus, and as no further change in the movements is seen, the action of the drug at this stage appears to be confined to this area. The second change, the lessened excursion of the levers, may be due in part to the acceleration, and may thus be considered a secondary effect of the

increased irritability of the excito-motor area; but it may also be ascribed to the action of the drug on the muscle of the auricle and ventricle, and may thus indicate that the influence of the drug has extended to these less susceptible parts of the heart. The third stage of arrhythmia is due to the ventricular irritability having been so greatly increased as to give rise to an idioventricular rhythm. The interference of the two rhythms then explains the major part of the variation in the strength of systole and the extent of diastole. The idioventricular rhythm indicates that the characteristic stimulant action on the cardiac muscle has extended to the ventricle. When this has attained a sufficient height it leads to fibrillary contractions in the ventricle; the previous appearance of these in the auricle appears to indicate that the stimulant influence spreads to this before it reaches the ventricle. The action of caffeine on the mammalian heart thus appears to consist in a descending stimulation which begins in the excito-motor area at the junction of the auricle and great veins, and extends into the auricles and finally to the ventricles. The effects can be explained by direct action on the muscle without the necessity of appealing to any nervous apparatus, and their experiments do not support the idea that the nervous apparatus of the heart is involved in the effects. If the action of caffeine on the dog's heart be compared with that of the digitalis series it is found that, as far as the direct action on the heart is concerned, they resemble each other in both, affecting only the heart muscle. But in the case of digitalis, the earliest changes seen are in the strength of systole and extent of diastole in the ventricle and auricle. These parts of the heart are thus affected sooner than the excito-motor area, and the contrast between the action of the two drugs may be explained by stating that while both act on the heart muscle the stimulation exercised by caffeine begins in the excito-motor area and descends to the auricle and then to the ventricle, and its effects on the rhythm (as far as these are caused by direct action on the heart) are of secondary importance. Further, the primary changes induced by the digitalis series are not so much evidenced by increased irritability of the parts affected as by increased contractility and lessened dilatation (increased tone), while there is no evidence of such a change in the late stages of caffeine poisoning, in which the ventricle is directly affected.—*Archives Internationales de Pharmacodynamie et de Thérapie*, 1901, vol. ix., p. 12.

The Mechanism of the Action of Igazol.—DR. V. CERVELLO has been investigating the therapeutic possibilities of a combination of formic aldehyde and chloral hydrate with terpine and iodoform, known as igazol. He has found that this product has a distinct value, used as an inhalant in the therapy of tuberculosis, and in the present study seeks to find some rational explanation for the good clinical results obtained. The early investigations on rats, rabbits, guinea-pigs, etc., demonstrated that, exposed to an atmosphere impregnated with this combination, the respiratory functions showed a marked alteration that consisted primarily in a much heightened excursion of the respiratory curve, i. e., the amount of respiratory exchange was greatly increased. This did not seem to be an irritative phenomenon, since with the increase in the amplitude of the thoracic vibration there resulted a slight diminution in the frequency of the breathing. The investigator further

claims to have shown that this substance excites the organic tissues to fix and to consume larger quantities of oxygen than under normal conditions. This oxidizing energy is largely resident in the blood, but does not alter the character of the hæmoglobin. This blood action is very characteristic and brings out the results of Tollens along this same line, that formic aldehyde is a powerful blood oxidizing and reducing agent.—*Archives Internationales de Pharmacodynamie et de Thérapie*, 1901, vol. ix., p. 217.

Treatment of Gastric Ulcer.—DR. A. ROBIN gives in very succinct form the following general directions for the treatment of this affection. The first indication is functional rest. Formerly the milk treatment was instituted immediately the existence of gastric ulcer was recognized; but the experience acquired by him in the treatment of this malady as well as the results obtained by Fournier and Gros during the last few years have demonstrated the necessity of putting the patient through a preparatory course of treatment, consisting of absolute rest, before adopting the milk diet. The following is the course of treatment: The patient is kept in bed; a towel steeped in hot water and covered with oiled silk and cotton-wool is placed over the epigastrium, and kept in position by means of a flannel bandage; no nourishment being given by the mouth, not even milk or water. The patient is given four enemata daily, each composed of two eggs beaten up with two spoonfuls of peptone, thirty grains of salt, a few drops of laudanum, and three ounces of glucose (20 per cent.). The whole enema should measure about six fluidounces. Two other enemata of simple warm water are given during the day to supply the organism with the necessary amount of liquid. To accustom the patients to this treatment it is well to increase the number of enemata gradually until complete tolerance is obtained. Where intolerance is present (which happens in about one in fifteen cases, according to his experience) one must endeavor to overcome it by increasing the laudanum to twelve or fifteen drops, or by suppressing the salt and the peptone, which seem to be the substances which usually provoke the intolerance. According to Fournier and Gros, the treatment should be continued for two weeks, and in some cases for a month, but Robin has never found it necessary to prolong the period of absolute rest beyond twelve days at the most, and frequently three or four days have proved sufficient. The indications for the termination of the rest cure are persistent rectal intolerance, progressive enfeeblement of the patient, and when, in spite of the treatment, the cardinal symptoms of the ulcer persist. It need only be mentioned that the alternative for the rectal method of introducing food, viz., the subcutaneous method, a suggestion which gave rise to a highly instructive debate at the recent Congress held in Paris, the conclusions arrived at were that the subcutaneous method was impracticable and frequently gave rise to untoward symptoms. Under the influence of the rest cure the distressing symptoms subside, and the loss of weight is checked. When the milk diet is begun the patient should take gradually increasing quantities until the daily amount reaches four quarts. This should be maintained for not less than six months, when a mixed diet may be ordered consisting of milk foods prepared with tapioca, arrowroot, rice, etc., with the addition of a few vegetables. After about two weeks the milk

is abandoned entirely, and the patient is allowed meats, fish, eggs, vegetables and cooked fruits. A certain proportion of patients, however, will require a supplementary treatment, these usually being such as show indications of neurasthenia and those who suffer from anemia. For the former the preparations of arsenic, more particularly sodium cacodylate subcutaneously, are advisable; and for the latter iron, preferably the perchloride, is the best medicine. In administering sodium cacodylate subcutaneously the best results have been obtained by the use of one grain daily for eight consecutive days, followed by an equal number of days of repose, and continued until the administration has covered forty days. This method of treatment has given excellent results in grave cases of cachexia. Various other methods of treating gastric ulcer have been tried, such as the introduction of bismuth subnitrate into the stomach, when after contact with the ulcer it is siphoned out; also the suggested washings of the stomach with a solution of silver nitrate, and with iron perchloride, none of which methods can be recommended, since the introduction of the tube in such cases is always more or less dangerous.—*Medical Press*, 1901, vol. cxxiii., p. 457.

[The introduction of the stomach-tube by experienced hands is nearly devoid of danger. *Magnesia ponderosa* by the mouth certainly facilitates healing and apparently does not irritate.—R. W. W.]

The Kidneys with Reference to the Employment of Diuretics.—DR. A. R. ELLIOTT says that with the exception of the irritant epithelial diuretics (turpentine, cantharides, etc.) the entire class may be said to exert their effect on the urine by acting indirectly through the circulation. Because of the necessity of avoiding all irritation of the kidneys, diuretics should act indirectly rather than directly; consequently, the secretory diuretics are contra-indicated in irritative and inflammatory renal conditions, and the simple diluents and salines are best adapted, since diuretics are mainly useful in overcoming concentration and hyperacidity of the urine. In an acute nephritis saline diuretics are permissible throughout the entire course of the disease, and exert a beneficial influence by increasing elimination and clearing the tubules of inflammatory *débris*. Subcutaneous saline infusion constitutes a most powerful aid to elimination in desperate cases. In chronic nephritis the cardiovascular diuretics are most useful, owing to the fact that oliguria and dropsy are usually the result of circulatory failure; and the dropsy, being of cardiac origin, may be benefited by cardiovascular stimulants, provided the kidneys are not too badly damaged. Dropsy of purely renal origin, however, is not amenable to favorable influence by diuretics. Although the morbid process in the kidneys may furnish us with our primary inspiration to diuretics, it is the condition of the heart and circulatory apparatus in most cases that determines the choice of an agent.—*Medical News*, 1901, vol. lxxix., p. 201.

Treatment of Rheumatism and Allied Disorders.—DR. D. K. COVERLY, writing of the treatment of rheumatism by the salicylates, says that the value of this medication has been materially lessened by the many drawbacks which its constant administration entails; the more prominent of these being headache, tinnitus, cardiac depression, and complete anorexia,

with subsequent gastric irritation. The urgency of these symptoms necessitates a suspension of the treatment, and the ground thus periodically lost accounts for the tardiness of the recovery. In consequence of this many attempts have been made, with more or less success, to replace the salicylates with compounds for which was claimed freedom from all objectionable features; but for some time these did not substantiate the claims made for them. Recently the introduction of salophen seems to have accomplished the purpose, since this drug possesses no irritating effects and is well borne by the stomach. It passes unchanged into the intestines, and is there decomposed into salicylic acid and paramidophenol; both of these contributing to its therapeutic action as an antirheumatic, antipyretic, and analgesic; but its efficiency as an antirheumatic is less pronounced because of the comparatively small amount of salicylic acid set free, though as an antipyretic and analgesic it remains a valuable remedy. Of still more recent appearance is aspirin, which chemically is acetyl salicylic acid, and occurs in crystals which are soluble in alcohol but not in water. In marked contrast to salicylic acid, this drug is agreeable in taste, slightly acidulous, and does not give rise to digestive disturbances. In the stomach it remains unchanged; but when brought into contact with the alkaline secretion of the intestines, salicylic acid is gradually liberated. After absorption not only does it act therapeutically as an antirheumatic, but as an analgesic and antipyretic as well, and its prompt elimination is readily accounted for by profuse sweats and increased action of the kidneys—*Therapeutic Gazette*, 1901, No. 11, p. 729.

Sodium Silicate, Sodium Fluorsilicate, and Sodium Fluoride in Therapeutics.—DR. ALFRED SIEGFRIED has given a thorough *résumé* of the occurrence, physiological action, and therapeutic indications of silicon sodium salts and their closely related fluorides. He shows that of late years almost nothing has been done with these bodies, and that many of the experimental results obtained by pharmacologists twenty-five years ago, and which are widely quoted at the present time, especially concerning toxicology, are not in accord with the facts.

The following pharmacotherapeutic indications are brought out: It is logical and of great value to use sodium silicate in acid poisoning. It is to be preferred to any alkali which develops carbon dioxide gas, and, moreover, has the advantage of coating the mucous membrane with a protective mucilage-like coat. In excessive acidity of the stomach it can be used in the proportion of one of sodium silicate to one thousand of carbonic acid water, as a table water. In acid diabetes it is useful as a table water. In gout it is especially valuable. In all of these diseases an alkaline salt is preferable, although a neutral salt is not contraindicated. It may be useful in hyperkeratosis or in lack of development of the horny layers of the skin. An extremely suggestive line is that of its use in phthisis. Inasmuch as fibroid phthisis offers the best prognosis of any of the forms of this disease, it is suggested that the use of sodium silicate may help in bringing about a certain increase in the silicious matter of the lung, and thus aid in the development and resistance of the fibrous tissue. This is in line with the widely believed idea that calcareous waters aid in bringing about a precisely

similar condition, i. e., chalky deposits and fibroid changes. Kobert in his work at the Görbersdorf Tuberculosis Sanitarium investigated the subject for over one year, and while not arriving at any convincingly positive conclusions, yet demonstrated that daily doses of fifteen to thirty grains of sodium silicate can be taken without any harm. His completed results await publication. So far as the fluorine compounds are concerned, recently antitussin for whooping-cough has been warmly recommended, and acute pneumonia has been treated with fluoroform. Further therapeutic possibilities may exist for the use of the fluorine compounds in aiding in the development of the teeth and in rachitis.—*Archives Internationales de Pharmacodynamie et de Thérapie*, 1901, vol. ix., p. 225.

Treatment of Syphilis by Injections of Succinimide of Mercury.—DR. M. HOROVITZ strongly recommends the use of this form of mercury for the treatment of syphilis. He employs it in a mixture with cocaine according to the following prescription: Mercury succinimide, 5; cocaine hydrochlorate, 2; distilled water, 100. The mixture has the following advantages: because of the addition of the cocaine no pain is felt, it works rapidly, does not produce any swelling, because of its high percentage of mercury its action is very energetic, and the dosage can be accurately measured.—*Centralblatt für die gesammte Therapie*, 1901, vol. xix., p. 641.

GYNECOLOGY.

UNDER THE CHARGE OF

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Appendicitis in Connection with Pelvic Disease.—FRAENKEL (*Centralblatt für Gynäkologie*, 1901, No. 40) believes that it is time to abandon the popular view that appendicitis is peculiar to the male sex. Infection may extend to the appendix from the diseased pelvic organs, or *vice versa*, in two ways, viz.: By an intraperitoneal route along the bloodvessels and lymph vessels of the suspensory ligament of the ovary (*ligamentum appendicula ovaricum*), or by direct contact of an abnormally long appendix with the uterus or right tube and ovary, or fixation of the adnexa at the pelvic brim; by an extraperitoneal route between the folds of the mesocæcum, or in consequence of the extraperitoneal development of the cæcum and appendix.

The most frequent pelvic complication of appendicitis is suppurative inflammation of the tubes and ovaries, especially on the right side. Rarely an abnormally long appendix may be found on the left side of the pelvis. The fact that torsion of the pedicle in cysts of the ovary and parovarium is often noted in connection with appendiceal trouble may be explained by the increased intra-abdominal pressure and peristalsis accompanying peri-

typhlitis. Since acute inflammation confined to the right tube and ovary is comparatively rare, when this condition is associated with appendicitis a direct causal relation must be inferred, especially in young subjects with right-sided pyosalpinx or "idiopathic parametritis, when gonorrhoeal or tubercular infection can be positively excluded and there is no history of a previous acute vaginitis.

The writer does not accept Edebohls' theory of the causal relation between movable kidney, chronic appendicitis, and right adnexal diseases, and believes that the palpation of slight enlargement of the appendix is an uncertain means of diagnosis, and that statistics with reference to the relative frequency of associated adnexal and appendiceal disease based upon this sign are open to grave error. Repeated attacks of localized pain, with occasional elevations of temperature just before or during menstruation, accompanied by gastro-intestinal disturbance, should direct attention to the appendix, and, after other means of relief have failed, furnish an indication for appendectomy.

In every case of appendicitis in the female a thorough examination of the pelvis should be made, preferably under anæsthesia, in order to avoid errors. The appendix should be examined in every case of abdominal section for pelvic disease, and should be removed not only when it is manifestly diseased, but even when it is adherent, since if it is spared the adhesions generally re-form, and morbid changes are apt to occur in the muscular wall of the appendix in consequence. On the other hand, during the course of an appendectomy the conditions of the uterus and adnexa should be noted.

When an appendiceal complication of adnexal disease is suspected the abdominal is preferable to the vaginal route. The right tube and ovary can be reached through the classical incision in the flank, but if both ovaries and tubes are affected the median is better.

Deep-seated abscesses may be drained per vaginam, or through the rectum in children and virgins; if situated too high up to be reached from below the pus may be evacuated through an abdominal incision, the diseased appendix and adnexa being removed subsequently.

Operation for Complicated Vesicovaginal Fistula.—WOLKOWITSCH (*Centralblatt für Gynäkologie*, 1901, No. 43) reports eight cases which he operated upon successfully by the following method: The cicatricial tissue around the opening is first dissected away; then the cervix uteri is freed and drawn downward, if possible without opening the peritoneal cavity, although this cannot always be avoided. All cicatricial bands are divided until the uterus can be drawn down almost to the vulva. The hemorrhage is not profuse, and it is important not to ligate the uterine arteries. A wide denudation of the vaginal mucosa is made around the edge of the fistula, the anterior surface of the cervix is completely denuded, and the raw surfaces are brought in contact by silk sutures. It is seldom necessary to suture the edges of the vesical mucosa separately. The vagina is then tamponed with iodoform gauze. If the peritoneal cavity has been previously opened a drain is introduced.

In order to drain the bladder thoroughly a suprapubic opening is made sufficiently large to admit the introduction of a small rubber tube, which is

sutured to the edges of the bladder wound. Drainage is favored by allowing the patient to lie on his belly, another larger tube being attached to it and leading to a receptacle, in order to prevent soiling the bed. The writer in recent cases has omitted the suprapubic drainage, with equally good results.

In the eight reported cases the functions of the bladder were completely restored, while there were no disturbances caused by fixation of the cervix to the anterior vaginal wall.

Vaginal Incision in Pelvic Abscess.—BÜRGER (*Centralblatt für Gynäkologie*, 1901, No. 43) reports 273 cases of pelvic abscess, including only those of suppuration of the tube and ovary. He emphasizes the fact that in pyosalpinx the conditions are essentially different from those which are present in ordinary abscesses, since even after evacuating the pus the diseased mucosa remain as a source of fresh infection and renewed suppuration. Hence the indications for incising pus-tubes per vaginam are comparatively limited. Of these fever is the most important, as it shows the presence of an acute process, with fresh pus, the escape of which into the peritoneal cavity during laparotomy is often fatal to the patient. Excessive tension of the sac and threatening perforation into adjacent organs are other indications for incision, which, however, is to be regarded simply as a palliative measure, to be followed later by abdominal section if necessary. When it is impossible to remove the pus by incision alone the diseased tube may be extirpated per vaginam.

In analyzing his cases the writer finds that in a considerable proportion it was necessary to repeat the operation in consequence of fresh suppuration in the opposite tube or imperfect drainage of the abscess cavity.

The results were seldom permanent, as the local pains usually persisted or were aggravated. A radical operation was necessary in 25 per cent. of the cases. In short, vaginal incision is to be regarded merely as a safe and easy way of temporarily relieving the patient from the symptoms incident to retained pus and of postponing the radical operation until it can be performed with less danger of infecting the healthy peritoneum.

Cancer of the Fallopian Tubes.—KNAUER (*Centralblatt für Gynäkologie*, 1901, No. 43) reports a case of adenocarcinoma involving both tubes and ovaries. He regarded the disease as developing primarily in the tubes, for the following reasons: The ovarian tumors presented the same structures as the tubal. Only portions of the ovarian stroma were affected, the cancer cells having invaded the normal tissue in different places, while in no instance was the germ or follicular epithelium involved.

The writer calls attention to the fact that while only four cases of cancer of both tubes have been reported, the simultaneous occurrence of the disease in both tubes and ovaries has not previously been noted.

Treatment of Adherent Retroflexion.—DIETEL (*Centralblatt für Gynäkologie*, 1901, No. 28) reports 112 cases, in 88 of which the displacement was of long standing. He employed massage and replacement under narcosis as well as surgical treatment. In 32 cases treated by massage 9 were successful. Of 23 in which Schultze's method was employed 20 patients were relieved so that they could wear pessaries, but in these the adnexa were not

adherent; 21 cases with adnexal complications were similarly treated, but only 12 were successful; 25 patients only were operated upon, the abdominal route being adopted by preference, with conservative treatment of the tubes and ovaries, 22 patients being cured. Hysterectomy was not performed in any instance.

Posterior Vaginal Enterocoele.—GONILLOND (*Revue de Chirurgie*, 1901, No. 5) performed posterior colporrhaphy and ventrofixation for the cure of complete procidentia in a woman, aged forty years. Two months later a vaginal enterocoele developed, the uterus remaining in normal position. The posterior vaginal fornix was opened, and the peritoneal sac was extirpated as in an ordinary hernia, after which the cervix was amputated, with a successful result.

These cases are quite rare. Condamin reports one in which an enterocoele developed as the result of coughing, the pelvic floor being intact, and the uterus, rectum, and bladder being in normal position.

Chloroform Narcosis.—BONNEAU (*Revue de Chirurgie*, 1901, No. 5) reports 1900 cases without a death. He uses a folded handkerchief, the chloroform being administered drop by drop, plenty of air being given at first. The tongue is drawn forward. Narcosis was usually complete in from eight to ten minutes.

Renal and cardiac diseases were not regarded as contraindications. Trendelenburg's position, the writer believes, is of advantage in chloroform anaesthesia. In twelve cases syncope occurred, and it was necessary to resort to artificial respiration.

Perforation of the Uterus.—REYBREYEND (*Revue de Gyn. et de Chir. Abdom.*, 1901, No. 2) includes under this heading accidental puncture of the gravid and non-gravid uterus, either with or without previous laparotomy. Cases of perforation with the sound or curette are not considered. In the former class of cases it is necessary to give a cautious prognosis, since abortion may occur after a considerable lapse of time.

Perforation of the uterus during vaginal operations occurs most frequently while enucleating sessile submucous fibroids.

With modern aseptic methods, less anxiety need be entertained about the result of this accident than formerly. Simple puncture requires no treatment. A considerable wound should be sutured at once after opening the abdomen. Hysterectomy is seldom necessary.

Chorio-epithelioma of the Uterus.—HITSCHMANN (*Centralblatt für Gynäkologie*, 1901, No. 28) reports the case of a multipara, aged thirty-eight years, who missed one period and began to have hemorrhages soon after, which continued for three months without symptoms of pregnancy. On examination the uterus was found to be enlarged, and that a tumor presented at the os internum. Examination of fragments showed that it was a chorio-epithelioma, whereupon the uterus was extirpated, death resulting three weeks later from metastasis in the thoracic and abdominal viscus, as well as in the brain. Microscopically the syncytium was slightly defined, there being isolated giant cells or delicate striæ.

OBSTETRICS.

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Two Cases of Idiopathic Swelling of the Parotid Gland in Newborn Infants.—KIEN (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1901, Band xlv., Heft 2) reports the case of a child born after spontaneous labor, and fully developed. The cranium showed no depression of its bones; the sutures and fontanelles were normal; the face showed an extraordinary appearance, caused by the swelling of the parotid glands. Examination showed that the submaxillaries were not involved; there was no abnormality in the mouth, nor was any infection in the mouth present. Steno's duct was patulous, and saliva escaped freely. The hands were somewhat swollen and cyanotic. The child's mother was normally shaped and healthy, and had already had three children, each of whom was healthy. The mother believed that the condition of her child was the result of a fright which occurred in the early months of her pregnancy. She had seen upon the street a child having a greatly deformed face. Four weeks after birth the swelling had very much diminished.

Madelung and Freund narrated to the writer a case coming under their observation of a similar nature. A review of the literature of the subject shows that some of these cases have been found to be sarcomatous, and in some Steno's duct was occluded.

The Treatment of Repeated Abortion and Premature Labor, with Fœtal Death.—LOMER (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1901, Band xlv., Heft 2) reports twenty-one cases of abortion and premature labor, with death of the embryo or fœtus. These were treated by prolonged rest in bed and by the administration of iodide of potassium and iron throughout the entire pregnancy. His cases may be divided into three classes: one, syphilitic, in which the syphilis was old or hereditary; the second class, in which the kidneys were very deficient in action and the patient was threatened with nephritis; and the third class, in which the patient was constantly absorbing necrotic material from a chronic endometritis. He believes that the treatment acts by preventing the rupture of vessels in the placenta. He also lays much stress upon the chronic anæmia present in these cases, for which he uses iron.

Cæsarean Section and Forceps Delivery in the Same Patient.—In the *Dublin Journal of Medical Science*, October, 1901, KIDD reports the following case: The patient was pregnant when first seen, and had a hard growth spring-

ing from the body of the cervix. This was apparently a fibroid, and extended toward the promontory of the sacrum. The head of the child was pushed aside to the ramus of the pubes on the left side. The tumor could not be dislocated, and accordingly Cæsarean section was performed and the child delivered in a semi-asphyxiated condition. On the evening of the third day the child had slight convulsions, and vomited blood and died. Upon autopsy, hemorrhage from the œsophagus and upper portion of the stomach was present. The mother made a good recovery from the operation. The patient was examined on convalescence, and it was found that the tumor had descended with the uterus and filled the hollow of the sacrum. The patient left the hospital with instructions to return should pain or disability occur or should she become pregnant.

She did not report for several months, until she applied for assistance in labor. On examination the membranes had ruptured, and the tumor acted as a wedge and prevented the descent of the head. The posterior wall of the uterus was drawn strongly upward, but it seemed possible that the head could come down between the tumor and the sacrum. Accordingly the patient was anesthetized and was delivered with forceps. The placenta was adherent very firmly and removed with difficulty. The patient made a good recovery.

It is interesting to note that the uterus, which had been very carefully closed at the Cæsarean operation, had successfully endured the strain of seventy-three hours' labor. If an explanation is sought for the successful forceps delivery it must be found in the fact that the tumor had undergone involution with the uterus, and had not essentially enlarged during the second pregnancy.

Prophylactic Perineotomy during Labor.—MANDELBERG (*Centralblatt für Gynäkologie*, 1901, No. 46) describes sixty-six perineotomies performed during labor in one of the maternity hospitals of St. Petersburg. Of the sixty-six patients sixty-two were primiparæ. The incision was made with a knife, in the central line, through the frænulum, extending from $2\frac{1}{2}$ to 3 cm. The lower extremity of the incision terminated in front of the sphincter of the bowel. The cut was made during a pain. After the uterus was emptied the incision was closed under anesthesia. It is claimed for this method that it prevents more extensive and serious tear, that union is better, that it diminishes the resistance to the head at the latter part of labor, that it prevents stretching of the uterine ligaments, and that it is in every way preferable to resistance to the head and to tear of the pelvic floor extending in several directions.

[There is much to be said in favor of this procedure. Some of the worst tears of the perineum and pelvic floor occur because the attendant makes injurious endeavors to protect the parts. During labor no pressure should be made upon the perineum proper, and the edge of the hand which supports the parts should cover the anus, but should not extend higher upon the perineum. The perineum should be left free to dilate as the head emerges.

The progress of the head should be regulated by the other hand of the attendant. Nature usually performs the operation described by Mandelberg. If no undue resistance is made to the exit of the fetal head the tear is

usually in the median line, is readily closed by suture, and heals without difficulty.

In our experience it is seldom necessary to cut the perineum, but it is better to allow a moderate tear in the perineum than, by our efforts to prevent it, bring about a manifold tear in the pelvic floor and vagina.]

A Case of Duchenne's Paralysis.—STOLPER (*Monatsschrift für Geburtshülfe und Gynäkologie*, Band xiv., Heft 1) reports the case of a multipara in labor, with the child in vertex presentation, second position, and with the head transverse in the cavity of the pelvis. The child was delivered by forceps, and it was necessary to extract the shoulders by pulling upon the occiput and the chin. As the shoulders were very broad, this was difficult, and required strong traction, with marked internal rotation. The fingers could be spontaneously moved. Except a small pressure-mark on the forehead there was no sign of injury. The arm was examined by the Röntgen rays, and no separation of the bones was found. The muscles involved in paralysis were the deltoid, biceps, brachialis internus, infraspinatus, and supinator longus.

Paralysis of the brachial plexus with the use of forceps occurs most frequently where flexion is wanting, and especially in face presentation. If the arm is strongly thrown upward and backward, traction upon the clavicle may produce the same result. In this case the roots of the fifth and sixth cervical nerves are pressed against the vertebræ. A similar pressure is made when the child is extracted by traction upon the head, with strong pressure upon the shoulders. In no case have the roots of the nerves been ruptured.

The prognosis is sometimes uncertain. It is best after cases following the use of forceps, and is not absolutely hopeless where traction has been made upon the clavicle. The treatment consists in the use of electricity and massage.

Deciduoma Malignum.—WINKLER (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1901, Band xlvii., Heft 2) reports the case of a woman, aged thirty-three years, who had had three normal pregnancies and labors. The last labor was four years before coming under observation. For the last four months she had had irregular hemorrhages and pain in the lower abdomen. She described a bladder-like body as having been expelled. She gave no typical history of abortion. On examination a tumor extending to the left side was felt above the symphysis pubis about the size of a child's head. It evidently was the enlarged uterus. On the left side, underneath the navel and on the left side of the vulva, there was a portion of tissue which gave indistinct fluctuation. The cervix was enlarged and soft, and the external os permitted insertion of the finger. The urine showed the presence of pus, epithelia, and a small quantity of albumin. A diagnosis was made of deciduoma with metastases, and hence inoperable. The patient shortly afterward died.

Upon autopsy the uterus was found gangrenous at the fundus, and a portion of the growth had involved the sigmoid flexure of the colon. Metastases were also present in the lungs and in the kidneys.

OPHTHALMOLOGY.

 UNDER THE CHARGE OF

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Periscopic Lenses.—PERCIVAL, Newcastle-on-Tyne (*Archives of Ophthalmology*, 1901, No. 5), gives formulæ for the radii of curvature of the surfaces of periscopic lenses. Periscopic lenses enable the wearer to see more clearly when moving his eyes from side to side—a manifest advantage. The side next to the eye must be always concave. The following table gives the radii of curvature for some periscopic lenses when the refractive index of the glass is 1.54; r_2 is the radius of curvature in mm. of the surface next to the eye (always concave); r_1 is the radius of curvature of the surface toward the light, away from the eye. The minus sign prefixed signifies that the surface before whose radius it is placed is concave toward the eye or convex to the light; the plus sign indicates the opposite, i. e., that the surface is convex toward the eye. It will be seen from the tables that the side toward the light is concave toward the eye in all except four cases, those being concave or divergent lenses.

CONCAVE PERISCOPIC GLASSES.

| Power. | r_1 | r_2 |
|--------------|-------------------|-------------------------|
| — 20 D . . . | + 172.8 mm. | — 32 mm. |
| — 17 D . . . | + 1512 “ | — $32^{104}/_{233}$ mm. |
| — 15 D . . . | — 612 “ | — 34 mm. |
| — 13 D . . . | + 1512 “ | — $42^{42}/_{59}$ mm. |
| — 10 D . . . | + 1512 “ | — 56 mm. |
| — 8 D . . . | — 843.75 “ | — 62.5 mm. |
| — 5 D . . . | — $174^6/_{31}$ “ | — $66^3/_{31}$ “ |
| — 2 D . . . | — 102 “ | — $74^1/_{31}$ “ |

CONVEX PERISCOPIC LENSES.

| Power. | r_1 | r_2 |
|---------------|-----------------------|-----------------------|
| + 20 D . . . | — $17^{41}/_{77}$ mm. | — 50 mm. |
| + 15 D . . . | — 20 “ | — 45 “ |
| + 12 D . . . | — 22.5 “ | — 45 “ |
| + 10 D . . . | — $24^6/_{11}$ “ | — 45 “ |
| + 8 D . . . | — 30 “ | — 54 “ |
| + 6.5 D . . . | — 37.5 “ | — $68^{28}/_{79}$ mm. |
| + 5 D . . . | — 54 “ | — 108 mm. |
| + 2 D . . . | — $64^4/_{59}$ “ | — 84 “ |

Considerations of the periscopic effect will determine how to correct a given case of mixed astigmatism. For example, suppose the vertical meridian

to be -2.5 and the horizontal $+2.5$. This can be corrected either with $+2.50$ sph. $\ominus -5$ cy. ax. 180 or -2.50 sph. $\ominus +5$ cy. ax. 90. Clearly the latter will allow greater lateral range, the concave surface being next to the eye. If the vertical field should be the more important, which would be very unusual, the first formula is to be used. In compound astigmatism a concave spherical surface must be on one side, in which case the requisite toric or compound astigmatic surface is to be ground on the other. This adds very materially to the expense.

[The same author remarks in a paper published elsewhere that the glasses commonly sold as periscopic are hardly worthy of the name.]

Monolateral Innervation of the Occipitofrontalis in a Case of Complete Bilateral Paralysis of the Oculomotor Nerves.—SALOMONSOHN (*Berlin. klin. Wochenschrift*, 1901, No. 26) reports the case of a man, aged forty-nine years, without distinct specific history, but given to the abuse of alcohol, who began to have double images April of last year, with pain starting in the left eye. No definite diagnosis was then possible, although both pupils, moderately dilated (4 mm.), were insensitive to light. Decided though slight reaction to convergence, especially on the left side, was present. Accommodation on both sides paretic. Visual acuity, fields and ophthalmoscopic appearances continued normal throughout. Two weeks later there developed a typical total paralysis of the left oculomotor nerve (complete ptosis, external strabismus, eyeball movable only by the external rectus, and rotation by the trochlearis). The patient was elsewhere subjected to five weeks' inunction treatment with gray ointment without the slightest result. In the beginning of August the right eye was found to be affected with paralysis of the internal, inferior, and superior recti, paresis of the inferior oblique, beginning ptosis, complete bilateral paralysis of accommodation. The paralysis of the oculomotor soon became total on this side also. Pupils absolutely fixed (convergence no longer possible). On both sides slight exophthalmus paralyticus. Condition thus far unimproved by potassium iodide. The patient complained of a permanent dull feeling in the face without objective signs of trigeminus involvement. Sense of taste for all substances abolished. Everything tasted disgustingly sweet, even when the various substances were diffused in the mouth and not permitted to touch the tongue only—solutions of salt, sugar, acetic acid, quinine. Marked disturbance of the olfactory nerve. Ol. terebinth., ol. menth. pip., ol. caryoph., tr. asafoet. not distinguished. Sal. ammoniac at once recognized in consequence of the irritation of the trigeminus. Gait, as to be expected, uncertain, but not ataxic. No swaying upon closure of the eyes. Patellar reflex present. No tremor of the hands. Tongue protruded straight. No disturbances of speech or deglutition. Facial nerves normal on both sides. Internal organs healthy, urine normal. Latterly the patient complains of headache, presents psychical changes, is often absent-minded, and forgets what he wants to do. Remembers the past well, but not recent events, and makes strange mistakes.

The writer remarks that great care is to be exercised in making an anatomical diagnosis in such cases, seeing that autopsies have shown various anatomical possibilities with the same clinical phenomena. For not only

have progressive palsies of the cerebral nerves been observed without any anatomical lesion (Eisenlohr, Oppenheimer), but they have also been occasioned by peripheral neuritic processes, basal neoplasms of various kinds, disturbances of the nuclei, degeneration of the nerve tracts by sclerotic foci in the course of the intermedullary nerve roots, and, lastly, in a combination of such processes. By far the most frequent cause of double palsy of the oculomotor nerves, especially in syphilis, is involvement of the nerves at the base; the involvement of the olfactory points the same way in the case reported. On the other hand, lues is quite uncertain in this case. The former abuse of alcohol may be a concurrent cause. The total palsy of the taste introduces a disturbing element. If we agree with Mauthner, the fact of binocular pupillary fixation and paresis of the accommodation already noted in April, is decisive for the diagnosis of nuclear palsy, for at that time a pure almost perfect ophthalmoplegia interna was present on the right side. The absence also of marked general cerebral phenomena and disturbance of the intellect likewise speaks loudly for the involvement of the nuclei (Mauthner). According to Bernheimer, however, the fibres of the oculomotor remain separate, extra nuclear to their exit from the brain, so that ophthalmoplegia interna does not with absolute certainty establish the diagnosis of nuclear palsy. Considering Wesphal-Siemerling's observations this case seems most likely to be a combination of nuclei and nerve degeneration.

A certain prognosis is hardly to be made. The present condition reminds one of Heinrich Heine's malady; but whether, as in the case of the poet, progressive muscular atrophy is to follow or whether the present palsies are the antecedents of a psychosis, as is often observed, and which are to be feared from the psychical changes which have lately arisen, or whether the process, whatever may be its nature, will extend to neighboring nuclear or nerve regions, or whether it can be arrested by potassium iodide—all this is beyond our ken.

Attention is called to a striking symptom, viz., monolateral innervation of the oculomotor nerve. The patient has complete bilateral ptosis. In spite of this he is able in varying degree to lift the right upper lid sufficiently to see with the right eye, but not through innervation of the levator palpebræ superioris, but by drawing up the right eyebrow and therewith the lid. If the eyebrow be fixed by pressure of the finger at the edge of the orbit, the right lid hangs as limp and smooth as the left. Mauthner first called attention to this symptom in cases of ptosis of longer duration after he had himself acquired complete ptosis on the right side through a trauma. He was able to innervate the right half of the occipitofrontalis alone, and thus to raise the lid. After recovery the power of such innervation was again lost. It is noteworthy in the case here reported that he raises only the right eyebrow, although he is affected with double ptosis. This, however, is very advantageous for him, since double images would at once appear in consequence of the strong divergence of the eye if both lids were raised. It is an interesting circumstance that the patient is unable to maintain monolateral innervation of the facial if the left lid is passively lifted. It shows plainly that the double images thus arising inhibit the subconscious purpose of raising the lid. Mauthner had already observed in himself that the monocular innervation was not under the control of his will, but followed as soon as he

opened the left eye (the ptosis was on the right side), although its continuance was unpleasant.

For the comprehension of the phenomenon two facts recently discussed may be brought to mind: Bell's phenomenon (lifting of the eyeball during energetic contraction of the orbicularis) and the orbicularis reaction of the pupil; whereas in both of these cases an overflow of a strong nerve impulse manifestly takes place from the branches of the facial to those of the oculomotor, the opposite occurs in Mauthner's phenomenon. The (palsied) levator palpebrarum is to be innervated. As this does not react, the innervation is strengthened as much as possible until it overflows upon the facial of the forehead. The monolateral innervation is not affected on the left side, probably because the right eye was the one which became diseased last. The anatomical basis of the phenomenon may be most easily understood upon the assumption (still *sub judice*) which Mendel has made from his observation upon animals, that the ocular facialis (frontal and orbicular branch) has its nucleus in the posterior portion of the region of the oculomotor nucleus.

[NOTE. Following Remak's suggestion, electrical excitation of the levator palpebræ superioris was tried, but without result, because the irritation occasioned reflex closure of the lids, which concealed any contraction of the levator which may have existed. The term "monolateral" innervation of the frontalis is moreover not quite correct for the case reported. The muscle is innervated bilaterally, but more strongly on the right side, especially so in its temporal portions, so that the eyebrow with the upper lid is only lifted on the right side.—ED.]

DERMATOLOGY.

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Remarkably Sudden and Complete Loss of Beard and Hair of Scalp, followed by Improved Vision.—J. R. CLARKE, of Marion, Kentucky (*American Practitioner and News*, July 15, 1901), reports the case of a man, aged eighty-six years, of fine constitution, and with a history of almost uninterrupted good health until ten years ago, when he was attacked with hæmoptysis, which continued frequently until one year ago; he was often expected to die from exhaustion following the great loss of blood, but at no time were there any pyrexial symptoms nor complications. He arose one morning feeling as well as usual, and after washing his face and combing

his hair, ate breakfast, went into the sitting-room, began reading, and became so interested that when his wife asked him to get ready for dinner he was much surprised, as he did not think it possible for the time to pass so rapidly. He went to the mirror to comb his hair and beard, the latter being thick and long, covering the upper portion of his chest. Finding the beard somewhat tangled, he parted it with the fingers. He dined as usual, not thinking of the beard, until his wife inquired what ailed his beard; he replied, "Nothing," but on grasping it in his hand, to his astonishment, it parted without sensation from his face, and he held it in his hand. Going to the mirror he found that the hair was everywhere else loose and falling, including the scalp, and by simply rubbing with the hand it all suddenly and easily came out. In a few days all the hair had disappeared from the entire surface of the body. A short time after losing the hair, the eyesight began to improve very remarkably, there was no more hæmoptysis, and the general health also improved in a marked degree.

[Lest some reader possibly may be disposed to discredit the accuracy of this history, it may be stated that similar cases, *as regards the sudden falling of the hair*, have from time to time been recorded. The improved state of the eyesight following the loss of hair is a most interesting observation, and serves as a valuable lesson, demonstrating the important relation of one part of the general economy to another in many obscure pathological conditions.—EDS.]

Pigmentation of the Skin Caused by the Demodex Folliculorum.—

DUBREUILH (*Journal de Médecine de Bordeaux*, 1901, No. 4) calls attention to the fact that the demodex, while usually a harmless occupant of the follicles, when it exists in great number, may give rise to abnormal pigmentation, as shown in the following case under his observation: A woman, aged forty years, brunette, had noticed for several years a fawn-colored discoloration around the mouth extending along the border of the jaws from one angle to the other. This discoloration was not accompanied by itching or inflammation. Upon close inspection the skin was found to be slightly wrinkled and roughened. Similar but less marked pigmentation existed upon the lateral and anterior parts of the neck and upon the chest. Upon examining scrapings from the affected parts with the microscope the demodex was found in extraordinary abundance. The author refers to similar cases observed by Amicis and Majocchi.

The Etiology of Lupus Erythematosus.—POOR (*Dermatologische Zeitschrift*, April, 1901) considers at length the principal arguments urged in favor of the tuberculous origin of lupus erythematosus. He does not find, as has been asserted by other authors, that this affection occurs most frequently in those the subjects of tuberculosis or with a distinct tuberculous family history. The local and general reaction which follows tuberculin injections in lupus erythematosus only occurs irregularly, and cannot be regarded as certain evidence of its tuberculous nature. The presence of giant cells cannot be regarded as proof of the tuberculous character of the disease, since these are found in other diseases which are in no way related to tuberculosis. The occurrence of forms transitional between lupus vulgaris and lupus ery-

thematosus may be explained by supposing that the existence of the latter malady lowers the natural powers of resistance of the tissues and prepares the way for the invasion of the tubercle bacillus so that there is in these cases a combination of two diseases. From the foregoing the author concludes that lupus erythematosus can be attributed neither to the direct invasion of the tubercle bacillus, nor to the remote effects of the toxins produced by this bacillus. It probably does not have a uniform etiology, but is produced by different kinds and degrees of peripheral and central irritations acting upon the variously reacting skin of the individual.

Is Alopecia Areata (La Pelade) Contagious?—M. CRUYL (*La Clinique*, April 27, 1901) reports that among thirty cases observed within a brief period by him there were no instances in which contagion could be traced. He does not think that the existence of this disease in the form of an epidemic has been proved, notwithstanding that instances in which it occurs in barracks are well known. This fact may be explained in other ways.

[It would seem that this disease is common in Belgium, thirty cases having been observed among the working class at the Civil Hospital at Ghent in the course of a few months.—EDS.]

Purpura following a General Gonorrhoeal Infection.—WEISZ (*Archiv für Dermatologie und Syphilis*, Bd. lvii., Heft 1 u. 2, 1901) reports the case of a man, aged twenty-four years, who, in a second attack of gonorrhoea with general symptoms, such as fever and marked psychical disturbance, with suicidal tendencies, had on both lower extremities numerous hemorrhagic macules. This purpuric eruption the author believes was due to the formation of emboli by the gonococci or to the toxins produced by this micro-organism. With the spontaneous cure of the gonorrhoea the purpura disappeared.

Lupus Carcinoma.—ASHIHARA (*Archiv für Dermatologie und Syphilis*, Bd. lvii., Heft 1 u. 2, 1901) reports three cases of lupus of the face associated with carcinoma. From a study of his own cases and 122 others reported by various observers, he is of the opinion that the variety of the lupus does not influence the occurrence of carcinoma, since in no instance did the preceding lupus present any special features. The course of lupus carcinoma differs from that of ordinary carcinoma of the skin in several particulars; it occurs at a comparatively early age; it advances very rapidly, the tuberculous infiltrate seeming to afford a very favorable soil for atypical epithelial proliferation, and recurrences are common after removal. Metastases are comparatively rare. The prognosis of this variety of carcinoma is very unfavorable. Although the various caustics may be employed with temporary benefit, operative interference is the most rational method of treating the malady.

Nodular Tuberculosis of the Prepuce.—SABRAZÈS and MURATEL (*La Semaine Médicale*, September 18, 1901) reports a hitherto undescribed form of tuberculosis of the penis. A man, aged twenty-six years, with tuberculosis of the lungs, kidneys, prostate, epididymis, and urethra, had upon the

anterior surface of the penis in its lower third a node the size and shape of an olive. This tumor was of fibrous consistence, painless, and attached only to the prepuce. A second node developed upon the scrotum, which suppurated and left a cicatrix with irregular borders. Histological examination showed a tuberculous structure and tubercle bacilli. The authors believe that the tuberculosis was propagated by way of the lymphatics.

The Contagiousness of Erythema Nodosum.—MOUSSOUS (*Archives de Médecine des Enfants*, July, 1901) has observed on two different occasions in his service erythema nodosum develop some days after a child has been admitted with this affection in the patient in the neighboring bed. He believes these cases can only be explained by contagion, and concludes that erythema nodosum is not a form of polymorphous erythema, but a specific affection.

The Etiology and Pathogeny of Sclerema Neonatorum.—COMBA (*La Clinica medica Italiana*, May, 1901; *Annales de Dermatologie et de Syphiligraphie*, October, 1901) reports two cases of sclerema neonatorum. In the first case the infant was born at term, fairly well developed; the autopsy showed a generalized infection with the bacillus of Friedländer, which had taken place through the umbilicus, the pus in the umbilical wound containing this micro-organism, and the right umbilical artery being inflamed. There was also a bronchopneumonia and an acute nephritis due to the same infection. In the second case the bacillus of Friedländer was also found, associated with the streptococcus. Infection probably occurred through the respiratory mucous membrane. Comba states that in all cases of sclerema there is a septicæmia the development of which is favored by the feebleness of the infant or by a local infection of some important organ. The micro-organisms or their toxins produce such a disturbance of the centres regulating thermogenesis that the temperature falls, and induration of the subcutaneous adipose tissue follows. The author does not think that sclerema neonatorum is a special nosological type; it should be considered as the result of an acute nephritis and of circulatory troubles secondary to an infectious process.

Goosegrease as an Excipient in Ringworm of Scalp.—G. T. JACKSON (*Journal of Cutaneous and Genito-Urinary Diseases*, June, 1901) refers to the value of this substance as an excipient in the treatment of ringworm of the tinea tonsurans. There is much goosegrease in the market that is not genuine, the best quality being very costly, for the reason that it is the fat taken from the dead but uncooked goose, a few ounces of fat costing as much as the whole goose. That employed by the author is made by the Schiefflin Company. For tinea tonsurans an ointment is made of 3ss to ʒj of the crystals of iodine to ʒj of goosegrease, to be worked into diseased areas by means of a stencil or stiff paint brush. It is also useful in tinea sycosis.

Bine Pigmentation following Injections of Morphine.—THIBIERGE (*Annales de Dermatologie et de Syphiligraphie*, Nos. 8 and 9, 1901), at a seance of the Société Médicale des Hôpitaux de Paris, presented a woman, aged

forty years, addicted to the use of morphine from the age of sixteen, in whom pale, bluish spots, similar to those produced by tattooing with Indian-ink, symmetrically distributed over the thigh, had followed injections of morphine. These spots were rounded or elongated, 2 to 3 millimetres in diameter, with a slight cicatricial depression. Histological examination of the spots showed the presence of black grains of unequal size, insoluble in alcohol, potash, and concentrated acids, and not giving the reaction for iron, and others transparent and refractive, with sharp corners. The first seemed to be particles of carbon, the others, particles of silica.

Eczema Produced by Formalin.—FISHER (*British Journal of Dermatology*, August, 1901), in a clinical note records his experience of the irritating effects of formalin upon the skin. He was accustomed to use this antiseptic in the preparation of museum specimens, more especially in the winter; and during several successive winters he suffered from an eczema of the fingers, which was at first attributed to the frequent wetting of the hands, but was subsequently found to be due to the use of formalin. Others who worked with him were similarly affected. Susceptibility to the irritant effects of formalin seems to be more or less gradually acquired. For a time the hands may be daily dipped into formalin solutions varying in strength from 1 to 5 per cent. without ill effects; then itching and vesicles begin to appear, which result in troublesome eczema if the use of the formalin is not immediately given up. The susceptibility of the skin is much increased after such an attack, mere handling of specimens preserved in formalin being sufficient to cause a return of the eczema.

The Histopathology of Two Cases of Cutaneous Tuberculides, in One of which Tubercle Bacilli were Found.—MACLEOD and ORMSBY (*British Journal of Dermatology*, October, 1901) examined the lesions from two cases of cutaneous tuberculides. These lesions were acne-like, bluish-red nodules, situated in the one case on the extensor aspect of both legs below the knees, in the other upon the forearms, hips, thighs, and legs. The histological appearances found were as follows: In both cases the initial changes appeared to be in the hypoderm, and consisted of proliferative changes in and cellular hyperplasia around the veins in that region. In both the cellular infiltration rapidly encroached upon and replaced the fat tissue, forming cellular areas more or less encapsulated by the remains of the interlobar septa. It is this early cellular infiltration which is to a large extent confined to the hypoderm, which gives rise to the deep-seated nodules which are scarcely visible clinically, but are usually appreciable to the touch. In both the cellular infiltration extended upward along the capillaries, forming foci in the neighborhood of the sweat-coils, hair-follicles, and sebaceous glands, and finally reached the papillary layer. In both the character of the cellular hyperplasia was similar. In the hypoderm it was more markedly tuberculous in appearance in the second than in the first case, but in both the foci in the corium presented equally the characteristic tuberculous architecture. Tubercle bacilli were found in the giant cell in the latter case. A fibrous stroma supporting the cellular hyperplasia was either entirely absent or was represented by œdematous, degenerated collagen and

elastin. In both cases the cellular hyperplasia developed at the expense of the tissue in the neighborhood and finally became necrotic. The papillary and the subpapillary vessels were congested as soon as the infiltration began to encroach upon the corium, accounting for the congested appearance which the nodules presented clinically. The epidermis was only secondarily affected.

The two cases clinically and histologically had certain marked features in common. In the second case there was a dactylitis and a tuberculous family history; tubercle bacilli were found in the lesions of this case. Although no bacilli were found in the lesions from the first case the similarity of the histological appearances left little doubt in the minds of the authors that it was also tuberculous.

[This report is of extreme interest, furnishing indubitable evidence of the tuberculous nature of these lesions.—M. B. H.]

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Influence of Boric Acid and Borax upon the General Metabolism of Children.—Boric acid and borax are much used as food preservatives, and the question of their influence upon the general nutrition of the consumer is one of great interest. The literature on the subject is very voluminous, but the conclusions drawn by the investigators are most conflicting. In order to obtain reliable data upon which to base conclusions, Drs. F. W. TUNNICLIFFE and OTTO ROSENHEIM (*Journal of Hygiene*, April, 1901, p. 168) conducted a series of comparative metabolic observations on the human subject, extending over a considerable period. The observations were made upon three children, who were kept under constant supervision. Their ingesta were accurately weighed and their excreta were collected daily without loss. Both ingesta and excreta were carefully analyzed by the most approved methods. The results of the very numerous analyses are given in detail. The general conclusions arrived at may be summarized as follows: Boric acid in small doses up to 1 gramme per diem, continued for some time, exerts no influence upon proteid or phosphorus metabolism, has no effect upon the assimilation of fat, and exerts no inhibitory effect upon intestinal putrefaction. Body-weight increases. The quantity of dry feces is not affected, but their nitrogen and phosphorus percentage is slightly decreased. Borax in continued doses of 1.5 gramme has no influence upon proteid or phosphorus metabolism, may or may not improve fat assimilation, does not interfere with increase in weight, has no effect upon the weight of dry feces

and their nitrogen and phosphorus percentage, and tends to increase intestinal putrefaction. Both boric acid and borax are quickly eliminated, hence cumulative action is improbable. Neither substance will affect the general health and well-being.

Purification of Water by Sodium Bisulphate.—Tabloids of sodium bisulphate have recently been recommended by Drs. Parkes and Rideal for the treatment of water by armies in the field. Three tabloids are dissolved in each pint of water, and fifteen minutes' contact allowed before drinking. They state that *B. typhosus* is killed by only five minutes' contact with the agent in the proportion of 15.5 grains to the pint, but recommend fifteen minutes to insure sterility. The process has been tested by DR. A. WARNER (*Public Health*, July, 1901, p. 700), who found that a contact of fifteen minutes causes a striking reduction in the number of typhoid bacteria in an infected water, but does not produce sterility. In a majority of cases the bacillus is completely destroyed by a contact of thirty minutes. *B. enteritidis* is as resistant as *B. typhosus*, and *B. coli communis* still more so. *B. cholerae* is destroyed in ten minutes. It has been claimed that the tabloids impart an agreeable acid taste and materially aid in quenching thirst, but Warner asserts that to some the taste is unpleasant and would probably soon become irksome. A soldier drinking five pints of water in a day would swallow over seventy-five grains of the salt, an amount which, Warner suggests, would have the effect of increasing thirst rather than of quenching it.

A New Test for Formaldehyde in Milk.—In the process of estimating nitrogen by the Kjeldahl-Gunning method in a number of samples of milk which had been preserved with formalin, A. G. LUEBERT (*Journal of the American Chemical Society*, September, 1901, p. 682) noticed a peculiar violet coloration of the potassium sulphate crystals and of the sulphuric acid surrounding them. This was especially marked when the milk was added to the potassium sulphate and the acid carefully poured down the side of the digestion flask so that the liquids did not mix, and did not occur with milk which contained none of the preservative. He recommends the following test: Five grammes of coarsely powdered potassium sulphate are placed in a 100 c.c. flask, 5 c.c. of the suspected milk are distributed over it by means of a pipette, and 10 c.c. of sulphuric acid (specific gravity 1.84) carefully poured down the side of the flask. The whole is now allowed to stand quietly until the color develops. If formaldehyde is present, the violet coloration occurs in a few minutes; if none is present, the liquid will at once assume a brown color, rapidly changing to black. The test is said to reveal the presence of one part of formaldehyde in 250,000 of milk.

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APRIL, 1902.

CASES ILLUSTRATIVE OF THE LOCALIZATION OF THE
MENTAL FACULTIES IN THE LEFT PRE-
FRONTAL LOBE.

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ATTEMPTS to localize the mind in the human body began almost as early as the recognition of its existence. Theories were successively formulated during many centuries which were wholly speculative and often without a vestige of reason, but which followed a logical sequence and led to rational, though usually mistaken, conclusions. The successive steps in this progressive development of an idea were the separation of the moral from the intellectual faculties; their localization in different organs; their recomposition in the brain; the restriction of the mind to a definite seat in some one of the various encephalic regions; and, finally, the assignment of individual mental faculties to separable cerebral areas. The several phases of localization enumerated were not chronologically distinct. During the whole period of from twenty-three to twenty-four centuries as new theories were developed older ones persisted. There could be no settled beliefs because there were no sufficient foundations of fact, and while much of the anatomy of the brain became known, little of its physiology or pathology was discovered.

In the first decade of the nineteenth century a more rational study of cerebral physiology than had been made before was undertaken by Gall, who was also the first to conceive the idea of a methodical plan of cerebral localization. His later work, in collaboration with Spurzheim, which included not only cerebral physiology and anatomy together with craniotomy, but also psychology, profoundly impressed the minds of his contemporaries and immediate successors. His theory of localization, founded upon a comparison of known or assumed individual

characteristics with the configuration of the cranium, though later proved unsound and repudiated by physiologists, doubtless inspired the remarkable activity in experimental research directed toward the elucidation of cerebral physiology which followed in the early part of the century.

In 1825, Bouillaud, relying upon pathological observations and accepting the methods of Lallemand, who had been the first to systematically and critically study cerebral symptoms with a view to their future comparison with coincident changes in cerebral structure, made the earliest defensible localization of a cerebral function. He had been led by what he had observed in cases of cerebral disease to seek a centre of control for articulate speech. Having become satisfied that intimate relations subsisted between a more or less complete loss of speech and a corresponding alteration of structure in the anterior regions of the brain, he submitted his conclusion to the test of comparison with cases collated by other observers. Whenever, either in his own cases or in others, he discovered loss of speech among symptoms, he later found lesion of the anterior cerebral lobes; whenever he found such a lesion he learned that loss of speech had been a symptom, and there was no exception to this rule. By a similar inductive process of reasoning from ante-mortem and post-mortem observation he determined that the control of muscular movements in general was resident in the posterior regions of the brain. If these early localizations of Bouillaud were less precise than those made for the same important functions in more recent years, it should be borne in mind that cerebral topography then existed only in an elementary form, and that he was unaided by any practical suggestions derived either from previous physiological experiment or from the observations of earlier or contemporary writers. Flourens had not yet published the results of his experimental researches, and previous experimenters had contributed nothing to localization; Andral, who was convinced that paralysis could result from a lesion in any part of the hemispheres, scouted the idea of a connection between loss of speech and lesion of the anterior cerebral lobes; Lallemand believed that nothing had been accomplished in cerebral physiology except by Gall and Spurzheim, whose fanciful theories he accepted with enthusiasm; Haller, Zinn, Lorry, Sabouraut, and all those who wrote about the brain at a still earlier period, were only concerned with its crossed action, without regard to function.

The experiments of Flourens and the observations of Bouillaud were practically synchronous. In 1824, one year earlier than the publication of Bouillaud's treatise,¹ Flourens² published his experimental study

¹ *Traité Clin. et Phys. de l'enceph.*, 1825.

² *Rech. Exper., etc., du Système Nerveux*, Paris, 1824.

of the nervous system in the lower animals. His conclusions, so far as the localization of functions in the cerebral lobes is concerned, were more indefinite than those of Bouillaud, and were entirely at variance with them as regarded the inclusion of motor centres of control. As to the intellectual and perceptive faculties, which he believed to constitute but a single faculty, he asserted their conjoint occupancy of the whole cerebrum.

Flourens continued his physiological investigations for twenty years or more, but no important advance was made by him or by other physiologists or pathologists until Broca, in 1861, localized the centre for articulate language in the posterior part of the second or third frontal convolution, and afterward exactly defined it in the posterior part of the third frontal convolution of the left side. This definite localization, like the earlier approximation of Bouillaud, was the result of careful comparison of symptoms with subsequent post-mortem disclosures.¹

In limiting the centre for articulate speech to a single convolution, Broca believed he had established a principle of localization which has since proved to be true for many faculties. Gall had first recognized the fact that many functions were localized in the brain, but failed in his attempt to establish their locations by an arbitrary delimitation of cranial areas. Bouillaud had only roughly approximated the truth in discovering that the control or exercise of different muscular movements was dependent upon the integrity of different cerebral regions of large and ill-defined extent. Broca believed, as did Aubertin, that if the localization of the faculty of articulate language in one frontal convolution should be substantiated it would establish the general fact that individual faculties are localized in single convolutions. This opinion seemed to be strengthened some years later by the demonstrations of Fritsch and Hitzig that various centres of control were limited not only to single convolutions, but, as in Broca's instance, to parts of convolutions. It is also likely to prove true for some of the special senses, and approximately so for others; but it has failed in establishment as an absolute law.

Broca's announcement of his discovery of a special centre for articulate language was at once challenged by his contemporaries, and was for some years a storm-centre of discussion. Aubertin accepted it without reserve as true. Trousseau, on the strength of a single case of left hemiplegia with loss of speech, unverified by necropsy, pronounced it to be not only unwarranted in fact, but entirely opposed to physiological principles. Hughlings Jackson, in 1864, after a judicial summary of the various considerations which favored and opposed Broca's conclusion, declared himself to stand on neutral ground.²

¹ P. Broca. *Bull. de la Soc. Anat.*, August, 1861.

² *Diseases of the Nervous System*. London Hospital Reports, 1864, vol. i. p. 460.

To-day no fact in cerebral localization is more absolutely unquestioned.

The following decade and the beginning of the next was a period of remarkable and successful activity in this special field of physiological investigation. The only positive and definite localizations made up to that time were those of the respiratory centre in the medulla oblongata and of muscular co-ordination in the cerebellum by Flourens, and of articulate speech in the left frontal lobe by Broca. In 1870 Fritsch and Hitzig, by the application of the galvanic current to the cerebral cortex of dogs, demonstrated centres of control for all the more important muscular movements of the body. In immediately succeeding years Ferrier, Horsley and Schafer, Hughlings Jackson, Putnam, Dalton, Bartholow, Wood, Braun, Goltz, Munk, and a great many other experimentalists and pathologists, repeated, elaborated, and verified the experiments of these observers, and extended the field of cerebral localization until it covered nearly everything in the topographical physiology of the brain which is known at the present time.

The positive results of this work include the determination of centres of control for nearly or quite all the groups of voluntary muscles, for general sensation, and for the more important special sensations of sight and hearing. Some question has been raised as to the correctness of Ferrier's localization of tactile sensation in the gyrus fornicatus and hippocampal region, but the weight of anatomical, pathological, and experimental evidence seems to justify his conclusion.

The evidence which points to centres of control for taste and smell in the hippocampal region is perhaps still insufficient to constitute absolute proof.

The existence of special heat centres—thermogenetic, thermotoxic, and thermolytic—though asserted, and by Wood and Ott localized in various parts of the brain, is still a subject of discussion, as is that of vasomotor and certain other centres.

The question of control of the intellectual and moral faculties—whether they reside in some distinct region of the brain or whether they constitute a function of the brain as a whole—has received less careful investigation, and has been consequently further from solution than any other in cerebral physiology. This has followed, naturally, from the difficulty of interpreting the results both of experiment upon the lower animals and of pathological observation of the human subject. Physiologists and pathologists have differed equally in opinion as to the significance of what they have observed. The two writers of highest authority who have given the subject consideration—Ferrier and Hughlings Jackson—arrived at conclusions which were radically different. This difference may be sufficiently indicated by brief quotations from Ferrier:¹

¹ Functions of the Brain, second edition, New York, 1886, pp. 427-460.

"The sensory centres are to be regarded not merely as the organs of consciousness of immediate sensory impressions, but as the organic register of their own sensory experiences. This organic memory is the physical basis of retentiveness, and the property of re-excitability is the organic basis of recollection and ideation."

"These sensations are accompanied in consciousness by feelings which are divisible into two great and opposite classes of pain and pleasures. Just as sensations are the subjective side of certain physical modifications of the nerves and nerve centres, so pleasurable or painful feelings may be regarded as the subjective expression of physical harmony or disharmony between the organism and the influences acting upon it."

"The various sensations, feelings, and desires, present or revived, singly or in associated combination, form the incentives to action, the motives to volition.

"The motor activities called into play by definite feelings and sensations, present or revived, constitute volitional movements; and the organic cohesion formed between the sensory and motor centres, persistently enduring in these centres, is the physical basis of our intellectual and volitional acquisitions in all their manifold range and complexity.

"We have, therefore, in the cortex centres of sensation and centres of motion, centres of sensory and centres of motor acquisition or registration, an organic sensory and an organic motor memory.

"The motor centres are not merely the centres of impulse, but also the centres of reproduction of volitional movements."

He finally thus summarizes his reasons for dissenting from Jackson's expressed belief in higher centres of mental control:

"It has been assumed by several writers, among others by Hughlings Jackson, that in addition to the sensory and motor substrata which have been demonstrated and defined by physiological and clinical research, there are other higher motor as well as sensory centres, in which all the motor and sensory functions are again represented and form the substrata of the higher mental operations. This hypothesis receives no confirmation from the facts of experiment, nor does it appear to me at all necessary to explain the facts either of normal or abnormal mentation. We have in the sensory and motor centres of the cortex the substrata of the respective forms of sensory perception and ideation, and of the individual acts of volition, simple and compound, as well as of the feelings associated with their activity. It seems more reasonable to suppose that there may be higher and lower degrees of complexity or evolution in the same centres than to assume the separate existence of more highly evolved centres, for which no evidence is obtained by the results of experimental research."

These higher centres, which were described by Hughlings Jackson as at once "the least organized, the most complex, and the most volun-

tary," the highest of evolution, and "potentially the whole organism," were, he thought, "shown by facts to be in the forepart of the brain, which serves in the motor aspect of the mind and represents the movements of all parts of the body."¹

The quotations, however inadequate they may be as an expression of the general conclusions of these authors, will serve to show their divergence of opinion in regard to the localization of the intellectual faculties.

Those who for whatever reasons believe, as does Hughlings Jackson, that there are higher centres which form the substrata for the higher mental operations, have without exception, like him, placed them in the frontal lobes. As the posterior portion of the frontal convolutions has been demonstrated to control special motor functions, including the co-ordination of muscles of articulation, mental control, if resident at all in the frontal lobes, must be localized in their prefrontal region.

Previous to the publication which I made in 1894² of some personal observations of traumatic cerebral lesions no attempt had been made to more specifically localize centres for control of the mental faculties; nor have there been such attempts since that time, so far as I know, by any other writer. In this and in a later publication³ an analysis of a considerable number of cases in which injury had been essentially limited to one or both frontal lobes led to the conclusion not only that such control probably resided in the prefrontal region, but that it was in the left, to the exclusion of the right lobe. In a series of 225 necropsies there were included 72 instances of laceration of one or both frontal lobes, exclusive of cases of pistol-shot wounds in which almost instantaneous death precluded history. In 33 cases morbid mental conditions were inappreciable by reason of primary and permanent unconsciousness; and in 11 others an early fatal issue, preceded by only partial consciousness or by the existence of delirium from the general cortical lesion, made the recognition of indications of the local injury equally impossible; 28 cases remained in which the attendant conditions permitted an estimate of the direct results of frontal lesion. These comprised :

| | | | | | | |
|---------------------------------|---|---|---|---|---|----|
| Laceration of left frontal lobe | . | . | . | . | . | 11 |
| " " right frontal lobe | . | . | . | . | . | 7 |
| " " both frontal lobes | . | . | . | . | . | 10 |

These cases were detailed and also summarized. In a second edition of the same work 11 cases subsequently observed were also summarized and were added to the first series, making an aggregate of 16 lacera-

¹ Evolution and Dissolution of the Nervous System. British Med. Journ., March and April, 1884.

² New York Med. Journ., November 10, 1894, to January 12, 1895.

³ Traumatic Injuries of the Brain, etc. New York, 1897, 1900.

tions of the left, 8 of the right frontal lobe, and 15 of both lobes together. I shall quote largely from a summary of the lesions as observed in the first series of 28 cases.

The associated lesions were disregarded as limited to areas of the brain which had been already demonstrated to have other physiological relations or to have no relation to the exercise of the mental faculties.

The lacerations were not always of the same character, situation, or extent. All but 1 involved the anterior region of the lobe; 13 had led to a more or less complete disintegration, either directly or as an effect of hemorrhages; 13 of the cortical injuries were confined to the base, and the 2 others were wholly or in part upon the antero-superior surface. The lesions were cortical and subcortical in case of either lobe, and when both lobes were implicated the dual injuries were usually of the same character. The symptoms held some relation to the situation and extent of the lesion. In the subcortical excavations and disintegrations there was abrogation of mental power rather than aberration in its manifestations, the patient's condition being sluggish and apathetic. In the cortical lacerations, in place of apparent default of intelligence, there were perverted memory, lack of attention and control, incoherence, delusions, or the stupor which comes from confusion rather than from paucity of ideas; the mind was alert to external impressions, though they were not always rightly comprehended. These distinctions, which are general, are by no means absolute. The localizing symptoms were naturally less pronounced or absent in cases in which the frontal lesion was trivial or in which, from the severity of the local injury, death occurred at an early period; this, however, did not prove to be the fact in every case. If recovery ensued the longer duration of symptoms should similarly increase the probability of determining with certainty the presence or absence of specific mental disorder. This inference is justified in several of the appended histories, in some of which the exact frontal lesion was well assured or positively ascertained.

The converse proposition—that laceration of the left frontal lobe is the sole traumatic lesion which occasions a direct loss or derangement of intellectual function—is, so far as can be judged from a study of the same series of cases, only a little less absolutely true. In the 225 necropsies death had been preceded by such deficiency or derangement in 4 instances in which this injury was not disclosed. In 1 of these—a case of pistol-shot wound of a parietal lobe—some slowness of comprehension was added to an hysterical melancholia, which had led to a suicidal attempt. This may be properly excluded, as mental disease existed before the reception of the injury. In each of the other 3 mental decadence was evident; in 2 general hyperæmia and œdema were excessive, and in the third a large localized subarachnoid serous

effusion compressed the frontal lobes. These exceptional cases are scarcely more than 2 per cent. of the whole number of fatal intracranial injuries verified by necropsy, in which the frontal lobes were not the seat of destructive injury; and in them those lobes, though not wounded, were in 1 instance the only parts affected by a limited compressing lesion, and in the other 2 were included in a general lesion of excessive severity. There were no instances in which a laceration of any other cerebral region was attended by characteristic mental changes.

In the second series, consisting of 11 cases, the lesion in each was laceration; in 1 it terminated in abscess, and in all but 1 involved the anterior or middle region of the frontal lobe or lobes; in 4 the lesion was confined to the base, in 1 to the deeper part of the subcortex, and in the rest to the superior convolutions. They all followed the rule established in the earlier series, that the deeper the laceration, and therefore the more destructive the injury, the more mental default preponderated over mental aberration. In line with this observation is the fact that in the recovering cases, which from the symptoms were presumably cases of frontal lesion, mental aberration was more frequently noted than mental default. As the patients recovered, it may be assumed that the lesion was less severe, and therefore irritant rather than destructive.

The salient points in each of the foregoing 39 cases, as before stated, were noted in brief and afterward summarized in the work before mentioned, *Injuries of the Brain, etc.* Still later cases increase the number of the writer's personal observations of frontal lesions verified by necropsy to 46. The whole number of these more recent cases, together with those included in the second series and previously unpublished, are here described in detail.

CASE I. *Cerebral tumor confined to the left frontal lobe.*—The patient, a Russian, aged thirty-two years, was transferred by Dr. Joseph B. Bissell from the insane pavilion of Bellevue Hospital to my care in the fourth surgical division. His mental condition was so far disordered that his history could be learned only from an account given by his wife, who was a woman of intelligence. She stated that he had been a man of irreproachable habits, who never drank or smoked; that he was well educated, speaking and writing four languages; was studious and an accomplished musician, holding the position of first violin in an orchestra of note; and that he never suffered from any sort of previous illness. She further stated that about eight weeks prior to admission he began to have constant headache referred to the inferior occipital region, and to show at times some uncertainty of gait, which was unilateral, but not always on the same side; that he became slow of apprehension, sometimes using the wrong word in writing, but immediately correcting himself; called things by the wrong names; mixed German with English speech; was incoherent, and, though he could read, could not write, misspelling and repeating words and mixing languages as he did in speech; that about the same time he began to suffer from

attacks of vomiting, and that these continued for about six weeks; that a week later his speech became difficult; that a little later still he became utterly unable to speak intelligibly or to write in any language, and lost all interest in his surroundings; that his mental condition was one of apathy rather than of despondency; and as in consequence he paid no attention to what was read to him, and never resorted to his violin, it was impossible to know whether he could comprehend what was read or whether he retained any power of musical expression; that five weeks later he began to talk irrationally; that his memory failed; and, finally, that he had seen everything in a red light with his right eye from the first, but that vision was otherwise unimpaired. This chromatic defect was obviated by the wearing of a black glass over the affected eye. He was somnolent and thick of speech from the beginning of his illness.

On admission to a surgical ward, November 17, 1900, he was well nourished, of rather slender physique, and of refined and delicate appearance. His temperature, pulse, and respiration were normal—98.4° F., 80, 20. He could walk, but with great difficulty, in consequence of a paretic condition of both lower extremities. There was also a barely perceptible paresis of the left side of the face, indicated by a flattening of the nasolabial fold and a slight drooping of the angle of the mouth. The reflexes on the right side were slightly exaggerated, and upon the left side were absent or diminished. The right pupil was slightly dilated, and reacted to light and accommodation. Chromatopsia still persisted; urinary and fecal discharges were controlled; and the urine was of normal character. His mental condition, as then observed, did not afterward essentially change, but its impairment was rapid and progressive. He was drowsy when admitted, as it has been said he had been from the first. He was afterward at times apathetic or lethargic, and these periods increased in frequency as time went on; but he never approached a condition of stupor. He would always respond to questions, and was often even garrulous when aroused. His attention, though readily attracted, could not be held, and consequently his speech was wandering and disconnected—compared by Dr. Peter A. Callan to the prattling of a child. His answers to simple questions which involved but one idea were rational and proper, though their comprehension was usually slow and difficult. His attempts to define or describe were unintelligible, and not infrequently ended in mere meaningless chatter. He suffered from limitation and confusion rather than from distortion of ideas. He had, so far as could be ascertained, no delusions; he was not emotional and was never violent. He always recognized his wife, his attendants, and his surroundings. His memory was defective; it apparently served him from day to day, but he had forgotten where he was born and where he had lived. He had varied forms and degrees of sensory aphasia—alexia, apraxia, and agraphia. He could read neither written nor printed words, though his sight had not suffered. He could recognize many objects when their names were told him, but of others he had no conception either of their names or uses. He could articulate distinctly, and was able to express in speech such ideas as he possessed, but was unable to write either voluntarily or by dictation. He spoke only in the Russian tongue, which, as stated by his wife, he had not spoken before for fourteen years, and, as he indicated to her, thought it queer it had come back to him, and was

annoyed that his efforts to speak in English or German failed. He could still understand either language when it was spoken.

An ophthalmic examination, made four days after his admission to the hospital by Dr. Callan, disclosed descending neuritis in both eyes, with paresis of the right external rectus muscle.

In the progress of the case general symptoms were few. For the first ten days he complained only of his eyes, but he was unable to indicate the nature of this trouble, which it seemed most probable was an orbital pain. At a later period frontal headache was a symptom. From the tenth to the fifteenth day vomiting was frequent. About the twentieth day he began to pass urine in bed, not from lack of control, but, as it seemed, as a matter of convenience; and at a still later period feces were passed in the same manner. His temperature was often normal, never subnormal, and never above 99.6° F. His pulse varied from 68 to 96, and his respiration from 18 to 24. On the day of his death his temperature was 98.6° F. to 98° F., his pulse 64 to 56, and his respiration 18.

Two days before the patient's death a final examination was made by Dr. Alexander Lambert, who noted: "Sensation delayed on the left side and slightly diminished on the right; all reflexes increased; ankle clonus present, more marked on the right side; tremor of hands, especially of right; slight tremor of tongue; vision impaired; patient could correctly count fingers when held before either eye; answered simple questions after some delay; seemed unable to formulate voluntary speech, and could comprehend ideas only slowly, if at all; could not write or read either words or figures; could recognize some objects when told their names, but not others; called nearly everything 'terpsichor.'"

On the day before his death he rather suddenly failed in strength, and on the thirty-first day after admission he died unconscious.

In the belief that the symptoms of mental disorder presented were focal, I made a diagnosis of cerebral tumor occupying the left frontal lobe, and thought it to be confined to that lobe, in default of other localizing symptoms, except amnesic aphasia, which could be accounted for on other grounds. It was conjectured that this tumor was either a glioma or a sarcoma. Abscess was excluded in the absence of previous head injury or of any disease which might have led to infection.

Necropsy. Brain, immediate inspection: Cranium and dura mater both notably thin; no indication of either old or recent traumatism or disease; no adhesion of either of the membranes at any point; no sub-arachnoid effusion; pial vessels moderately distended; left hemisphere larger than the right; median surface of left frontal lobe bulging across the superior longitudinal fissure and slightly indenting corresponding surface of the right lobe; superior surface of the left frontal lobe presenting in its central and anterior portions a distinct prominence, which was of a faint and dull red color as seen through the pial and arachnoid membranes; posterior to this tumor an area extending nearly to the median line and to the precentral lobule, giving a sense of fluctuation; left frontal convolutions broad and flattened, and sulci indistinct.

After a week's interval of immersion in a 2 per cent. solution of formalin, horizontal section of the brain was made through the central plane of ventricles, disclosing a tumor of pinkish color and of moderately firm consistency, strictly confined to the left frontal lobe, three

inches long in its antero-posterior diameter and largely involving the inferior portion of the lobe. Its posterior half was an empty cyst. The left ventricle was displaced inward and backward. The anterior portion of the left caudate nucleus was flattened and pressed backward. Fibres from Broca's convolution were apparently not involved; the entire remainder of the subcortical structure of the left frontal lobe was practically destroyed.

The brain was subsequently submitted to Dr. Harlow Brooks for more minute examination. He made the following report:

"As a whole the brain is large and well formed, and the markings of the two hemispheres are symmetrical. The left lobe is occupied by a well-defined tumor, which distends its inner aspect and appears to be limited to it. The sulci on the right side are deep and the convolutions broad and ample; on the left side the same conditions obtain, except that the convolutions are flattened as though from internal pressure.

"The pia-arachnoid membrane shows no lesion in the hardened specimen with the exception of congestion of the vessels, especially of the veins. The membranes are not adherent to the cortex, not even over the site of the tumor, but the vessels in this immediate region are more injected than elsewhere. The large vessels at the base of the brain show no lesion other than the general congestion and a very moderate degree of arterio-sclerosis. The trunks supplying the deep parts of the brain are in a similar condition, but noticeably less markedly congested, probably from hypostasia.

"The sulci and gyri of the cortex are not typical on either side, and it is therefore difficult to accurately locate the situation of the tumor, although its outlines are distinct, and it is of a different color from the contiguous structures.

"The tumor occupies the anterior two-thirds of the third left frontal gyrus; the triangular portion (Broca's convolution) is entirely free from the growth, though its anterior border has been considerably compressed, atrophied, and forced backward. The superior mid-portion of the gyrus also is not immediately involved, but has been considerably compressed and is displaced upward in its anterior portion. The lower border encroaches to a slight extent on the inferior extremity of the second convolution.

"The surface of the tumor is raised above the general surface of the cortex from 1 to 2 mm. Its boundaries, except in its anterior inferior perimeter, are sharply defined by deep sulci, which have their counterparts in the markings of this region of the right hemisphere. The cortical surface of the tumor also shows sulci similar to those of the like region on the involved side of the brain, except that here they are more shallow. The lower boundary of the neoplasm has invaded the overhanging portion of the left third frontal convolution for the distance of 1 cm.

"The general tissue of the brain presents a normal appearance excepting in those parts invaded or compressed by the neoplasm. The cortical layer of gray matter is thick and regular, but it is differentiated with great difficulty over the surface of the tumor, where the growth seems to have infiltrated, in most places entirely through the surface.

"The greater part of the tumor is made up of a cyst-like cavity which now encloses a considerable mass of fibrin clot. This cyst occupies almost the entire portion of the left frontal lobe, the inner wall of which it has bulged, so that the right hemisphere has been displaced. Its thickest wall is the cortical one, which measures from 2 to 5.5 cm. It is made up of a spongy, highly vascular, neoplastic tissue, which imperceptibly merges with that of the cortex. Internally and laterally the outlines of the cyst are mapped out by a well-defined capsule, varying from 1 to 3 mm. in thickness. The inner surface of this membrane is apparently not of new-growth, but is evidently a thickening of the neuroglia and connective tissue.

"The cavity of the cyst is multilocular. Its inferior portion has excavated the left frontal lobe almost to the cortex. Neoplastic invasion of the brain tissue has taken place only in the external portions of the lobe. The cyst has, however, excavated or displaced so much of the white matter of the frontal lobe that only 3 cm. of the normal tissue bordering on the great median fissure remains. It has also compressed the tissues posterior to it, so that the entire left basal ganglia have been forced backward, and the left internal capsule has been displaced so that it occupies a position almost at right angles to the longitudinal axis of the cerebrum; this has been from the forcing backward of the anterior portions of the putamen and caudate nucleus. The cyst has also intercepted or displaced the efferent fibres from Broca's convolution, and has certainly destroyed all association fibres leaving or passing to the left third frontal convolution. Probably many fibres of the first and second frontal convolutions are also interrupted.

"The cyst does not communicate with any of the ventricles, but its walls have greatly compressed the anterior portion of the left lateral ventricle, with the result that the posterior horn on this side has been considerably dilated.

"Except for moderate dilatation of the ventricles, the medulla, pons, and cerebellum show no gross lesions."

"*Microscopical Examination.* The tissue of the tumor is highly cellular. Its most remarkable characteristic is the presence of true embryonic neural elements. These neuroblasts are not abundant, but a few may be found in nearly every field; they are flask-shaped, and usually have one or more distinct processes, sometimes of considerable length. Often these neuroblastic cells contain several ill-formed nuclei and show irregular karyokinetic figures, as though an attempt at reproduction of the partly differentiated cells was being made. Branching, irregular-shaped cells are much more numerous, and in places can be shown to enter into the formation of an ill-defined stroma like that of the embryological spongioplasm. These differentiated cells do not make up the greater bulk of the tumor, which consists mostly of polymorphic cells and of a stroma resembling the structure of a mixed-cell sarcoma. Vessels are numerous. They are largely poorly formed, and their walls are made up in part by the cells of the growth. Corpora amylacea are frequent in all parts of the tumor; apparently they have been formed from or in the degenerated nervous tissue as the new-growth was displacing it.

"Sections through the cortex of the left third frontal convolution in the invaded portion show no microscopical distinct line of invasion, the growth being apparently an infiltration process, with substitution of tumor tissue for the degenerated neural elements. Neuroblasts are not

frequent in these borders of the tumor, but the sections contain a good many remnants of the original nervous tissue, especially of degenerated ganglion cells, the cytoplasm of some of which still shows the presence of Nissl bodies. The nuclei of several of these ganglion cells show curious arrangements of the chromatin, as though an attempt at mitotic cell division were being made. Some of these cells also exhibit a deposit of amyloid in them; perhaps this accounts for the formation of corpora amylacea, which are found to be frequent in the older portions of the tumor.

"The tumor is a true neuroglioma."

CASE II. *Laceration of left frontal bone.*—Ambulance history: The patient, aged seven years, had been assaulted with the blunt edge of a hatchet, and was unconscious and in extreme shock. The pupils were moderately dilated, reacting to light; breathing slow and stertorous.

On admission to hospital patient was conscious, moaning and irritable; pulse barely perceptible; right arm and leg rigid; slight hemorrhage from mouth; hæmatoma of both orbital regions and of right fronto-parietal region; four small wounds of the scalp made by blows in the left fronto-parietal region, through which a depression of bone could be felt. An incision connecting these wounds disclosed a comminuted fracture of the left parietal and frontal bones involving the coronal suture, from which brain was oozing.

From the first to the fourth days the temperature ranged from 100.6° to 102.2° F., and the pulse from 86 to 66; urinary and fecal control was lost, consciousness was retained; and a little pus was discharged from the anterior portion of the wound.

On the fourth day the temperature rose rather suddenly from 101.4° to 104.6° F. and the pulse from 76 to 106.

On the fifth day the temperature was twice reduced by alcohol baths to 104° F., and each time rose again to 105.2° F. Sensation was diminished upon the whole right side, and the right arm was anæsthetic and markedly parietic.

On the sixth day the range of temperature was 104.2°, 102.8°, and 104.4° F.; range of pulse 94, 128, 98. Convulsive paroxysms involving the right face and the flexor muscles of the right arm occurred at hourly intervals, and there was a loss of power in the right leg.

On the seventh day the convulsive movements extended to the extensors of the left arm and flexors of the right leg, and pus was discharged freely from the wound. The range of temperature was from 103.8° to 105° F.

On the eighth day the convulsions became shorter and less frequent, with rigidity of the left arm during the intervals, and the patient died.

The mental condition remained unchanged from the time consciousness was restored, on admission to the hospital, until its final loss. The patient never moved unless disturbed, never spoke, and never showed any signs of mental action except as he cried out and tried to avoid disturbance. He looked at his mother without sign of recognition. He had no apparent knowledge of urination or of defecation. He lay motionless, with a slight color in his face, breathing gently and with the facies of consciousness, living a merely vegetative existence.

Necropsy. Purulent effusion covered both hemispheres and filled the basic fosse; laceration of the anterior third of the first and of the whole of the second and third left frontal convolutions. Superficial contusion

of the right parietal lobe from the fissure of Sylvius to within three-fourths of an inch of the longitudinal fissure, and of the lower part of the right occipital lobe as far up as the angular gyrus.

CASE III. *Contusion of left frontal lobe, with gunshot fracture of vertex.*

—The patient was shot at San Juan, Cuba, July 1, 1898. The bullet fractured the vertex at the left fronto-parietal junction, without penetrating the cranial cavity. He remained five days unconscious in the field hospital. After regaining consciousness he had motor aphasia for sixteen days, but there was no subsequent impairment of speech. No further details of the early history of this case were obtainable beyond the fact that he was trephined. Since that time he had been always conscious of certain mental deficiencies.

He entered St. Vincent's Hospital September 29, 1898, where he remained under observation until November 18th. During this period he presented no symptoms except those of mental decadence. He was slow of comprehension, greatly deficient in power of attention, melancholic, apathetic, and emotional, and his memory of recent events was impaired. He suffered from vertigo when walking, but had no loss of muscular power or co-ordination. His nutrition failed.

The osseous wound was exposed, November 18th, by a semicircular flap which included the periosteum and disclosed the dura mater. The cranial wound of original operation was one and a half inches long antero-posteriorly and one inch wide, and was situated just above the squamous suture crossing the coronal suture, and thus involving both the parietal and the frontal bone. The dura mater was greatly thickened, adherent to the edge of the osseous opening, and without pulsation. After separation of the dura mater from the osseous edge a needle was passed through it into a meningeal cyst, from which one to two drachms of serous fluid was discharged, and dural pulsation was restored. The dural membrane was then incised, and the brain surface thus disclosed was seen to be soft, grayish in color, and non-vascular; and softening was found by the needle to extend one-half inch downward. During the two days following operation the patient declared himself to feel "quite naturally for the first time since the wound was received." On the third day afterward the cerebral wound became infected, apparently from sloughing points in the scalp flap, due to injudicious pressure by compress, and death resulted later from cerebral abscess.

The original lesion was found on necropsy to be confined to the posterior portion of the three frontal convolutions of the left side.

CASE IV. *Laceration of left frontal lobe.*—Patient admitted to hospital in deep coma, with general muscular rigidity and loss of fecal and urinary control. Temperature, 97° F.; no other symptoms. On the following day he was evidently conscious, and, by intelligent movements, attempted to avoid irritations, but would not speak or respond to orders given to him. The pupils and reflexes were normal. Ecchymosis appeared about the right ear. Temperature, 100.2° F. On the third day he answered questions rationally. On the sixth day he at times answered rationally, but was stupid. On the seventh and eighth days he was incoherent, and did not know where he was. On the ninth day he lost consciousness, and on the tenth day he died.

Necropsy. Fracture through middle and posterior basic fossæ. Pial hemorrhage over whole of left hemisphere. Deep laceration of anterior third of left frontal lobe, involving the first, second, and third convolu-

tions, one and three-quarters by three-quarter inches in diameter, and filled with a grumous material. Superficial laceration of anterior part of the left temporal lobe. Brain oedematous.

CASE V. *Laceration of left frontal lobe.*—Patient found unconscious in the street. On admission to the hospital he was irritable and irrational, but not delirious. There was free hemorrhage from the left ear. Temperature, 98.8° F.; pulse, 62; respiration, 20. The pupils were moderately dilated; no loss of control of the bladder or rectum. Ten hours later the left arm was convulsed and then became paralyzed and anæsthetic. He also became delirious and lost control of the sphincters. Temperature, 101° F.; pulse, 62. He died on the following day.

Necropsy. Dura mater lacerated in left temporal region, and arteria meningia media wounded. Extensive epidural hemorrhage. Laceration of whole superior and lateral aspects of left frontal and temporal lobes. Fracture through left petrous portion and middle basic fossa.

CASE VI. *Laceration of left frontal lobe.*—The patient, who had fallen down stairs, was entirely unconscious, with slightly contracted pupils, general muscular rigidity, and loss of fecal and urinary control. On the second day he was conscious, but apathetic, and would make no response to questions. There was continued muscular rigidity and lack of control of urine and feces, normal pupils and reflexes, and ecchymosis about the right auditory meatus. On the third day he made only inarticulate answers to questions, and had delusions. From the fourth to the sixth day he was much of the time in a condition of stupor, and was at times rational and at others irrational. From the seventh to the tenth day he was quiet, unable to comprehend his surroundings, sometimes incoherent, and usually confused in his response to questions. On the eleventh day coma supervened; the temperature, which had previously ranged at all times from slightly below to slightly above normal, rose to 104.6° F., and on the twelfth day he died. Muscular rigidity was never relieved. The pulse was usually slow—60 to 70.

Necropsy. Linear fracture extending through petrous portion of right temporal into right occipital bone; dura mater thickened and adherent; pial hemorrhage over whole left hemisphere. Laceration of anterior third of left frontal lobe, one and one-quarter inches long and three-quarters of an inch wide, involving the first, second, and third convolutions, extending deep into the lobe, and its cavity containing disintegrated grumous material; small, superficial laceration of each temporal lobe; brain oedematous, with punctate extravasations; no evidence of arachnitis.

This patient was sixty years of age, and was intoxicated at the time he was injured.

CASE VII. *Atrophy of left frontal lobe.*—Patient fell, striking his head upon the curbstone; transient unconsciousness; hemorrhage from right ear; temperature, 97.8° F.; pulse, 80.

Sixth day. Stupor and muttering delirium

Seventh day. Patient very restless—temperature, 99° F.; pulse, 86—at times irrational and at times delirious; comprehended and answered questions, but did not know where he was; temperature, pulse, and respiration normal.

Twelfth day. Patient apathetic.

Thirteenth and fourteenth days. Delirious much of the time; speech thick and difficult to understand.

Fifteenth day. Apathy; no delirium; temperature, 99° F.; pulse, 100.

Seventeenth day. Noticed things; had delusions; at times delirious.

Eighteenth day. Patient rational, but still had delusions.

Nineteenth to twenty-second day. Progressively increasing weakness and apathy, followed by stupor; dysphasia; temperature, 98° to 99° F.

Death on the twenty-fifth day; temperature rose on that day from 102.4° to 109.4° F.

Necropsy. Fracture through right petrous portion into middle basic fossæ; no hemorrhages; subarachnoid effusion confined to right side; excessive general cerebral œdema, left frontal lobe much smaller and firmer than the right.

CASE VIII. Laceration of the left frontal lobe.—The patient was struck on the right side of the head with the butt of a whip, and immediately became unconscious, but afterward walked to the hospital with assistance. On admission his temperature was 97° F., pulse 80, and respiration 24; he was incoherent and without memory of how he had been injured or of his name or place of residence; his pupils were normal, though sluggish in reaction; and he had slight hemorrhage from the right ear. There was no other external evidence of injury. Later in the day he was restless and at times delirious. On the second day he could give an account of the manner of his injury; a small area of ecchymosis appeared over and below the right mastoid. Temperature, 102.2° F.; pulse, 100.

On the fourth day his mental condition was dull and comprehension difficult, though he answered questions rationally. Hemorrhage from the ear continued in small amount; the pupils were contracted and still sluggish, and mechanical restraint was necessary to keep him in bed. Temperature, 99.2° F.; pulse, 86.

On the fifth day he was dull and apathetic, but could comprehend and answer questions sensibly. Temperature, 99° F.; pulse 78 to 82.

On the eleventh day the mental condition was unchanged except that the patient was more drowsy and stupid; lingual and facial paralysis, which began on the second day, persisted; and vesical and rectal control was impaired.

Thirteenth to fifteenth days. The patient was brighter and more talkative, having numerous and varied delusions; physical condition unchanged. Temperature, 98° to 99° F.; pulse, 62 to 84.

Sixteenth day. The patient was in a state of semi-stupor, and when aroused said he was "simply resting to-day, as yesterday he had worked hard;" or, again, that he had been out to a dance hall. Temperature, 98.2° F.; pulse, 68.

Eighteenth day. The patient was delirious for the first time since the day of his admission. Temperature, 99.6° F.; pulse, 82.

Twentieth day. Delusions continued; patient's mental condition more alert; recognized his wife and attendants; nutrition unimpaired.

Twenty-third day. Mind more active than at any previous time; comprehended and answered simple questions; mastoid ecchymosis barely perceptible; lack of rectal and vesical control continued.

Twenty-fourth to twenty-eighth days. Gradually increasing stupor. Temperature, 98° to 99° F.; pulse, 78 to 86; respiration, 20 to 24.

Twenty-ninth to thirty-first days. Patient difficult to arouse; no other change in symptoms.

Thirty-third day. Patient died. Temperature, 103° F.

Necropsy. Fracture through the right petrous portion into posterior basic fossa. Epidural hemorrhage in moderate amount in right temporal region and in posterior fossa. Hemorrhage over left frontal lobe, extending posteriorly over the temporal and inferior parietal regions. Laceration of the left frontal lobe extending through the cortex and involving the first and second frontal convolutions in their middle portion. Moderate cerebral oedema of the right hemisphere and excessive oedema on the left side.

CASE IX. Laceration of the left frontal lobe.—The patient was struck upon the head by a tackle-block which had fallen thirty feet. He was found unconscious by the ambulance surgeon, with both pupils widely dilated and irresponsive to light. Temperature, 97.2° F.; pulse, 86; respiration, 26. On admission to the hospital his symptoms were unchanged. A compound comminuted depressed fracture of the left parietal bone extended from its anterior inferior portion to the parietal eminence, and thence to the mastoid process; the cerebral membranes were disintegrated; and hair, dirt, and pieces of scalp and bone were driven into the cerebral substance; and much brain matter had been extruded and lost. The lacerated and disintegrated brain area involved at least the posterior half of the left frontal and upper part of the temporal lobe; just how far the injured area extended anteriorly and inferiorly could not be accurately determined without resorting to unjustifiable exploration. Semi-detached brain matter was cut away and the brain wound made clean; the osseous fragments were elevated or removed, and the scalp was sutured over the osseous opening. Consciousness returned one hour later, and for fifteen minutes the patient muttered and laughed; the pupils were still widely dilated and irresponsive to light and accommodation; the pulse, temperature, and respiration were normal.

On the second day he was irrational and irritable, but not delirious. He was unable to comprehend what was said to him either in English or in Norwegian, his native tongue; his pupils continued widely dilated. There were no symptoms of muscular disorder; pulsating hernia cerebri, one and one half inches in diameter, with an offensive discharge, appeared just above the mastoid process.

On the third day he was more rational and seemed to recognize his friends, but his answers to questions were unintelligible. His speech was mere gibberish, and he laughed and cried at intervals.

On the fourth day he sat up in bed, began to take nourishment by the mouth, recognized his wife and his attendants, was incoherent at times, irritable when aroused from sleep, and still talked gibberish. The hernia cerebri was reduced to about one-half its previous size.

On the fifth day he regained urinary and fecal control, and was more intelligent and less irritable. The hernia cerebri was sloughing and still increasing in size.

On the sixth day he was very restless and irritable, crying or laughing at intervals, but always having a foolish smile.

On the seventh day he could understand English, but could not speak it, and after each effort to do so began to cry; and this was repeated for an hour or more at a time. His pupils were still dilated. During

this week his temperature ranged from 101° to 99° F., and his pulse from 60 to 90.

At the end of the second week, after an intervening period of active delirium, he again became rational, was still very emotional, and could use a few words of English; his pupils were unchanged, and he still wore a perennial and fatuous smile.

At the end of the fifth week his pupils began to respond to light and accommodation, the hernia cerebri had disappeared, and he was less emotional. He could count up to seven, but no farther, could write ten, but not one hundred, and could tell upon demand the name of his wife or of his son.

At the end of three months he could understand speech if uncomplicated and if the words were slowly and distinctly spoken. In response to inquiry he said that if he should return to his work he would be unable to comprehend orders which might be given to him.

Before his injury he usually spoke in the Norwegian language; for a few days after that time, as already stated, his speech was mere gibberish. When he regained speech it was English only; then a little later he remembered a few words of his native tongue; but his Norwegian vocabulary was not afterward increased, though his range of English was perceptibly extended.

At the end of the three months he was still emotional and cried often, though, as his wife stated, she had never heard him cry before he received his injury. She stated also that he took no interest in ordinary affairs. He could not read either English or Norwegian, though he could read both previous to his accident. He could count as far as seven with some instruction and practice, and could write his name and address and spell them correctly. He had entirely lost the memory of some words, and could not repeat words spoken to him, as coat, vest, etc., but could remember the meaning of the greater number of words. He could recognize the name of the town in which he was born, his age, and his occupation. In general, his memory of events occurring during his lifetime was not much impaired. His power of attention was somewhat improving, but his mental powers were generally weakened.

The symptoms in this case, with the exception of the long-continued dilatation and inaction of the pupils, were at all times confined to disorders of the mind and speech.

The patient was a man aged thirty-five years, of robust physique and temperate habits, and was without constitutional taint.

CASE X. *Atrophy of both frontal lobes.*—The patient, aged seventy-six years, who all his life had been in perfect health and whose mental condition was unimpaired by age, was thrown from a light wagon while driving. Loss of consciousness was immediate and complete, and continued for fifteen minutes. He then walked to a carriage, in which he was taken to his house. He at first refused to enter, saying that it was not his own, and begged to be taken home. This primary delusion continued until his death, two or more years afterward, and was constant and consistent. On the way home he complained of headache, and vomited once.

When visited by his physician, a little later, his temperature was subnormal and his pulse infrequent—56. His temperature remained subnormal, at about 97° F., for several days, while his pulse progres-

sively increased in frequency to 72. The left frontal region was contused and abraded, and the left orbital region was ecchymotic. He had walked to his room, had no recollection of his accident, and recognized no one that evening but his physician.

During the ensuing six weeks he had no physical symptoms. His temperature was habitually 99° F. and his pulse 72. He had marked mental disorder, a loss of all memory of his accident, and delusions, which were usually of locality, and were absolutely continuous. He had several melancholic attacks, in which he cried and wrung his hands—at one time on account of his fancied absence from home, and at another from apprehensions of disease of various organs or of pecuniary difficulties. He was always coherent; his attention could be fixed; his memory of things which happened before he was hurt remained unimpaired; and his logical processes were always correct, even when his premise was a delusion.

At the end of six weeks loss of fecal control, which had been for a few days occasional, became constant, and for a year or more was not regained; urinary control was retained. At or about this time he awoke one night screaming with pain, which seemed to be in the left frontal region; pulse and temperature were normal. A little later he had a paroxysm of active delirium, which continued for ten days, but still with normal pulse and temperature; and after this his mental condition somewhat deteriorated. His delusions became more varied; he had restless and sleepless nights; his loss of memory of recent events became pronounced, and he was at times incoherent.

At the end of the year he regained fecal control for about three days out of seven.

At the close of the second year no essential change in his condition had occurred. He was in fairly good physical health. His delusions were numerous. His primary delusion that he was away from home never varied, and he was most ingenious in explaining his surroundings upon the basis of its reality, even to the extent that the street in which he lived was duplicated, and that his neighbor's house, which he recognized, had been moved, and that the furnishings of his room had been reproduced in whatever locality he fancied it was in. His other delusions were largely connected with people long since dead, who were still living in his mind. His memory of recent events became entirely lost. His speech was incoherent, hesitating, and indistinct. He had a childish terror of the sound of a wagon, not only when he was in the street, but when in the house he heard it through the windows, though he had been a horseman all his life. His injury had been the result of a collision from behind with a runaway team, and though he never had any recollection of the accident itself, the terrifying impression made upon his mind by the sounds of the wagon and the horses coming upon him had survived. He still recognized his friends, enjoyed their society, and conversed rationally, aside from the delusions.

In the fourth month of the third year after his injury was received he died without the development of new symptoms. His strength failed, and loss of consciousness shortly preceded death.

At no time in the progress of his case was the patient lethargic, apathetic, aphasic, or paretic. His mind was always alert, and after the initial stage he always recognized people, enjoyed conversation, and even maintained a sense of humor.

Necropsy. There was a moderate amount of subarachnoid serous effusion. The brain was much atrophied and hardened in its anterior portion, comprising the frontal and anterior half of the temporal lobes.

The results of an examination of the brain and membranes made by Dr. Harlow Brooks is appended.

"The entire dura mater is thickened; the thickening is much more pronounced over the frontal regions, especially the left, where a distinct internal false membrane can be separated from the dura proper. This membrane has an average thickness of 0.5 mm., but in places over the frontal region is as much as 1 to 1.2 mm. thick. It contains numerous congested vessels and spots of recent capillary hemorrhage. There are also many brownish patches over the surface, indicating where previous hemorrhages have taken place. Numerous fine, calcareous particles can be felt in its structure.

"Microscopically, the dura mater proper shows fibrous thickening, and many of the fibres show calcareous degeneration. Its vessels are greatly thickened, and the overgrowth is greatest in the adventitia, though the subendothelial connective tissue is also hyperplastic; calcification of the intima is manifest in many places.

"The subdural membrane is made up of a dense mesh of connective tissue fibrils, many of which are calcified. Bloodvessels, both old and newly formed, are numerous, and there are a great many areas of hemorrhage, some recent, some old, but all apparently of capillary origin. In places the tissues are filled with pigment, no doubt formed by the breaking down and absorption of blood extravasation. The cytoplasm of leucocytes and connective-tissue cells found in the locality of these patches is charged with this pigment.

"Although this membrane is of considerable age, the presence of new capillaries and of recent hemorrhage proves that the membrane is still increasing in thickness.

"The arachnoid membrane is thickened, but its external surface is still almost entirely covered in by endothelium. No adhesions exist between the subdural membrane and the arachnoid.

"The subarachnoid endothelium is incomplete, and the connective-tissue columns which unite the arachnoid to the pia mater are thickened. As a whole, the pia mater is proportionately much more thickened than the arachnoid membrane, due to the perivascular connective-tissue hyperplasia and to the actual hypertrophy of the connective-tissue elements of the vessel walls. In addition there is a formation of new capillaries and other evidences of active connective-tissue hyperplasia.

"The bloodvessels of all the membranes are universally injected; the arteries and capillaries fully as much so as the veins.

"On gross examination the atrophied portions of the brain have the appearance of being pressure-atrophies of rather slow formation. The pia mater is adherent to these spots, and on its removal small particles of the cortex adhere. Atrophy is present in both frontal lobes, but is much more marked in the left; yet the convolutions even here can be distinctly traced, though small and of very irregular volume as compared with the corresponding fellow of the right side.

"The bloodvessels throughout the brain, both in the atrophied and in the macroscopically normal portions, show a very marked connec-

tive-tissue thickening, most extreme in the adventitia, but also shown in the subendothelial coat.

"Sections of the atrophied convolutions from the left frontal lobe show the number of capillaries increased; some are apparently of recent formation, and small blood extravasations have taken place through their imperfectly formed walls. This last condition is not general, but is found in small areas only. The neuroglia in these patches is more than usually cellular.

"The outer layer of the cortex of the atrophied convolutions shows a good many spaces of tissue rarefaction very like that which is seen in the molecular layer where great œdema of the membranes is present.

"In the layer of small pyramidal cells the same condition persists, though in less degree. There is a diminution in number of the ganglion cells of this layer. A few of those present show various stages of actual morphological disintegration, while many show both cytoplasmic and nuclear degeneration.

"In the layer of large pyramidal cells changes are not so marked. Disintegration of the ganglion cells is not evident, though some are shrunken and filled with coarse brown pigment. Sections stained with Nissl blue show that most of the cells of this layer have a normal arrangement and staining reaction of the chromophilic plaques; the shrunken cells, however, show cytoplasmic degeneration in various degrees.

"The white matter beneath the atrophied convolutions shows the vascular changes noted above, and also an apparent increase of neuroglia cells, most evident in the immediate proximity of the capillaries or arterioles.

"The basal ganglia and the deep tracts show no evident changes aside from the general vascular lesions; but those portions of the basal nuclei which jut on the ventricles show a maceration of their superficial layers, with erosion of the epithelial covering, probably due to maceration and pressure from the distended ventricles.

"The vascular changes are alike in all the portions of the brain examined.

"The ganglion cell degenerations are found only in the atrophied cortex. The sections examined were from the anterior tip of the left temporosphenoidal lobe and from the left frontal lobe at the angle of demarcation.

"Sections from the left parietal cortex show an apparently normal condition, except for the general vascular changes.

"Doubtless some of the ganglion cell changes are partly post-mortem; but it is beyond question that the larger parts are true degenerations, due to disease processes.

"There is no regularity in the type of cytoplasmic degenerations found in the various sections; perhaps the most frequent alteration is a granular disintegration of the chromophilic bodies, with more or less advanced chromotolysis; cytoplasmic and nuclear chromophilia are notably absent.

"Undoubtedly one of the primary lesions has been a subdural hemorrhage of considerable extent, probably sufficient to cause by direct pressure the atrophy of the frontal and temporosphenoidal lobes. The subdural membrane was formed in the absorption and removal of the clot, and as is usual in membranes of this origin the newly and imper-

fectly formed capillaries were subject to frequent ruptures with resulting hemorrhages.

"I believe that the general arterio-sclerosis, both in the vessels of the membranes and of the cerebral tissues, is a condition independent of the pachymeningitis hæmorrhagica, unless, indeed, it be somewhat of an antecedent, rendering the meningeal vessels more easily ruptured by the traumatism.

"It seems to me that the chronic inflammation of the arachnoid and pial membranes is due to two causes—the presence and pressure of the hemorrhagic exudate, and the general arterial disease.

"The remarkable atrophy of the anterior portions of the brain is, of course, to some extent a pressure atrophy, and the formation of the new capillaries in the affected areas is probably evidence of the method of removal of the compressed tissue. While no doubt the pressure and consequent atrophy had something to do with the destruction of the ganglion cells, it seems more likely that the primary injury produced by the contusion, probably associated with marked intracerebral vascular disturbances, were the most active agents. That there were at the time of injury lesions in the brain tissue is, I think, supported by the proliferation of the subcortical capillaries, and the imperfect nature of these channels is shown by the presence of the minute blood extravasations about them."

In this case the progress of the symptoms can be traced in the light of the post-mortem disclosures. In the first stage they were those of the primary lesion—a general contusion of the brain and the membranes, practically limited to its anterior portion, and more particularly to the left frontal lobe.

In the second stage there were chronic meningeal inflammation, organization of the blood effused in the pial hemorrhage, and a cirrhotic process in the contused portion of the cerebral tissue. The symptoms were still almost exclusively mental, as they had been from the time he reached his own doorstep after the accident. As the structural alterations became more profound, mental aberration became more pronounced, and mental decadence progressively increased.

The loss of rectal while vesical control was retained is not readily explained. It was not due to impairment of the mental faculties, because it occurred while the mind retained its vigor and still controlled the analogous excretory functions, and was in some degree repaired at a later period when the mind had become weakened. There was no loss of consciousness nor a general paralytic condition to which it could be attributed. It may be possible that the rectal and vesical sphincters has each a special and independent centre of control.

CASE XI. Laceration and contusion of both frontal lobes.—The patient, who had fallen a distance of from fifteen to twenty feet, was found by the ambulance surgeon in a stupid and irrational condition and in shock. On admission to the hospital he muttered incoherently, answered simple questions, but sometimes irrelevantly, and had nearly normal pulse, temperature, and pupils. During the night he became

delirious, and the following day was more stupid than before, though he could still answer some questions; urinary control was lost, and there was muscular tremor of the right extremities. On the third day he was wildly delirious and had spasmodic movements of the right lower extremity, and exaggerated reflexes. Afterward delirium became less active, spasmodic movements ceased, and both urinary and fecal control was lacking. From the eleventh to the fifteenth days he was semi-conscious and was easily aroused, but would make no response to questions. There was general muscular rigidity, and the pupils were moderately dilated and irresponsive to light. On the seventeenth day he became entirely unconscious, and on the eighteenth day his extremities became relaxed and his neck and arms rigid. His temperature rose to 104° , and he died.

Necropsy. No cranial fracture; large subarachnoid serous effusion; slight arachnoid opacity; brain substance oedematous; left lateral ventricle distended with serum; laceration of left prefrontal lobe in sulcus between first and second frontal convolutions, and of right prefrontal lobe at nearly corresponding point, each laceration about one-half inch in diameter and extending to base of cortex; several minute contusions of inferior surface of both frontal and of both temporal lobes, and a deep depression in third right frontal convolution.

CASE XII. *Laceration of both frontal lobes.*—The patient, while intoxicated, fell upon an elevated railway stairway, striking upon his head, and was found by the ambulance surgeon in a condition of stupor; and when aroused was incoherent; no history or indication of chronic alcoholism. On admission to the hospital he was bleeding from the right nostril, was still stupid and incoherent, and had normal pulse, temperature, and pupils. He remained in a condition of stupor, though conscious, until just before his death, on the fourth day. Fecal and urinary control was lost; rigidity of the left side of the neck and right side of the body was well marked; dysphagia was progressive; the corneal reflexes were nearly abolished, and temperature gradually rose to 105.2° F.

Necropsy. No fracture; no epidural hemorrhage; thin sheet of cortical hemorrhage covering the whole superior lateral and anterior portions of the brain and the tentorium; cortical hemorrhage in all the basic fossæ, and large in amount in the left anterior and in the right middle and posterior fossæ; laceration of under surface of the left frontal lobe in its whole middle two-fourths, involving the first, second, and third orbital convolutions, and extending deeply into the subcortex; similar cortical and subcortical laceration of inferior surface of right frontal lobe, but somewhat smaller in extent; both these lacerations were filled with clot, which was continuous with the hemorrhagic effusions described. Another laceration extended quite across the inferior surface of the left temporal lobe, and another confined to the cortex involved much of the inferior surface of the right cerebellum. The last was not an apparent source of hemorrhage.

CASE XIII. *Abscess of both frontal lobes from pistol-shot; laceration.*—Bullet of 0.32 calibre entered the head one inch behind and one-half inch above the right external angular process of the frontal bone.

Primary symptoms. Hemorrhage from right nostril; blood and brain matter escaping from wound. Patient conscious, but apathetic; pupils contracted and slightly reacting to light; respiration slow and

shallow. Urinary control retained. Temperature 98° F., pulse 76. Fragments of the internal table of bone were removed, and wound healed without suppuration.

Later symptoms. None, aside from an abnormal mental condition until the twenty-fourth day, when the patient became unconscious. He was always apathetic, and when sufficiently roused to make answer to questions was incoherent; and his power of comprehension was deficient. Vesical and rectal control were lost with consciousness, and not regained. At about the same time the lower extremities became rigid, and their reflexes were exaggerated.

Death occurred on the thirty-first day. The temperature until the eighteenth day ranged, with two or three exceptions, from 98° to 100° F., and afterward from 102° to 106.2° F., the pulse was ordinarily from 40 to 60.

Necropsy. There was no epidural hemorrhage; the dura mater was adherent, and there was a diffused subarachnoid seropurulent effusion. The crista Galli was broken off. Upon section the anterior half of the left frontal and anterior third of the right frontal lobe were each found to contain a large area of thick, creamy material, including fragments of bone and lead. A cylindrical area of softened material one-half inch in diameter extended from the disintegrated portion of the left frontal lobe through the left lateral ventricle to the postero-inferior part of the left occipital lobe, at the termination of which the bullet was lodged, one-half inch below the surface. Small particles of bone and lead and a few grains of powder were found in the left frontal lobe outside the area of disintegration. The frontal sinuses communicated with the cranial cavity and contained a seropurulent fluid.

CASE XIV. *Laceration of both frontal lobes.*—Patient found in an unconscious condition. On admission to the hospital there was orbital ecchymosis and subconjunctival hemorrhage on the left side, moderately dilated pupils, and loss of urinary control. He was delirious through the night, and next morning had left hemiplegia. He could answer simple questions, but his attention could not be fixed for a longer time. He recognized his wife with indifference. There was incomplete left hemiplegia, normal pulse and respiration, and slightly elevated temperature. During the first ten days his symptoms were variable. Movement was restored to the left side, but the left arm became and continued rigid; patellar reflexes were lost; rectal and vesical control were regained and lost again; the patient's mental condition was at times apparently normal, at others he was delirious, and at others still he had mild delusions; he was always restless, and his right arm continually moved aimlessly; the pupils were usually, not always, sluggish, and sometimes unsymmetrical, and then the right was the larger. From the tenth to the twenty-sixth day, when he died, he was sometimes delirious, but usually apathetic and without interest in his surroundings. He had many delusions, but often reasoned logically from his false premises. His general condition remained unchanged.

Necropsy. Indirect fracture of left orbital plate posteriorly; no epidural hemorrhage; four areas of meningeal contusion, one over each frontal lobe, and one over each parieto-occipital junction, each about two by two and one-half inches in diameter, with opacity of arachnoid membrane and subarachnoid serohemorrhagic effusion; subcortical excavation of whole right and greater part of left frontal lobe filled

with disintegrated clot and extending into motor area on right side; some blood in right lateral ventricles; excessive general hyperæmia.

CASE XV. *Laceration of the right frontal lobe.*—Patient knocked down in a street altercation; later, walked with assistance to police station. He there gave the ambulance surgeon his name and residence, and had sufficient intelligence to give a wrong address. His mental condition was apparently normal. He became comatose when he reached the hospital, and remained so until his death on the fourth day.

Necropsy. Acute arachnitis over right hemisphere. Laceration of inferior surface of right frontal lobe posteriorly and involving the corpus striatum and optic thalamus. Comminuted fracture of right orbital plate and of lesser wing of sphenoid bone.

CASE XVI. *Abscess of the right frontal lobe.*—Patient kicked by a horse; when admitted to hospital five hours later was still unconscious. Temperature 97° F., pulse 70, respiration 24. Hemorrhage from both ears. Wound over right eye, one on left side of head, and another between the eyes; the latter involving loss of greater part of nasal bones and of some of the ethmoidal cells; pupils slightly dilated and fixed; loss of vesical and rectal control; spasmodic contractions of various groups of muscles.

Second day. Patient conscious and rational; temperature 98.2° F., pulse 120.

Third day. Patient restless; mental condition normal; said he had no pain; asked intelligently for whatever he required. Temperature rose from 98.5° to 102° F., and pulse from 95 to 122.

Fourth day. Patient restless; had delusions and talked much to himself, but answered questions rationally. Temperature 104.2° F.

Fifth day. Stupor.

Sixth day. Coma and death. Temperature 107.6°; post-mortem, 108.6° F.

Necropsy. Fracture of both orbital plates; ethmoid cells disintegrated; no opening into cranial cavity; acute arachnitis, limited to right frontal region; subarachnoid purulent effusion communicating from the vertex with a cerebral abscess situated immediately below the superior cortex and occupying the inner lateral half of the lobe; cerebral tissue about the anterior margin of the abscess blackened and necrotic; brain generally cedematous.

The sudden elevation of temperature and occurrence of mental disorder in this case on the fourth day marked the beginning of meningeal inflammation. These phenomena I have noted elsewhere¹ as diagnostic of the invasion of this process in the course of traumatic injuries. The mental condition was unaffected while the effects of injury were confined to the right frontal lobe.

CASE XVII. *Laceration of the right frontal lobe.*—Pistol-shot wound in right temple. Patient walked five miles to hospital, and on admission was conscious, coherent, and intelligent. No aphasia, no paralysis, no affection of special senses, and no loss of control of sphincters.

On the second day condition unchanged; wound enlarged and frag-

ments of bone removed from cerebral cortex. Examination was made with view to counter trephination, but it was thought that the ball had been deflected by falx cerebri, and project of operation was abandoned.

On the third day the patient was a little more difficult to rouse, but the facies was intelligent, and he responded rationally to questions.

On the fourth day he became unconscious, and died.

Necropsy. Bullet passed through central part of right frontal lobe to median line, and was then deflected by falx cerebri; passed through lateral ventricle, and was lodged in the inferior part of the left occipital lobe, just below the cortex. There was much cortical hemorrhage over the right, and a large subarachnoid serous effusion over the left hemisphere.

CASE XVIII. Laceration of the right frontal lobe.—The patient, a boy, aged six years, was struck upon the head by a piece of scantling thrown from the roof of a five-story building. One hour later he was still unconscious and was restless and hyperæsthetic. The skin was warm; the pulse 64, full and regular, and the respiration normal; the pupils were moderately dilated, symmetrical and responsive to light, and the left side was hemiplegic. A pulsating tumor occupied the right fronto-parietal region, through which could be felt a depression of bone. Immediate incision was followed by the escape of disintegrated brain substance, mingled with blood and coagula. The posterior part of the frontal, the anterior half of the parietal, and the upper part of the squamous portion of the temporal bone were broken into fragments, which were greatly depressed and were elevated one by one. Only one bit of bone, not larger than a finger nail, was removed. The flow of blood during this procedure was profuse, and the last fragment elevated was of small size and closed a wound of the superior longitudinal sinus.

In the meantime the patient had become apparently moribund and was resuscitated with some difficulty. A large portion of the right frontal and a small portion of the right parietal and temporal lobes had been removed. On the following day the temperature was 101° F., and the pulse 76; the pupils were moderately dilated, consciousness was regained, and the mental condition was normal, with incomplete left hemiplegia and facial paralysis. A fungus cerebri formed which was not the source of any considerable trouble. The wound was healed at the end of six months, and the paralysis lasted six months longer. During this time his mental condition was absolutely normal; his disposition was cheerful; and his convalescence was unmarked by emotional disturbances.

It is the testimony of his family and their acquaintances, confirmed by the family physician, that for eight years his mental and bodily health was unimpaired. In his fourteenth year he rather suddenly became epileptic, and since then, eighteen years, the paroxysms have been frequent and severe, excited by any mental exertion or excitement. At the present time his memory is good and no mental decadence is apparent. He is, however, irritable and excitable.

RÉSUMÉ. Case I. Symptoms: Almost exclusively mental. Primarily: aphasic disorders and slowness of apprehension. Later: apathy, incoherence, mental aberration, and progressive mental decadence; loss of memory; loss of power of comprehension in general and especially of

comprehending or speaking familiar languages; continued sensory and motor aphasia; and loss of urinary control from enfeeblement of will.

Lesions: Confined to left frontal lobe; tumor excavating and displacing greater part of its white substance, with destruction of much of its cortex; right frontal lobe merely indented; general cortical tissue of brain normal.

Case II. Symptoms: Apparent abolition of all mental action, while consciousness was retained.

Lesions: Laceration of anterior third of first and whole of second and third left frontal convolutions; superficial contusion of right parietal and occipital lobes. Secondly: acute arachnitis.

Case III. Symptoms: Primarily, temporary motor aphasia; followed by some mental deficiency noted by the subject; later, slowness of comprehension, deficiency of power of attention, apathy, and loss of memory; change of character, patient melancholic and emotional.

Lesions: Gunshot fracture of left frontal and parietal bones; subsequent meningeal cyst; softening of posterior portion of left first, second, and third frontal convolutions.

Case IV. Symptoms: Stupidity, incoherence, and disorders of mentation.

Lesions: Deep laceration of anterior third of left frontal lobe involving first, second, and third convolutions.

Case V. Symptoms: mental condition irrational, but not delirious; later, delirium.

Lesions: Laceration of whole superior and lateral aspects of left frontal and temporal lobes.

Case VI. Symptoms: Apathy, refusal to answer questions, delusions, stupor, incoherence, and deficient comprehension.

Lesions: Deep laceration of first, second, and third left frontal convolutions in anterior third; superficial laceration of temporal lobes; general contusion.

Case VII. Symptoms: Stupor, irrational condition, and inability to comprehend surroundings; apathy and delusions.

Lesions: Atrophy of left frontal lobe, and general oedema.

Case VIII. Symptoms: Incoherence; loss of memory; slowness of comprehension; apathy, and delusions.

Lesions: Laceration of middle portion of first and second left frontal convolutions, extending through cortex; excessive oedema of left hemisphere.

Case IX. Symptoms: Primarily; muttering and laughing delirium, followed by an irrational condition, and lack of comprehension. Later, emotional condition, and speech in unmeaning words; loss of his native language, aphasia, and mental enfeeblement.

Lesions : Laceration and disintegration of posterior half of left frontal lobe and upper part of temporal lobe. Compound fracture of left frontal and parietal bones.

Case X. Symptoms : Primarily, a delusion of locality which became permanent ; loss of memory of recent events ; further delusions, melancholia, and mental decadence.

Lesions : General atrophy of the anterior half of the cerebral hemisphere, most marked upon the left side, the result of pial and cerebral contusion.

Case XI. Symptoms : Stupidity, incoherence, and refusal to answer questions.

Lesions : Laceration and contusion of both frontal lobes involving first and second convolutions ; general contusion.

Case XII. Symptoms : Stupidity and incoherence.

Lesions : Laceration involving middle two-fourths of first, second, and third orbital convolutions, and extending deeply into subcortex. Similar laceration of right frontal lobe somewhat smaller in extent. Superficial lacerations of inferior surface of right temporal and of right cerebellar lobes ; cortical hemorrhages.

Case XIII. Symptoms : Apathy ; incoherence and deficient power of comprehension.

Lesions : Abscess of anterior half of left frontal, and of anterior third of right frontal lobe ; softening in course of bullet track through left lateral ventricle into occipital lobe.

Case XIV. Symptoms : Deficient power of attention and apprehension. Subsequent variable mental condition ; sometimes normal, sometimes delirious, usually apathetic, with mild delusions.

Lesions : Subcortical laceration of whole right and of greater part of left frontal lobe ; limited meningeal contusions, general cerebral contusion.

Case XV. Symptoms : Mental condition normal till coma ensued on arrival at hospital ; answered questions intelligently.

Lesions : Laceration of inferior surface of right frontal lobe, extending to right corpus striatum and optic thalamus ; acute arachnitis over right hemisphere.

Case XVI. Mental condition normal till fourth day, when acute arachnitis supervened, with characteristic elevation of temperature and mental disorder.

Lesions : Acute arachnitis, limited to right frontal region, and communicating with a cerebral abscess situated immediately below the cerebral cortex.

Case XVII. Symptoms : Pistol shot wound ; patient walked five miles to hospital ; conscious, coherent, and intelligent ; no mental disorder before final coma on the fourth day.

Lesions: Bullet passed through the middle of the right frontal lobe laterally and then deflected by falx cerebri through left lateral ventricle into left occipital lobe; cortical hemorrhage and limited meningeal contusion.

Case XVIII. Symptoms: No mental disorder at time of injury or for eight years afterward. Patient then became epileptic. At present time he is irritable and excitable, as is usual in cases of long-established epileptic conditions.

Lesions: Disintegration and removal of large part of right frontal and of small portion of right parietal and temporal lobes.

There have been several cases reported of equally extensive and even greater destruction of the right frontal lobe without disturbance of the mental faculties. There is one of Tavignot, quoted by Pitres,¹ in which, by the kick of a horse, this lobe was reduced in its whole extent to a mere mass of granular detritus, though intelligence was preserved and the patient responded to questions with precision. Bramwell² exhibited a tumor of the right frontal lobe in a case in which its removal had been followed by a hernia cerebri as large as the head itself, twenty-three inches in circumference by five and one-half inches in depth, destroying almost the whole lobe. The patient lived three years, and "the memory and mental condition remained good." An item in the *New York Medical Record*, 1898, vol. ii., p. 104, calls attention to a case reported by Porta, in which, by an accident, a man lost the whole of his right cerebral hemisphere. He recollected being taken to the hospital; the wound closed in eighteen months, and he recovered with left hemiplegia. "His intellectual functions are said to have been unimpaired." Still another case of serious right frontal lesion without intellectual impairment, one of traumatic abscess, is recorded in Senn's *Principles of Surgery*, 1890, p. 273, and is thus stated: "The patient filled in a creditable manner a responsible and important government position for thirty years, and died from another cause. The necropsy showed an abscess cavity, the size of an orange, located in the anterior right lobe of the brain, which communicated with the external surface through a fistulous opening in the skull."

I shall add to this series of cases two instances of left frontal lesion reported by other observers, the records of which are so full in detail and so accurate in statement as to give them unusual value.

They are: First, the well-known "Crowbar" case, reported by Harlow.³ The subject, a young man in robust health, was wounded September 18, 1848, by the premature explosion of a blast. A tamping-iron, 3 feet 7 inches in length, 1½ inch in diameter, and weighing

¹ *Lesions du Cent. Ovale*, Paris, 1877.

² *Lancet*, 1900, vol. ii. p. 1432.

³ *Publications of Massachusetts Medical Society*, 1868, vol. ii. p. 321.

13½ pounds, was driven quite through his head, entering through the left superior maxilla and emerging in the median line through the left frontal bone, just below the coronal suture. He was able to speak a few moments later, was carried for some distance in a sitting position with slight assistance, and was then able to walk up stairs. He was at first rational, but two days later became delirious with lucid intervals, and for the week following was again in normal mental condition.

The sight of the left eye was lost. *Hernia cerebri* formed, and beneath it an abscess which, when opened by excision of the cerebral extrusion, discharged about eight ounces of pus.

His mental condition as noted from time to time was thus set down.

October 6th (he was injured *September 18th*). "Appears demented or in a state of mental hebetude."

15th. "Intellectual manifestations feeble; exceedingly childish and capricious, but with a will as indomitable as ever."

20th. "Mind clearer, but still very childish."

April, 1849. "Left ptosis; can only depress and abduct the *globus oculi*; no other muscular impairment, physical health good; applied for employment, but his employers considered the change in his mind so marked that they could not give him his place again. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation which are no sooner arranged than abandoned in turn for others. A child in his intellectual manifestations, he has the animal passions of a strong man. Previous to this injury he possessed a well-balanced mind, which was radically changed."

In 1860 he had a long illness, "fell suddenly in a fit while at dinner," and fits continued till his death, May 21, 1861, twelve years and more after his injury was received.

No post-mortem examination was made, but the skull was afterward exhumed. From data afforded by Dr. Harlow's examination of the skull, and from an examination of the patient made by Dr. Bigelow two years after the accident, Ferrier was enabled to determine that "the whole lesion was situated anterior to the coronal suture and to the left of the median line. The iron comminuted and carried away the entire left lesser wing and one-half the greater wing of the sphenoid bone, and a large portion of the left orbital plate. No doubt the whole (cerebral) track is included within that region of the brain which I have described as the prefrontal. The only other region which the bar could have injured is the tip of the temporal lobe and the outer root of the olfactory bulb."

Second: A case reported to the Italian Psychiatric Society, October 18, 1896, by Obici and Tambroni.¹

Symptoms: Mental enfeeblement, characterized by loss of power of abstract connection, default in the power of psychical orientation, irritability; enfeeblement of character and of altruistic sentiment; tendency to suicide.

Seven months after commencement of the disease convulsions and paresis became manifest, and apoplexy supervened.

Necropsy. "Glioma occupying a large part of the left prefrontal lobe; no excess of ventricular fluid; no intracranial pressure."

Three generalizations were made from an analysis of my first series of cases. Some slight changes in the first of which, due to added experience or in the way of explanation, are necessary.

1. In every instance but two in which consciousness was retained or regained and the mental faculties were not perverted by general delirium, laceration involving the left frontal lobe was attended by default of intellectual control, and the lesion was usually, if not always, of the prefrontal region and implicated either its superior or inferior surface. Subcortical disintegration, or deep or extensive laceration of the cortex, was specially characterized by abrogation of mental power, and superficial laceration by aberration in its manifestations.

In one of the two exceptional instances referred to, in which laceration of the left frontal lobes was not attended by default of intellectual control, the supervention of final coma within two hours was so nearly immediate as to practically withdraw it from the class of cases under consideration.

The other case seems to have been made exceptional by reason of the mental condition having been regarded as normal on the one day only in which the patient emerged from a general condition of stupor.

In the first series of cases the inferior surface of the lobe was the more frequently implicated, but in the second it was the superior; and the abrogation of mental power seemed to be proportionate to the extent of the lesion rather than to its situation in the prefrontal region. The original proposition has been modified therefore in regard to these points. The other propositions remain unchanged.

2. In every instance in which laceration was confined to the right lobe the mental faculties were unaffected, except as they were obscured by stupor or delirium occasioned by coincident general lesion.

3. Compression or contusion of the left lobe only exceptionally produced specific intellectual disturbance.

This generalization is based upon an examination of an entire series of 295 cases in which the history was supplemented by necropsy.

¹ British Journal of Mental Science, 1897, p. 599.

It may be questioned, however, whether in recovering cases the lesion may not often be contusion, either limited to, or especially pronounced in, the left frontal lobe. This was undoubtedly the fact in Case I., and is not improbable in recovering cases in which delusions and other mental disturbances replace stupor, apathy, or other indications of simple mental decadence.

The large number of cases cited, with the analysis of their symptoms and lesions, are probably sufficient in themselves to form a basis for conclusions. They represent the personal observation and record of 800 cases of intracranial traumatism of which more than 300 were subjected to either operative or post-mortem inspection. Excluding those cases in which death had been preceded by primary and permanent unconsciousness, they were all germane to the present inquiry as showing either the presence of left frontal lesion where mental symptoms had been noted, or the absence of such symptoms where the lesion was situated in any other region of the brain. In many excluded, because unverified, cases, the coincidence of predominating symptoms of a mental disorder, with external indications pointing to a left frontal lesion, afforded at least a corroboration of the inferences which the post-mortem examination of the other cases proved to be well founded.

There are a still larger number of cases, unverified by necropsy, and therefore debarred in evidence, which are scarcely less convincing than those in which the essential condition of proof is obtainable. A number of such cases were included in the series of condensed histories appended to my work on *Brain Injuries*, to which reference has been made. Some well-marked instances were those numbered: CCXXXVI.; CCXXXIX.; CCXL.; CCXLI.; CCXLV.; CCXLVIII.; CCXLIX.; CCLIV.; CCLXXII.; CCLXXVIII.; CCLXXXII.; CCLXXXIII.; CCLXXXIV.; CCXC.; CCXCI.; CCC. The recovering cases, in fact, often present the most characteristic symptoms. In fatal cases the immediate effects of the concurrent general contusion may continue in some degree to the end, and during their continuance it may be difficult if not impossible to clearly recognize the evidences of the distinctive frontal lesion. If the cerebral circulation becomes readjusted and the impairment of consciousness or the delirium which the general vascular disturbance has occasioned passes away, there is no longer difficulty in referring stupor, apathy, delusions, or other indications of mental disorder or default to the lesion of specific centres of control to which it is properly attributable. It is to be noted that mental aberration no less than mental default is the result of lack of control, such symptoms "indicating activity on the lower level of evolution" (Jackson) and its inhibition on the higher level.

BRAIN ABSCESS IN TYPHOID FEVER DUE TO BACILLUS TYPHOSUS.

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DEFINITE infectious lesions of the brain or of its membranes would seem to belong to the more rare complications of typhoid fever. In his careful review of the literature up to 1898, W. W. Keen¹ mentions fifteen cases of meningitis and four of abscess of the brain as complications of typhoid fever or as sequelæ of the disease. In the cases of meningitis the bacillus typhosus was found in all cases, and in pure culture in twelve, while in none of the cases of abscess was any bacteriological examination made. In an extended search through the subsequent literature I have found reports of but five similar cases definitely established by autopsy or otherwise, four of them being meningitis and one brain abscess. Troisier and Sicard² report a case in which a fibropurulent exudate was found at the base of the brain, containing the bacillus typhosus in pure culture, symptoms of meningitis having appeared four days before death. Jemma³ reports a case of cerebro-spinal meningitis in which the exudate was drawn off by lumbar puncture, and found to contain bacillus typhosus, no mention being made of other organisms. Tarchetti⁴ mentions a case of meningitis in which the infectious agent was found to be bacillus typhosus. Fisher⁵ describes a fatal case of meningitis in typhoid, bacillus typhosus being found in the serous exudate. He does not state whether other organisms were present. Brown⁶ reports a case of abscess of the brain in typhoid, with recovery after operation. The pus contained staphylococcus pyogenes aureus in pure culture. We have thus nineteen cases of meningitis, in all of which bacillus typhosus was present, in the great majority in pure culture; and five cases of brain abscess, in none of which it was found. Most of the severe cephalic symptoms which have been noticed in the course of the disease have been found to be due or attributed to thrombosis of one of the cerebral arteries, of which Osler⁷ has recently described four cases and Debove⁸ one. Such being the status of affairs, the following case will be of interest as the only instance of brain abscess in which bacillus typhosus has been found to be the etiological factor:

Miss M. B., aged forty-nine years; occupation, teacher. Attending physician, Dr. W. G. Willard. Physician called April 7, 1901. Patient states that this is the first illness in years. During the last weeks of the school term just closed she has felt unusual fatigue and malaise. In three days immediately preceding had some chilly sensa-

tions, fever, and headache. On examination, temperature was 103° F., pulse 90. The tongue was moist, with a moderately heavy, white coating. There was no abdominal tenderness or distention; no rose spots were present, nor had she had epistaxis.

April 2d, probably the eighth day of the disease, a few rose spots appeared on the abdomen and chest. On this day she was removed to the Presbyterian Hospital. The course of the disease during the second week was even and all symptoms moderate. The temperature ranged from 101° F., A. M., to 103.5° F. or 104° F., P. M. She had an annoying dry cough, and was occasionally nauseated after taking milk foods. The stools were normal. Urinalysis showed a trace of albumin, but no casts. Diazo reaction questionable. During the third week the temperature ranged from 100° F., A. M., to 103° F., P. M. Her main complaint was of nausea and eructations, especially when milk foods were given. At the beginning of this week the agglutination test gave a probable positive result. Urinalysis showed a normal specific gravity, with absence of albumin and casts. During the fourth week the temperature ranged from 98.6° F., A. M., to 103.4° F., P. M.; pulse, 90 to 100. Toward the end of this week she complained of nervousness, and had a great dread of being left without an attendant, even for a moment.

The fifth week showed a temperature markedly remittent and sometimes intermittent, ranging from 98.4° F., A. M., to 101.4° F., P. M.; pulse, 84 to 94. Patient was still nervous, and toward the end of this week for the first time complained of slight ache on the back of the head. The following day she had some neuralgic pains on the left side of the head and neck. Sleep was fairly good. May 6th, the thirty-third day of the disease, temperature, 98.4° F.; pulse, 92. At noon she slept. On awakening seemed lost and talked incoherently; was nauseated and vomited after taking liquid nourishment; pale, and skin cold. At 2.30 P. M., a convulsion, epileptiform in character. Morphine, $\frac{1}{4}$ grain, given by order of house physician. At 3.20 P. M., a second convulsion, and shortly before 4 P. M., a third; the clonic spasms lasting five minutes. These were not completely unilateral, but very much more marked on the right side than on the left. The pupils were equally contracted; the tip of the tongue slightly abraded. At 4 P. M. the pulse was 152 and very weak; temperature, 101.2° F. At this time a thorough physical examination was made, with mainly negative results; no inequality of pupils, no paralysis; no sign of middle-ear disease; no phlebitis. There was at this time a marked mitral blowing systolic murmur, which had not been present previously, and which disappeared in twenty-four hours, as soon as the heart regained its tone.

By evening she seemed to understand what was said to her, but her speech was disconnected. At 8 P. M. temperature was 99° F. (rectal); pulse, 124. Urinalysis showed considerable albumin and numerous hyaline and granular casts. These disappeared in a few days. During the week immediately succeeding the convulsions the temperature ranged from 98.6° F., A. M., to 100.5° F., P. M. The mind, though clearer, was still much confused; and at times she complained of frontal headache, which was relieved by the ice-bag, which had been employed most of the time since the convulsions. At this time—end of the sixth week—there was marked amnesic aphasia; ice-cream was termed cotton-batting, and her nurses were called seamstresses, etc.

During the seventh week the temperature did not rise above 99.8° F. The pulse varied from 86 to 100. Sleep at night was good, and the urine was free from albumin and casts. In the earlier part of this week the patient had frequent headaches, and once told the nurse that she felt a twitching on the right side of the face; there was none visible, however. During the eighth week there was a slight evening elevation of temperature, 1°; much less headache; mental operations approaching normal; rarely at a loss for the right word; sleep sound at night. Early in the ninth week she again complained of severe headache in the forehead and vertex and back of the head. There was occasional retching.

On the night of June 1st, the fifty-ninth day, the patient slept well until 4 A. M., then awakened with pain in the back and the top of the head, nausea, and chilliness. Temperature was 99.6° F., pulse 100. At 7.30 A. M. temperature was 103.8° F., pulse 124; speech was incoherent. During the sixty-first and sixty-second days the symptoms were the same, but with a gradually deepening stupor; temperature 103° F., and Kernig's sign present.

Early in the sixty-third day—June 5th—the muscles of the right arm showed convulsive movements when the arm was held away from the body. Albumin and casts were again present in the urine. Temperature at 6 A. M. (rectal) was 104.8° F.; pulse 98; at 9 P. M., 105° F.; pulse 124. During the sixty-fourth day the temperature dropped until by evening it was 100° F.; pulse quite irregular; raised right hand to head. On the sixty-fifth day the temperature was normal at noon. Patient comprehended questions and answered intelligently; took liquid nourishment; at times raised hands to head as if in pain. On the sixty-sixth day—June 8th—tendency to slight spasm returned; patient was much weaker. Temperature at 6 A. M., 97.6° F.; pulse, 132. Tendency to spasm continued throughout forenoon; pupils equal; patient steadily growing weaker; died at noon.

Autopsy (June 8th, 3 P. M.—Dr. Crowder). *Anatomical diagnosis.* Suppurative basilar meningitis; abscess of the left temporal lobe; purulent exudate in lateral ventricles; healing typhoid ulcers in ileum; acute splenic tumor; cloudy swelling of solid viscera; moderate diffuse arterio-sclerosis; chronic interstitial nephritis; healed tuberculous nodule of left upper lobe; misplacement of uterus; fibroids of uterus; catarrhal appendicitis.

The body is that of a well-developed, fairly well-nourished woman. It is still slightly warm. There is slight rigor mortis and posterior lividity. Beneath the clavicles and above the right breast are subcutaneous ecchymoses, about the locations of punctures made by hypodermics. The abdomen is distended and tympanitic.

The intestinal coils are distended with gas, particularly the cæcum and ascending colon. The peritoneum is smooth and shiny. The abdominal cavity contains a slight amount of clear, straw-colored fluid. The foramen of Winslow is patent. There are a few adhesions along the posterior border of the spleen. The vermiform appendix is free and has a mesentery throughout its whole extent. Its superficial vessels are greatly congested.

The pleural cavities are empty and free from adhesions. The pericardial cavity contains about two ounces of a clear, straw-colored fluid. The layers of the pericardium are smooth and shining.

The left lung presents a smooth pleura throughout, and is of a pale gray color; the upper lobe is spongy and the lower soggy. Over the posterior surface of the lung, covering about two-thirds of this surface, and of a dark bluish color, is an area slightly depressed below the surrounding tissue. The lung crepitates faintly. On section the lung is grayish-pink, the dark reddish-blue area extending in for 3 to 4 mm., and a moderate amount of blood and frothy serum exudes on pressure. A small puckered scar at the middle of the anterior surface of the upper lobe covers a calcareous nodule the size of a split pea. There is a slight thickening of the pleura at the apex. Except for the calcareous nodule, the right lung corresponds in every way with the left.

The heart weighs 260 gms., and its surface is smooth and shiny. The left ventricle is in contraction; the right contains a large amount of fluid blood. The endocardium and valves are normal throughout, excepting the anterior mitral curtain, which shows slight diffuse thickening, with three circumscribed, yellowish, opaque areas, which present a smooth surface and are not visibly elevated. The myocardium is firm, its texture fine, and its color a reddish-gray, with gray areas of fibrosis toward the tips of the papillary muscles of the left side.

The pulmonary artery is normal. The thoracic and abdominal aorta, and the beginnings of the coronary arteries, show small, diffuse, slightly elevated areas of sclerosis, presenting a slightly roughened surface.

The spleen weighs 300 gms. The lower extremity shows a cylindrical process marked off from the body by a deep notch in front, and a groove extending most of the way around. It presents torn adhesions along the posterior border, but is otherwise smooth, and of a slaty-blue color. It wrinkles easily, and is soft and elastic. It cuts readily. The pulp is of a dark reddish-brown color; the Malpighian corpuscles are only partially visible; the connective tissue is not visibly increased.

The liver weighs 1650 gms. Its external surface is smooth and paler than normal. It cuts with normal resistance. The cut surface has a grayish color and an opaque, boiled appearance. The lobular markings are faintly visible. The liver substance for a few millimetres, just under the capsule of the upper surface of the right lobe, is quite distinctly yellow. The gall-bladder is normal.

The pancreas is normal.

The stomach contains a quantity of partially digested food; its mucous membrane is smooth and thrown into folds, which are removed by stretching, and is covered by a layer of tenacious mucus. Toward the cardiac extremity there are a few very small submucous ecchymoses.

In the small intestine, two inches above the cæcum, is a large Peyer's patch showing a distinct diffuse pigmentation, and presenting at its centre an ulcer 3 cm. in diameter superficially, apparently in a healing condition. Approximately two feet from the cæcal end of the ileum is another Peyer's patch showing three areas of smaller diameter, which are apparently the seats of very superficial healing ulcers. The intestines show no other gross lesions.

The vermiform appendix is 9 cm. long; it is congested in its distal extremity, and rather firm. Its lumen is obliterated for the last 2 cm., and contains a small amount of viscid material.

The suprarenals are normal.

The left kidney weighs 160 gms.; the right, 155 gms. The external

surface of the left kidney is smooth, and it cuts with normal resistance. The capsule strips reluctantly, tearing the parenchyma with it in all places, and leaving a finely granular surface. The cortico-medullary relation is about one to three. The cortical markings are very distinct, and the cortex is pale in color; the glomeruli are visible as pin-point dots. The right kidney corresponds in every way to the left.

The body of the cervix lies horizontally, extending to the left of the median line, the body of the uterus forming a sharp angle forward and to the right. The anterior and posterior surfaces of the uterus show each one pea-sized subperitoneal fibroid. The cervical and vaginal mucosae are normal; the hymen is intact. The urinary bladder is contracted, empty, and free from changes.

The scalp and skull are normal. The dura is adherent to the skull in the parieto-frontal region. There are no changes over the convexity of the brain. The base of the brain from the optic commissure backward over the pons, medulla, and cerebellum is covered with a thick, yellow, purulent exudate; and beneath the dura there is a considerable quantity of turbid, yellowish, serous fluid. The left temporal lobe is much larger in its posterior part than the right, and presents distinct evidence of fluctuation throughout most of the lobe. The exudate entirely surrounds the peduncles, and is found on the upper surface of the cerebellum. The ventricles are filled with turbid, brownish-yellow fluid, and the choroid plexus covered by exudate similar in appearance to that found at the base of the brain externally. A large cavity is found in the basilar part of the left temporal lobe, containing a bloody, thick, purulent material. There are no signs of middle-ear disease.

Histological Examination. The alveoli of the lung are slightly collapsed, and contain a few polymorphonuclear leucocytes, erythrocytes, and round cells; the bronchi are normal; the vessels are highly congested; the pleura is slightly hyperplastic.

The myocardium shows segmentation and some fragmentation, with considerable connective tissue among the muscle cells.

The spleen shows indistinct Malpighian corpuscles; there is intense congestion, with a great increase in the proportion of erythrocytes; the connective tissue shows slight hyperplasia, especially in the walls of the bloodvessels.

The liver contains numerous areas of focal necrosis; there are marked fatty change and moderate congestion.

The appendix shows a large number of leucocytic cells in the mucosa and submucosa, and a congestion of the vessels of the submucous and muscular coats.

The kidney shows capsules of the glomeruli to be thickened. The protoplasm of the tubular cells is often fragmented and the nuclei unstained; the tubules show many casts. The vessels are highly congested, and their walls show hyperplasia of the connective tissue.

The wall of the cerebral abscess shows a definite thin capsule of new connective tissue. Inside of this is a thick exudate, composed principally of polymorphonuclear leucocytes, with a few round cells, some red blood cells, and much necrotic material. Just outside of the capsule is an area of round-cell proliferation, containing small and large round cells and plasma cells, many showing karyokinetic figures. The surrounding brain substance shows some chromatolysis and some granular disintegration of the neuroglia. Paraffin sections stained by

Loeffler's methylene blue show groups of short, blunt bacilli with rounded ends, on both inside and outside of the capsule. Sections stained by Gram show no organisms.

Bacteriological Examination. Smears from the lung show a coccus which stains by Gram; from the liver and spleen a short bacillus destained by Gram; from the kidney and heart's blood no organisms. Plates from the lung contain a few yellowish, granular colonies of staphylococci, which retain Gram's stain and produce good yellowish or lemon-colored growths on agar and potato; some acid, without coagulation, in four days' milk, and liquefaction of gelatin in four days; diagnosed staphylococcus pyogenes aureus. Plates from the liver and spleen contain a fair number of colonies of bacillus coli communis and a few of an organism similar to that found in the cerebral lesions. Plates from the heart's blood and kidney show no growth.

During the autopsy, blood-serum and agar tubes were inoculated, with strict precautions, from the cerebellar exudate and from the cerebral abscess before it was opened. Smears from both show only a short bacillus with rounded ends, which destains by Gram, present in large numbers. Five inoculations on blood-serum—three from the cerebral abscess and two from the cerebellar exudate—and ten glycerin-agar plates from each, show in every instance pure cultures of a short, blunt, highly motile bacillus, with rounded ends, which destains by Gram. The morphology is entirely similar to that of bacillus typhosus. Stained by Loeffler's tannic acid method, the bacillus shows from ten to sixteen fairly long, wavy flagella, which have a peritrichal arrangement. The cultural appearances on ordinary laboratory media indicated the advisability of inoculations on differential media, which were made as follows:

Hiss' Agar-gelatin. (a) Agar, 5 gms.; gelatin, 80 gms.; beef extract, 5 gms.; NaCl, 5 gms.; glucose, 10 gms.; distilled water, 100 c.c.; titrated to 1.5 per cent. acid (phenolphthalein). Inoculations made in tubes containing about two inches of medium, by placing on the surface a small drop of a bouillon culture. At eighteen hours (37° C.) the upper inch of the medium is uniformly clouded, with small projections of growth into the unclouded portion. At thirty-six hours the whole of the tube is uniformly clouded; no gas is apparent. A similar inoculation with a stock bacillus coli communis produces in forty-eight hours (37° C.) a stronger clouding of the medium for the upper one-fourth of the tube with small gas-bubbles. (b) Agar, 10 gms.; gelatin, 25 gms.; meat extract, 5 gms.; NaCl, 5 gms.; glucose, 10 gms.; distilled water, 1000 c.c.; titrated to 2 per cent. acid (phenolphthalein). In plates inoculated from glycerin-agar cultures, thirty-six hours at 37° C., deep colonies are very small, spherical, or slightly ovoid, clear, and transparent, showing under the microscope many short, thread-like, wavy projections, which sometimes seem to be separated from the colony. Surface colonies are small, circular, and clear; microscopically show a yellowish nucleus, with a very thin transparent periphery, often prolonged into thread-like processes, extending out and down into the medium.

Laboratory Stock Gelatin. Growth in stab inoculations at 22° C. is slow. At seventy-two hours a thin white growth is visible along the stab; delicate, not continuous; no gas. There is very little change in thirty days. Similar inoculations of hog cholera show at seventy-

two hours a vigorous continuous growth; of bacillus coli a strong growth and gas formation.

Elmer's KI Potato-agar. Potato, 500 gm.; distilled water, 1000 c.c.; boiled one-half hour; filtered; gelatin, 100 gm., added; neutralized by NaOH; filtered; sterilized; 1 per cent. KI added; sterilized. Inoculated from glycerin-agar culture and plated. At twenty-four hours (37° C.) no growth is apparent. At thirty-six hours 3 colonies are visible to the naked eye; at forty-eight hours, 12; at seventy-two hours, 41. They appear as minute, shining, clear points. Under the microscope the deep colonies are very small, slightly brownish, homogeneous, with finely beaded edges. The superficial colonies show only an intimation of a nucleus; they are almost transparent throughout, homogeneous, very finely granular, with distinct, regular edges.

Sugar-free Bouillon. Chopped beef-heart infused in distilled water at 60° C. one-half hour. Filtered; inoculated with bacillus coli; incubated twelve hours; sterilized; filtered; peptone, 10 gm., NaCl, 5 gm., added to 1000 c.c.; neutralized (litmus); filtered; sterilized. Twelve tubes inoculated from glycerin-agar examined at intervals of two days for indol show no indol production, except tube 8, at sixteen days, which shows a possible trace. Comparative inoculation with bacillus coli shows positive indol production at thirty-six hours.

Arsenious Acid Bouillon. Sugar-free bouillon, with (a) 0.005 per cent., (b) 0.01 per cent., (c) 0.02 per cent. arsenious acid added. Inoculation from glycerin-agar in (a) shows no growth in seventy-two hours at 37° C. to the naked eye; but, microscopically, some multiplication is apparent. Both b and c show no growth at all in six days. Bacillus coli grows fairly well in c.

Dextrose Bouillon. Sugar-free bouillon, with 1 per cent. dextrose added. Inoculations from glycerin-agar in fermentation tubes show no gas production in six days. In test-tubes 3.2 per cent. acid is formed in six days as shown by titration to neutral (phenolphthalein), with tenth standard NaOH. In the same medium the bacillus of hog-cholera produces a bubble of gas and 3.4 per cent. acid in six days.

Lactose Bouillon. Sugar-free bouillon, with 1 per cent. lactose added. Inoculation from glycerin-agar produces no gas in six days in fermentation tubes. In test-tubes 0.3 per cent. alkalinity is produced in six days, as shown by titration to neutral (phenolphthalein) by oxalic acid and NaOH. The bacillus of hog-cholera produces under the same circumstances no gas and 1.1 per cent. alkalinity.

Sucrose Bouillon. Sugar-free bouillon, with 1 per cent. sucrose added. Inoculation from glycerin-agar in fermentation tubes produce no gas in six days; in test-tubes 2.6 per cent. alkalinity in six days. The bacillus of hog-cholera produces no gas and 2.9 per cent. alkalinity in six days in the same medium.

Laboratory Stock Litmus Milk. Inoculation from glycerin-agar produces no discoloration on the fourth day. In four weeks there is an intensification of the blue color. The milk is not coagulated.

Petruschky's Litmus Milk Whey. Milk clotted by rennet and strained; whey neutralized with citric acid (litmus); heated at 100° C. one-half hour; filtered; neutral litmus added; sterilized. Inoculation from glycerin-agar produces in twenty-four hours at 37° C. a very slight cloudiness and acid reaction of 1.8 per cent.; at forty-eight hours a slight cloudiness and acid reaction of 2.4 per cent.; at seventy-

two hours acid reaction of 2.6 per cent.; no further change at ten days. In the same medium bacillus coli produces in forty-eight hours marked cloudiness and 9.8 per cent. acid.

Capaldi-Proskauer (9), *Media I. and II.* Inoculation from glycerin-agar in I. shows no growth; in II. shows slight cloudiness and 1.2 per cent. acid in forty-eight hours at 37° C.; alkaline on the sixth day. In I. bacillus coli produces some cloudiness in forty-eight hours; in II. marked cloudiness and 3.7 per cent. acid in forty-eight hours, remaining acid at ten days.

Laboratory Stock Glycerin-Agar. The surface of solidified plates is inoculated with a spatula over an area one-half by two inches with (a) laboratory stock typhoid, (b) hog-cholera, (c) bacillus coli. The plates are then incubated for seventy-two hours at 37° C., and the resulting growth carefully scraped off. The plates are then re-incubated for twenty-four hours at 37° C. to insure no further growth. Streak inoculations from glycerin-agar on *a*, one through the middle of the scraped area and another on fresh medium at one side produce a good growth on the fresh area and no growth at all over the scraped area. Similar inoculations on *b* and *c* produce growths along all streaks, those over the scraped areas in each case being less vigorous than the parallel side streaks on fresh medium, plates in each case being incubated forty-eight hours at 37° C. On plates similarly prepared with the bacillus under discussion, and inoculated similarly in streaks with (a) stock typhoid, (b) hog-cholera, and (c) bacillus coli, *a* produces a good growth on the fresh medium and no growth at all on the scraped area; *b* and *c* grow on both, though not so well on the scraped area as on the fresh medium.

Agglutination Experiments. Positive agglutination of the bacillus is obtained with blood from the liver and spleen of the patient, dried on blotting paper at dilution (estimated) of 1 to 50. With a serum from a severe case of typhoid fever, having an agglutinative power of 1 to 1000 on stock hospital bacillus typhosus, a positive reaction is obtained in dilution of 1 to 500. Agglutination is regarded as complete when large clumps of bacilli are produced, with practical cessation of motility and clearing of the field of single organisms, bacilli from twenty-four hour glycerin-agar cultures suspended in distilled water being used for tests.

Intraperitoneal injection of a forty-eight hour culture on glycerin-agar in distilled water in a guinea-pig causes evident illness, with weakness and loss of appetite. Temperature after thirty-six hours, 105.5° F.; after three days, 104.8° F.; after four days, 104.2° F.; animal apparently well.

Serum from this guinea-pig before inoculation gave no agglutination reaction with stock cultures of bacillus typhosus, hog-cholera, nor the organism under discussion. After receiving, on each second day for thirty days, intraperitoneal injections of increasing quantities of live bacilli in distilled water—the last dose being the forty-eight hour growth from ten glycerin-agar tubes—the pig's serum gives a positive reaction with this bacillus in a dilution of 1 to 10,000. With four races of typhoid bacilli from different hospital stock cultures this same serum gives a positive reaction in dilutions of 1 to 4000, 1 to 1000, 1 to 2000, and 1 to 400, respectively. With the hog-cholera bacillus a reaction occurs at 1 to 50, which is suggestive at two hours. On bacillus coli the serum has no effect whatever.

There can be no question that we have here to do with an abscess of the brain caused by a true *bacillus typhosus*. The organism is differentiated from the colon group by all cultural appearances and reactions, and from the Gaertner group by its growth on gelatin and in the sugar and milk media, as well as by the results of agglutination experiments. The characteristic growths of the other organisms of the genus on the same media is sufficient guarantee of the correctness of conclusions drawn from the cultural appearances, and the agglutination results are as definite as could be wished, if we bear in mind the inconstancy of the effects of so-called "specific sera" on their own and nearly allied races.

I wish to express my gratitude to my teacher, Dr. Hektoen, for his many suggestions and helps in the course of the bacteriological examination, and to Dr. W. G. Willard, of Chicago, for the clinical history.

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MODERN MASTOID TREPHINING OPERATIONS,

WITH NOTES ON ONE HUNDRED RECENT OPERATIONS.

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MASTOID trephining is a term growing less common, both because the crown-trephine has almost wholly passed out of use in this field and because increasing elaboration and differentiation have substituted special names to indicate the varying purpose and technique of the intervention. Too often these specialized procedures, as in other surgical lines, have been veiled behind personal names which, even to the specialist, convey hazy if not erroneous ideas as to their nature; and, while the best otological text-books generally present the complex matter fairly, the same can rarely be said of the current papers in the journals. The specialists in each surgical field are apt to write for special audiences at medical meetings, are prone to use technicalities and personal names that convey little to the general reader, and their writings are generally given to the special journals both by choice of

the writer and through the indifference of the other editors. Yet it is of great moment that the general practitioners, who must render first aid in the majority of ear diseases, should have clearer and sometimes fuller information in these lines, not only that they may better advise their cases, but also that in the numerous instances where the operative intervention must be carried out with no help from the especially trained colleague, they may do so unburdened by the many exploded teachings which still occupy the surgical works or by the too specialized descriptions of otological literature.

Even the fundamental facts in this field of practice have not yet been clearly grasped by many otological practitioners; their anatomical knowledge is still encumbered by old misunderstandings, and the semi-isolation of the specialist has at times led him to rest too much upon his individual experience, which may have been both narrow and wholly exceptional. Some have urged that operation should never be needed, since they have met no cases which, to them, presented indications clearly demanding it; others have seen in hospitals such series of cases, already past all chance of resolution, that they feel that non-operative measures are generally inefficient and signs of culpable timidity. Dispensary practice, in spite of its wealth of opportunity, is far less deeply instructive than the close acquaintance and control of patients possible in private practice, especially in the more isolated communities, and may beget superficial views and needlessly radical tendencies to prompt operation; and the material is so different, and the personal tendencies and class tendencies both in habits and pathology vary so utterly, that our ideas must be broad to the limit of definiteness if they are to embrace any majority of our experiences. Some elementary considerations, therefore, seem called for.

Almost all demands for the interventions under discussion arise from infective, purulent catarrh of the middle ear. This middle ear is not an unknowable field beyond the ken of the general practitioner; less still a tiny field of which the tympanic membrane, often miscalled the drum, is the major part. It is a varyingly complex mucous tract extending out and back from the nasopharynx, where it begins as the Eustachian tube mouth visible in the rhinoscopic mirror, and it comprises the tube, tympanum, and pneumatic cells—the last most conspicuous in and about the mastoid process, but by no means confined to it, since they frequently pervade the whole temporal bone from zygoma to sella turcica, and may invade the adjacent occipital bone behind and below. The auditory canal, with the drumhead at its bottom (Fig. 1), gives us access to the middle of the tract—its strategical key, as we may say—since here the battle to save the hearing and to forestall the numerous malign possibilities can be most promptly, readily, and successfully fought. The conducting apparatus of drumhead and ossicles here

located is easily damaged, and then becomes a hinderance instead of an aid to hearing ; and while experience has exploded most of the claims as to any great relief of deafness by their removal, it has justified this measure when they seem obstructive in the suppurative conditions. The tympanum or drum-cavity comprises the antrum and the attic spaces, as well as the lower cavity just within the drumhead (" atrium "—Leidy). Those less obvious upper portions, which are with difficulty seen or reached from the canal, have great pathological importance, since no direct drainage by way of the Eustachian tube is possible to them ; nor, when it fails, is there a yielding membrane to give, in a safe direction, easy exit to pent-up secretion. The tympanic attic is sub-

FIG. 1.

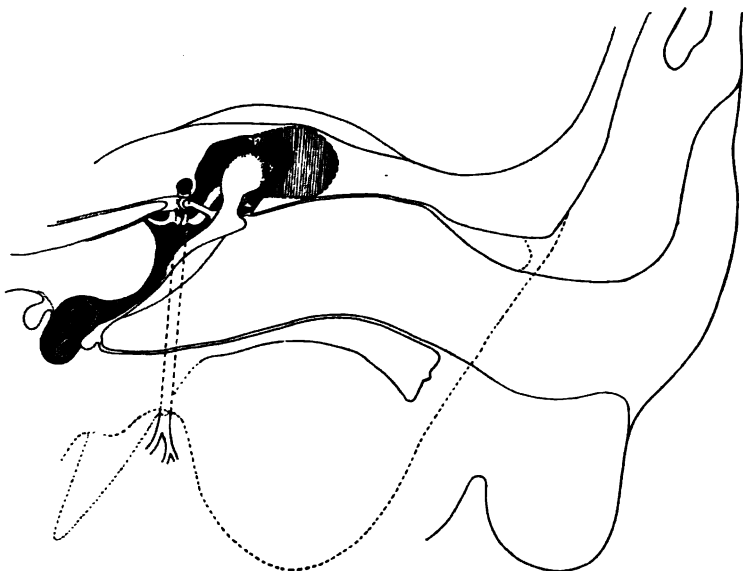


Diagram of canal and tympanum, showing the relation of the attic and shaded antrum to the canal and the mastoid process, and the course of the facial nerve above the stapes and downward to the stylomastoid exit.

divided by the ossicles and their ligaments, and can generally drain through the lower cavity, from which it is separated only partially by soft-tissue septa ; but the antrum has only its limited opening into the attic, which may be so obstructed that any septic contents can pass with least resistance into the mastoid cells and involve them in its supuration. Bearing in mind that all these cavities are lined by delicate mucoperiosteum, we realize that mere superficial ulceration may destroy the vitality of the underlying bone, even if this has failed to become itself infected. Hence the rapidity with which extensive temporal caries may follow nasopharyngeal suppuration ceases to be any marvel.

But the antrum is not an isolated structure—the thin tegmen covering it and the attic connects rather than separates its lining and the dura mater of the middle cerebral fossa; its posterior wall is almost as thin toward the sigmoid sinus; the bony canals of the facial nerve and horizontal semicircular canal form its inner wall, and the bulb of the jugular and other vessels are, in rare cases, in close proximity, and in all cases receive its emissaries—so its septic involvement readily gives rise to life-imperilling processes. Outward, its walls may be dense and impervious; not only preventing the forcing of an exit in this direction, but even shutting in almost every sign of the underlying inflammation.

Accepting the demonstrable fact that the tympanic antrum is involved in almost every suppuration, and is the centre of the carious process in most cases requiring intervention, it is evident that our trephining procedure, to be efficient, must succeed in draining it, and not rest content with having entered the cellular structure of the mastoid process. There are some cases, especially among those due to influenza, in which the empyema seems limited to the mastoid tip, full resolution having occurred in the tympanic cavities. In these rare instances it may suffice to open and drain the lower areas, without penetrating to the antrum; but, even of these, some will prove disappointing if more thorough work is not done. For the rest, antrum drainage is always requisite in the acute cases, while in the mastoid empyemas dependent upon chronic suppuration this must constitute but a part of a more radical measure. And in the large group of the chronic suppurations which resist all milder efforts to cure them, operation for exenteration of the tympanic cavities must be considered, even though no evidence of mastoid empyema or caries is recognizable, for experience shows that too large a number are maintained by caries or cholesteatoma, and sooner or later have extension of trouble which may then prove irremediable.

This distinction between the acute and the chronic suppurations is cardinal, yet it still lacks due general acceptance even among otologists. With the misleading histories so commonly given, we realize too late in some of our cases that we have done only the simpler antrum operation where we ought to have exenterated all the tympanic cavities. Great good may nevertheless be achieved in affording a temporary relief, under which the patient improves and the suppuration seems nearly cured; but a partial success is really a failure, and recurrence of local lesion and intracranial extension may yet follow the imperfect intervention, while the dashing of the patient's hopes may suffice to prevent his assent to later and more urgent operation. At best, other advice will often be sought, and all credit of benefit go to the second operation, while any mischance may be ascribed to the failure of the first. Infal-

libility will never be attained. The human organisms upon which we operate are too various, and are subject to ups and downs that must hugely influence results, and the highest skill in diagnosis and treatment cannot preclude some failures; yet some of these may be foreseen and forestalled under simple rules.

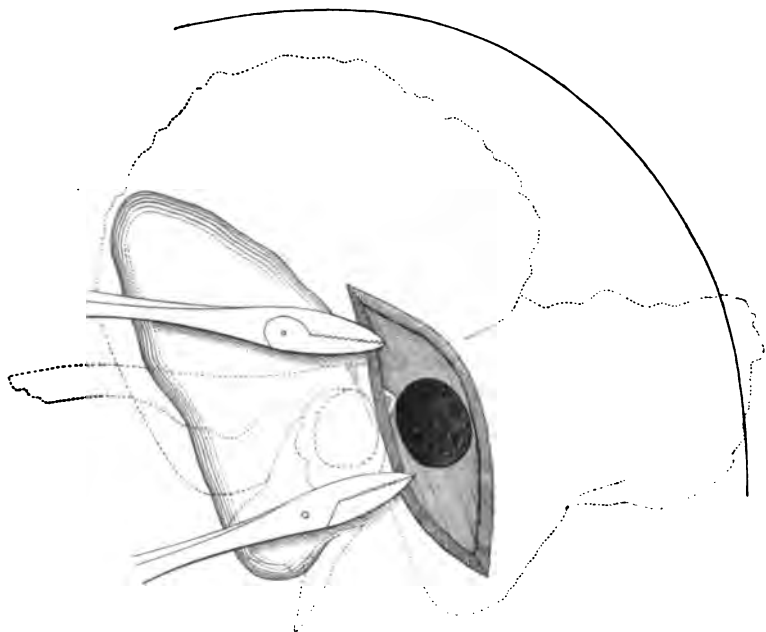
Space is lacking to go into full detail as to the indications for operation. When, in a suppurative catarrh of the tympanum, pain, tenderness on pressure, fever, swelling, and redness of the mastoid region persist beyond a week after free incision of the drumhead and adjacent wall, with complete rest in bed and vigorous use of hot, sterile douching in the canal and heat or cold to the mastoid, there ought to be little delay in opening to the antrum to seek and evacuate retained pus. Fever or rigors may be lacking, pain absent except at night, swelling and other local evidences hardly if at all recognizable, even on the upper back wall of the canal, and yet experienced surgeons will "feel" the need of exploratory operation. Undefined as may be the indications, it is yet clear that delay is more dangerous than intervention. In the hands of a skilled surgeon operation should hardly more than double the scarce measurable dangers of etherization; and unless extensive lesion is found there should be practically no scarring, as to which we must pardon our patients for being at times more solicitous than as to deafness or even life-risk. For mere suppuration without disquieting symptoms we must as yet be very cautious in urging operation, and only after full effort has been made to improve the general and local health should we decide that tympanic exenteration is needful.

From the foregoing considerations much of our operative work readily shapes itself. In the acute empyemas of the mastoid we must drain the purulent area, which is almost always the antrum, with few or many of the adjacent cells. To this end we incise a little back of the insertion of the auricle, carrying the knife to the bone and from the tip of the mastoid process to the upper part of the helix, thus giving an ample wound of the soft tissues. The periosteum is then lifted (the gouge often makes the best raspator), especially forward, until the back margin of the canal is reached, with its "spina suprameatum." (Fig. 2.) This thorn-like suprameatal spine, with a depression behind it, can be recognized with the finger in almost every case; is the only reliable element of any "post-meatal triangle," and furnishes our best landmark. Close behind this is the typical point for opening the bone. The floor of the middle cerebral fossa may be no higher than the spina, but can hardly be lower; the knee of the sigmoid sinus may come thus far forward, instead of lying the average ten millimetres back and up; the antrum may be but a millimetre within the cortex in an infant, or an inch away in the adult with a large mastoid; yet in general we can safely chisel inward, forward, and upward from a point close behind

the spina, and a depth of some fifteen to twenty millimetres should enter the antrum. Remembering that this cavity lies up and back from the canal, separated from it only by a wedge-shaped partition of bone, we have a constant guide to it furnished by a probe in the canal, the roof of which is about level with the floor of the antrum.

As there is no means of foretelling the relation of the lateral sinus to the operative field, it is essential to safe work that we employ, instead of any blindly-entering boring device, the chisel or spoon, which enable us to remove scale by scale of the bone without risk to the underlying

FIG. 2.



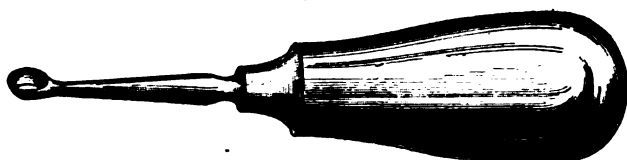
Mastoid incision and bone opening for simple antrum exenteration, to be extended in every direction required. The soft tissues are pressed forward to reveal the spina suprameatum. The region shaved within three inches from the canal.

structures. Further, it is needful to rest the thumb or the finger at the very edge of the chisel, so as to exercise full control. So working, whether by hand-power only or with the mallet to propel the chisel, the bone can be quickly cut away without risk to the sinus, if in the field, or of entering precipitately into a cavity such as we often find occupying the whole mastoid process. The old plan of entering on the convexity of the mastoid at about the position of the incision through the soft parts will frequently endanger the sinus, as this is the point where it commonly comes closest to the surface; and to use a gimlet, as some

even of the recent surgical works advise, is permissible only in dire emergency. Personally, I have come to prefer the spoon as devised by J. F. McKernon, with fenestrated cup, short shank, and pear-shaped handle (Fig. 3), the whole but twelve centimetres long, supplemented by a gouge ten millimetres broad, similarly mounted, and a strong but narrow-beaked rongeur, which can enter deeply into a narrow tract. (Fig. 4.) A flatter spoon is safer for removing the inner table for the free evacuation of extradural collections, and a narrow and more beaked spoon for exenterating the antrum or other tympanic cavities. (Fig. 5.) For the mallet I have no use, and regard its blows as deleterious.

When the mastoid empyema or caries is due to *chronic* tympanic suppuration, the antrum operation, however thorough ("antrectomy"—

FIG. 3.



McKernon's spoon, with straight-edged, fenestrated cup.

FIG. 4.



Back-curved, short-handled gouge.

FIG. 5.



Double-ended beaked spoon for antrum and attic.

M. Sheild), will generally prove inadequate. Not only must the antrum be drained and scraped out, and all septic or suspicious foci elsewhere similarly dealt with, but we must empty and render free for good future drainage all the tympanic cavities. This "radical operation," better defined as "tympano-mastoid exenteration," owes much to Kuester's "Cure of Suppuration in Rigid-walled Cavities," as well as to Stacke and other otologists. Its essence is that we throw attic, antrum, and lower tympanic cavity, together with all affected mastoid cells, into one with the deeper part of the canal by removing the septa which separate them, and then by plastic measures cover in promptly the walls of this large cavity with healthy epithelium. (Fig. 6.) Remembering that the upper back wall of the canal is formed by a wedge-shaped plate of the squama

("scute"—Leidy), we see that this, with the drum-membrane, malleus, and incus are to be removed, as well as so much of the back wall of the canal as is needful to give present and future access to the whole tract involved. Some operators work from within outward, most from without inward—I prefer to work in both directions; but in any case the soft tissues are to be shelled out of the external canal to give good access to its back wall.

The incision for this operation must be wider, passing above the auricle to or even through the temporal artery, and this or the previous incision can be enlarged by cutting backward. Its lower or mastoid portion may be a single cut to the bone, but the upper part is best carried only to the fascia and a flap of the superficial tissue turned down as far as the zygoma and its suprameatal ridge; then the periosteum

FIG. 6.

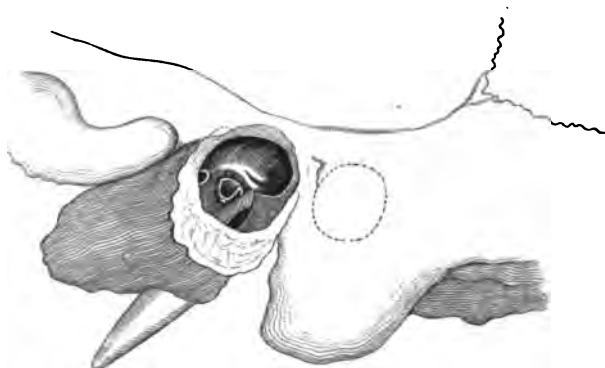


Diagram of tympanic exenteration, with removal of the outer wall of attic and antrum, uncovering the stapes, and above it the ridges of the facial and horizontal semicircular canals. The site of the usual mastoid opening (which some needlessly make as part of this operation) is dotted.

is cut below this line and detached forward and downward into the depths of the canal. Incision within the canal is needless if the gouge is used to shell out the soft tissues, for they rupture at their thinnest part, close to the drum-head, and can be readily turned forward out of the way; but all roughness and crushing is to be strenuously avoided, since these are the best tissues to preserve for our plastic work. With the bare temporal bone before us we can proceed according to the indications—exenterating the tympanum only, if this seems alone involved, or removing the entire mastoid and curetting open every adjacent cell which is even suspected of complicity. Those just within the digastric fossa are especially to be remembered, for they are often large and especially vulnerable. Nor must we in suspicious cases limit our work to the bony structures, for infection has often passed down or back along

the muscles and their fascial sheaths, following the great vessels of the neck or burrowing under the occiput, and any infected glands, especially in tubercular cases, should be thoroughly curetted.

In this exenteration of the tympanic cavities we can work from within outward, as advocated by Stacke, or from without inward, as urged by Kuester, or combine the methods, as done by Schwartze and others, the object being always to obtain quickly, safely, and thoroughly the elimination of the diseased foci. As an hour or more is often required to do the work thoroughly, all waste work is to be deprecated; so the opening of the healthy mastoid merely to introduce a "protector" in front of the facial nerve seems a clumsy procedure when the chiselling can be restricted to the affected region. By entering the upper back wall of the canal some ten millimetres exterior to the annulus, and working a notch with a shoulder past which the chisel will not slip, the attic and the antrum can be extensively opened before the tympanic margin is touched, a bridge of the bone being retained as a "protector" in front of the facial canal, and broken away by safe *outward* traction when it has been so thinned as no longer to be a safeguard. That this is all done at the bottom of the narrow though bare external canal, and is somewhat impeded by blood, is no reason for opening more bloodvessels, as in the Schwartze-Stacke operation; and only occasionally does it seem needful or advisable to chisel away the whole of the back wall of the canal merely to gain a little room.

When some cases unexpectedly reveal outward extension of the diseased process, and demand full mastoid exenteration, we may regret that we had not saved time by doing this curetting first; but as it can later be as safely and thoroughly done at small cost of time, this group furnishes no reason why we should subject the larger number to needless mutilation.

The combination of the two procedures above spoken of must be the rule for all cases of mastoid caries with chronic tympanic suppuration. A few of these would be cured by simple mastoid exenteration; but as it is impossible to foresee this, the "radical operation" is safer and more truly conservative. To call it "tympano-mastoid exenteration" leaves no doubt as to what is to be done, nor does it apparently bind the operator down to "trephining" or "chiselling" when he may prefer the curette. In many of these mastoids an old bone sinus will be already present, and the underlying large carious cavity is best opened fully by the rongeur. Work inward which is not fully guarded from undue penetration is especially dangerous, since the inner table of the mastoid is often extensively lost, and the sinus or other dural surfaces are open to easy injury. Indeed, the pachymeningitic thickening may be accompanied by so much softening or even ulceration that gentle use of the probe is not safe against penetration into the bloodvessels or

the brain. It is wiser wholly to avoid the dangerous directions until the work in other parts has been completed; then it will be easier to see exactly what remains to be done and to complete it with safety; or if hemorrhage compels us to desist, we will leave little undone. A shallow spoon can most safely be carried along any bare dura, cutting away outward the soft margins of the inner table and exposing any extradural abscess; yet even the smooth back of this spoon, if not gently used, may penetrate. Some authorities counsel against the removal of pachymeningitic granulations, believing them wholly protective; but individual decision must be made on this point, and as they are generally infected, the presumption is against the safety of leaving them uncuretted. Avoiding, then, all needless extension of the operation, yet conscientiously following to its end every unhealthy tract in bone

FIG. 7.

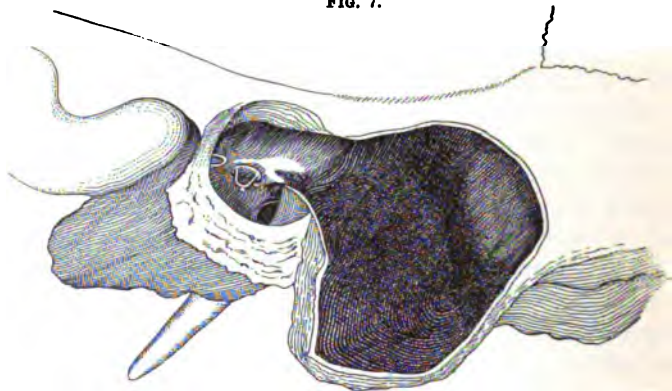


Diagram of tympano-mastoid exenteration, showing the empty cavities thrown into one with the auditory canal, the lower posterior wall of which forms a ridge protecting the course of the facial nerve in its descent to its exit.

or soft tissues, we remove the back wall of the canal, so as to throw into one cavity, without intercepting prominences, all of the affected cavities, gaining an open wound of which every part should remain accessible for the future. (Fig. 7.)

To this end the plastic idea advocated by Stacke and Kuester has given great aid. Instead of packing the extensive cavity more or less completely from behind, and maintaining such dressing for months until new tissues fill it, the prime effort is to inaugurate a skin covering of the cavity as a diverticulum of the external canal, draining, and open to ready access through the natural opening. The mastoid wound need not be maintained except in rare instances of inveterate cholesteatoma-formation, and these will generally declare themselves by refusing to heal, so the attempt at prime union can usually be made by complete but not very close suturing of the mastoid incision. The flaps by means of which the bare bone is covered are variously made, generally

from the soft tissues of the canal, supplemented perhaps by skin from the exterior of the mastoid, and secondarily by Thiersch grafts. One important principle has been too generally neglected: The cartilage of the canal is useless for grafts, melting down itself, and too often involving in its necrosis the better tissues. It should be excised in forming the flaps, therefore, and in doing this the canal wall should be *split* into the periosteal and skin layers between which the cartilage lies. A square inch of tissue can thus be made to yield two flaps of this extent, one of which is generally turned up and the other downward, to cover as much as possible of the cavity wall. Some surgeons merely excise these soft tissues, so as to gain free opening through the back wall of the canal; some make flaps of the entire thickness, which are ill-fitted, by their thickness and by reason of the contained cartilage, satisfactorily to close this cavity, while their area is but half of that obtained by the method above described. The osteoplastic method of Kuester seems a wholly needless procedure, suggesting clumsiness of the surgeon rather than up-to-date resource. Packing is to be carried through the canal in all but the simple antrum operations (which may be allowed to close with blood-clot), so as to hold in place the plastic flaps—an adjustment best made before the posterior wound is closed, but after all the sutures have been placed.

Healing after the operation varies according to its extent and the vital powers of the patient, being attained in one to four weeks in the simple antrum exenterations, in three to six weeks in the tympanic interventions, while for the total covering of a large mastoid wound six to eight weeks is a safer allowance. But of this time little need generally be spent in confinement. Three to seven days in bed, with dressings as infrequent as pain, fever, discharge, and odor will permit, and as much longer detention in the house, usually suffices. Bandaging is needless after the first week in some and after the second week in all but a fraction of the cases; stitches are generally removed on the fifth or sixth day, and packing is rarely called for after the first week. Irrigation should be used if odor is present; and cleansing by way of the canal, with cauterization, by silver or chromic acid, of exuberant granulations is requisite in the later stages to prevent stenosis of the opening back into the antrum and mastoid.

Facial palsy will be caused in a few cases, rarely in careful hands, except when the caries already surrounds the nerve. For these it is fair to claim that it has been precipitated rather than caused, for the lesion would generally have spontaneously occurred, and all too often would have proved more persistent. The surgical paralysis is generally cured in from six to twelve weeks. If we avoid too tight packing, which may press injuriously upon it in the upper part of its course, and in removing the back wall of the canal go no lower than the middle

of the tympanic margin, we ought to run little risk. To watch the face for evidence of irritation of the facial should be merely an adjunct to the more surgical plan of knowing where one is and what he is doing, with avoidance of the well-defined path of the nerve. This is exposed to injury from the geniculate ganglion just above the trochlea of the tensor tympani, throughout its backward course above the oval window and in its vertical portion from three millimetres back of the middle of the tympanic margin to its stylomastoid exit, but in my own hands has been almost never injured.

As to the hearing, the operation can be considered purely conservative, its early performance frequently saving hearing which might have been lost in the longer-continued inflammation; and the operation, by terminating the disease, generally gives the only reasonable chance for all possible retention of function. The removal of the drum-head and ossicles which constitute a part of the tympanic exenteration has proved of little value for the improvement of hearing, but simply conserves what has been left by the suppuration.

Among the mastoid operations of the past two years, numbering one hundred, forty-nine were the simple antrum operations, generally for acute lesions, thirty-nine were radical tympano-mastoid exenterations, and but twelve were done after Stacke's method of tympanic exenteration for chronic suppuration without mastoid caries. Two only of the acute mastoid empyemas—one scarlatinal and one grippal—occurred under my own care, unless we include a feeble old Hebrew who refused to come into the hospital, but attended irregularly at the out-clinic until long after operation was clearly unavoidable, yet who ought probably to have been saved from it by rest in bed. This does not imply that most of the others might have been saved by skilful treatment, but it does bear out my contention that vigorous hot douching and such methods cannot be so harmful as claimed by some authorities, since quite six hundred acute suppurations—nearly two hundred of them with mastoid tenderness, redness, and swelling—when first seen were so treated within these two years, and three at most are known to have gone on to mastoid caries. Where the suppurative process, having reached or passed its acme, has brought the bony tissues to the verge of destruction, theory as well as abundant experience convinces me that *heat* is our best aid in combating the danger. This can best be applied by the hot douche in the canal, which carries the heat to and even through the affected tissues, supplemented by dry heat alternating with mere protection over the mastoid. Warmth and moisture may be deleterious, but heat pushed to the limit of tolerance should have all the constringent effect claimed for cold, while more conservative of the vitality. More than one thousand mastoid inflammations which I have treated with heat have gone on to prompt resolution, with hardly a dozen instances of

failure to abort the empyema, while it so chances that almost every one of the few cases treated with ice have gone on to operation.

The mortality after mastoid operations varies largely through extraneous causes, few if any deaths being ever ascribable to the intervention. My list of eleven deaths in this hundred equals my whole previous loss in some three hundred operations, and includes three children dying of pneumonia and other unrelated conditions; two in whom fatal meningitis was present before operation and was unrelieved by removal of the primary focus of infection in the temporal bone, or in one case by exploratory opening and flushing of the meningeal cavities. Two succumbed to pyæmia from lateral sinus thrombosis, one to brain abscess which had been found and drained, and one to recurrent cerebral apoplexy. Early operation can do much to prevent the occurrence of these fatal complications, and even after they have arisen may snatch from the very jaws of death cases which must otherwise have speedily died. In this latter group may fairly be counted the cured cases of brain abscess, and several among some thirty marked extradural abscess cases, in three of which ulceration had already destroyed an area of the dura. Yet there are acute cases in which meningitis seems toxic rather than infective and may even antedate the mastoid involvement, just as many cases die without evidence of a drop of pus, still less caries, in any part of the ear. If the infected tract within the temporal bone be always followed to its end, with good drainage of the frequent extradural collections, and a conscientious penetration on into the meninges or into the brain when there is probability of involvement of these deeper parts, the death-list will be further reduced. Tubercular and other forms of leptomeningitis ought to yield nearly as good results to aseptic irrigation as have been obtained in the peritoneal cavity, if the mechanical difficulties of flushing, without dangerous pressure, the intracranial cavities can be overcome. To this end I believe an irrigation tube, with minute openings at its spatula tip, will serve a very good purpose, since it can be passed far under the brain and, lifting it away from the dura, permit of a good flushing even to the base.

Another point as to these intracranial explorations seems to demand a few words. Most writers teach and practice a penetration through the mastoid in the cases where they have reached or even approached the dura and see cause to open further. I believe this is bad policy, for a carious mastoid cannot be made or maintained so aseptic that it is fair to open communication between it and healthy intracranial tissues. The route may be an easy and direct one and the best line of drainage for any brain abscess that has been found; but it is far safer to make the search from separate and absolutely aseptic skull openings in the usual surgical way, since negative exploration is more likely to be harmless when thus carried out.

CORRECTED MAL-UNION IN FRACTURES OF THE RADIUS AND ULNA OF BOTH FOREARMS.¹

BY CARL BECK, M.D.,
OF NEW YORK.

THOROUGH adaptation of the fragments is essential to preserve the functional ability of the bones of the forearm. Fracture of either the radius or the ulna alone when perfect coaptation is not secured may prevent supination to such an extent that the unfortunate patient may be prevented from following his occupation. How much more is the functional ability impaired when, after fracture, both bones unite in false positions, with overlapping of the fragments and angular deformity.

To what extent the Röntgen rays enable us to overcome some of the technical difficulties, even in desperate conditions, is illustrated by the following case:

Both forearms of a laborer, aged thirty-four years, were caught in the wheel-strap of a powerful machine and broken. The patient was brought to the hospital, where proper efforts were made to reduce the displaced and partially splintered fragments.

At first reposition seemed to have been successful, and the swelling disappeared, but both hands remained stiff and paralyzed, and, excepting the thumbs, were without sensation.

Extensive oedema having repeatedly been present, the disturbance in motion as well as in sensibility seemed to me to be of an ischæmic nature rather than caused by a direct trauma to the nerves. The muscular atrophy, which is still present, also points to a breaking down of contractile muscular elements. By the courtesy of Dr. Norris, of Passaic, I had an opportunity to see the patient, for the first time on July 24th, three months after the injury. I then found angular deformity, abnormal mobility, and the functional disturbances described above on both sides.

The skiagraphs of both forearms showed considerable overlapping. In the right forearm overlapping of the radial fragments existed in the middle, and lateral deviation of the ulnar fragments an inch below. The skiagraph of the left side shows the overlapping of both sides, and also the formation of a bone bridge between the lower fragment of the ulna and the upper fragment of the radius, which alone would exclude any possibility of rotation (Fig. 1).

On August 3, I concluded to correct the position of the fragments of the right forearm by exposing and freeing them. Under the guidance of the skiagraph I made a semilunar incision in an oblique direction, and overcame the shortening by resorting to forcible extension. The fragments were then united by silver-wire sutures. The *modus operandi* consisted in freeing the old adhesions thoroughly with knife

¹ Case presented to the Surgical Section of the New York Academy of Medicine, October 14, 1901.

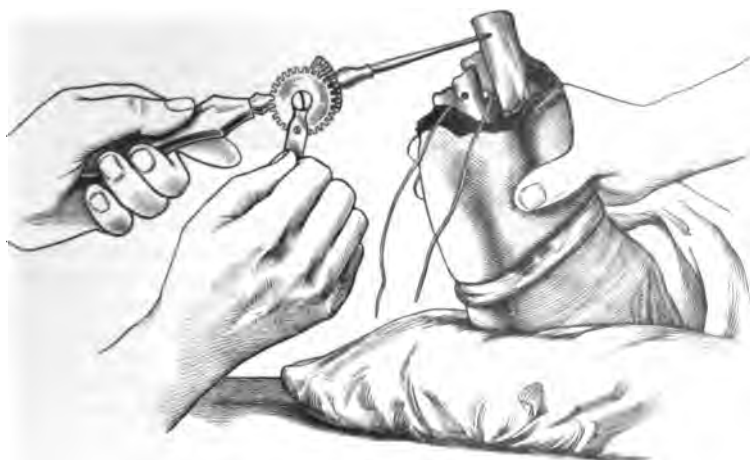
and chisel and folding the arm, so to say, completely. By encircling the two antibrachial fragments with a strong bandage the folded mass

FIG. 1.



Overlapping of fragments and formation of a bone bridge between radius and ulna.

FIG. 2.



Boring holes while the forearm is folded.

could be steadied by an assistant, so that the necessary holes could be bored (Fig. 2). As shown by the skiagraph (Fig. 3), taken six weeks after this operation, the radial fragments are in ideal apposition. The ulnar fragments show slight lateral displacement, while the callus formation has assumed so fortunate a character that no other depression or protrusion remains.

Five days after the first operation the left side fracture was exposed in the same way. In spite of extensive exposure of the fractured area the fragments could not be forced into apposition except by shortening them. So I made a virtue of necessity by giving the ends of the fragments a triangular shape, which enabled me to indent them into each

FIG. 3.



Fragments wired.

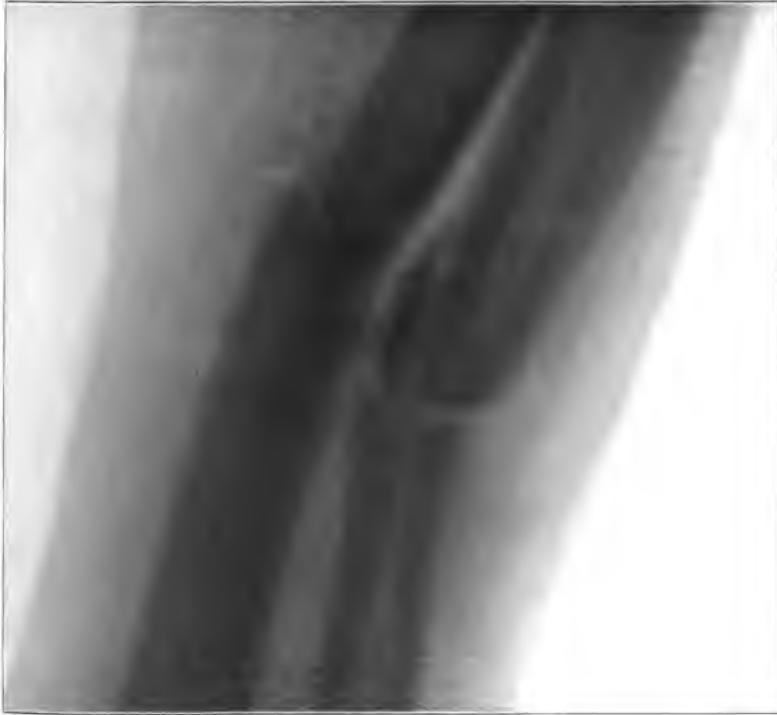
other. As the skiagraph (Fig. 4) shows, taken four weeks after this operation, the apposition of the radial fragments is perfect. The ulna does not show lateral deviation, none of the medullary lines presenting any axial divergence. The skiagraph showed, however, that there was a slight angle, which could be corrected in time by simple pressure.

Union took place by first intention without any reaction. The healing process was quicker on the left side, which is explained by the absence of wire-suturing. To be sure that consolidation was perfect, I immobilized the arm until the present time, leaving off the plaster-of-Paris splint temporarily for the employment of massage. The final result will, no doubt, be perfectly satisfactory.

Thus we see how the happy era of combined asepsis and skiagraphy permits of the correction of even the most extensive deformities in a simple and safe manner.

From a theoretical point of view it should be expected that on account of the more abundant callus-proliferation, induced by the irritation of the wire sutures, consolidation would be quicker and more thorough. But practice proved the contrary in this instance, and it seems to me

FIG. 4.



Fragments indented.

that indentation permits of more accurate adaptation and immobilization. At the same time larger surfaces for agglutination are obtained and the soft tissues are less liable to be disturbed. The triangular indentation of the fragments should therefore be preferred, whenever possible.

The *modus operandi* for indentation is practically the same as demonstrated by Fig. 2.

ALBUMINOUS EXPECTORATION FOLLOWING THORACOCENTESIS.¹

BY DAVID RIESMAN, M.D.,

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TAPPING of the chest for the removal of fluids is ordinarily such a simple and safe procedure that the possibility of serious accident scarcely ever enters the physician's mind. This feeling of security is of decided advantage, inasmuch as it insures a desirable confidence and makes resort to aspiration more prompt and more frequent than was the custom among our predecessors, by whom the gravity of the operation was greatly magnified—so much so, indeed, that many patients died for the want of timely tapping.

The knowledge that accidents may occur is not, however, a real handicap; it merely serves to make us more circumspect in performing the trifling operation, and renders us unsurprised and ready when untoward circumstances arise.

It is not my purpose to review all the complications that may accompany or follow tapping of the chest, such as syncope, pulmonary and other forms of embolism, convulsions, etc. These are adequately dealt with in the articles and works of Leichtenstern, Foucart, Dandridge, and Wilson Fox. I merely intend to give an account of one particular complication, which, although exceedingly rare, has in the past aroused remarkable interest, viz., albuminous expectoration. A survey of the literature shows that this was first described by Pinault in 1853. His patient was a man, aged thirty-four years, who had been suffering for three months with left-sided pleural effusion. Three litres of fluid were removed by tapping, with instant relief of the dyspnoea. In the evening of the same day, however, the shortness of breath returned, mucous râles appeared over the chest, and the patient began to expectorate large quantities of an albuminous fluid, bringing up two cuspidorfuls (about a pint). Cases were subsequently reported by d'Espine, in 1869; by Woillez, in 1872; by Marrotte, in 1872; by Behier, in 1872; and by Lande, in 1873. The entire subject was thoroughly reviewed in 1873 by Terrillon, who collected twenty-one observations, including his personal cases, with two fatalities.

Very little attention was bestowed upon the condition outside of France, yet it is of great interest to note that one of the first publications made elsewhere was that of Dr. William Pepper, in 1874. In a case of hydrothorax seen with Dr. T. J. Yarrow, of Philadelphia, Dr.

¹ Read before the College of Physicians of Philadelphia, December 4, 1901.

Pepper withdrew by aspiration seventy-five ounces of serum. A troublesome cough followed, but was relieved by deodorized tincture of opium. The expectoration set in eighteen hours after the tapping, and continued for twenty-four hours, the patient bringing up three gills of fluid altogether. This fluid closely resembled that removed from the chest. A dangerous syncopal attack, from which the patient was aroused by stimulants, occurred six or seven hours after the operation.

In 1873 the subject of albuminous expectoration formed the theme of a heated discussion in the Société Médicale des Hôpitaux in the Académie de Médecine. This was at the time when, through the efforts of Dieulafoy and Potain, in France, and Bowditch, in this country, aspiration was coming into use, and, as a novel method of procedure, had its advocates and opponents. This discussion stimulated the publication of numerous cases and of opinions on the etiology of the condition, and the French literature of the period contains a large number of reports, among others those of Prévost, Dujardin-Beaumetz, Moutard-Martin, Féréol, Hérard, Deenos and Drivon, and that of Laboulbène, who was one of the first to give a careful analysis of the expectorated fluid. Cases were also reported by Dieulafoy, and later by Hayem and Tissier, and others.

In the German literature I was able to find but three cases—those of Kredel, Schütz, and Scriba, although Leichtenstern, in the article already referred to, makes comprehensive reference to the condition. Strümpell also mentions it.

In England a full report of the French discussion was published by Labbé in the *British Medical Journal* in 1873, and some observations were recorded by Johnson and by Duffin. A clear account of the subject was given by Wilson Fox in his monumental work on *Diseases of the Lungs and Pleura*. The first case reported in detail seems to have been that of Gee, in 1886. A brief and interesting report of a case had been made, however, by Fraser, in 1876. West, Haviland Hall, and Hale White published cases in 1896.

In American literature, aside from the case of Dr. Pepper, I have found scarcely any reference to the subject; Bowditch was aware of the condition, but I was unable to find his original publication. Systematic writers, such as Donaldson, Osler, and Whitney, refer to the subject.

The history of the case which I have had the opportunity of observing is as follows:

Miss J. R., aged forty-eight years, born in Philadelphia; was always healthy, except for sick headaches, from which she suffered all her life, until a year ago, when, at about the time of the menopause, they disappeared. Her mother died of dropsy at the age of sixty-two;

her father is living and healthy ; two sisters died at the ages of twenty-two and twenty-four years, respectively, of unknown causes, but not, it is stated, of lung trouble ; an aunt died of consumption.

A year ago, at the climacteric, she began to suffer with shortness of breath and puffiness of the face and neck. She has had occasional slight cough for the past six months. From time to time she would have intense attacks of dyspnoea and orthopnoea, particularly upon slight exertion. At no time did she have any fever or any severe pain in the chest. Her appetite has always been poor, and for some time she has had slight difficulty in swallowing. Her bowels were at first constipated, but now are regular. She has been under medical treatment throughout the year of her illness. The diagnosis at first was nervousness dependent upon the change of life ; more recently she has been treated for emphysema of the lungs.

Upon examination, in November, 1901, I found her to be a large, fairly well-nourished woman, with cyanosis and puffiness of the face. She was sitting up in bed and breathing rapidly and with great difficulty. The openings of the sebaceous ducts all over the face were unusually large, the lips blue, the sclera dark and cyanotic. The neck was greatly enlarged, and all the natural depressions, particularly those above the clavicles, were obliterated. Above the inner end of the right clavicle a large and hard gland could be felt. The chest was crossed by large veins, especially on the right side, above and below the breast. Resonance was impaired on the right side from the clavicle down to the liver, dullness and resistance being more marked above than below, and extending over, behind the manubrium, to the left border of the sternum. There was some tenderness on percussion near the right apex. Posteriorly there was dullness, but not flatness, at the right base, and some tenderness on percussion. Tactile fremitus was almost absent at that point. Auscultation revealed inspiration and expiration to be equal in length, and harsher over the right side in front than over the left. Posteriorly breathing was quite distinct over both lungs, inspiration being a little harsher on the right side than on the left, and expiration just below the right scapula having a distinct bronchial quality. Vocal resonance was well marked over the right side, and had a slightly twangy character. The heart did not seem to be enlarged ; the apex-beat was not palpable ; there was no murmur, and the sounds were not abnormal. The temperature was 97.8° F. ; the pulse 108. There was a small amount of clear, tenacious sputum, which did not contain any tubercle bacilli.

The existence of the impaired resonance, diminished tactile fremitus, distant bronchial breathing, and twangy vocal resonance led me to suspect the presence of fluid ; and on my second visit I made an exploratory puncture and withdrew a syringeful of clear serum. On the following day, November 11th, at noon, I tapped in the seventh interspace, postaxillary line, and by means of the aspirator drew off three pints of a somewhat turbid, yellowish fluid, frothing readily. After a little less than a quart had been removed the patient began to cough. At first the cough was dry, but toward the end of the tapping a little frothy fluid was brought up. The patient seemed to be dyspnoeic and became blue in the face ; and, as the cough grew more intense, I suspended the tapping and administered a small dose of morphine. As she did not seem to grow any worse under observation I

left her at 12.30 P.M. and saw her again at 6 P.M. During this interval she had expectorated about five ounces (160 c.c.) of a turbid, amber-colored fluid, covered with a layer of froth half as thick as the fluid itself, containing a few streaks of blood and some flocculi. The cough had not diminished to any great degree, but the expectoration had gradually lessened. The patient was very much more comfortable than she had been at noon and decidedly better than before the tapping. Her temperature was 99.1° F.; respirations, 44; pulse, 108. On auscultation moist râles could be detected over the entire right lung from top to bottom, and there was the most exquisite crepitation on inspiration that I had ever heard. Pneumothorax was not present. On the following day there was still a little cough, but the patient was much better than she had been for a long time. The difficulty in swallowing had disappeared, and there was good resonance over the right lung.

Unfortunately, the chest filled up again, and I had to aspirate a second time, on November 24th, this time drawing off about a quart of fluid. There was again intense cough, but no true albuminous expectoration, only a little tenacious sputum. At the last examination, made to-day, I found the chest again full to the top. The dulness behind the sternum has increased in intensity and in extent, reaching now about two inches beyond the left sternal border, and fusing on the right side with the dulness of the fluid. A large, hard gland has also appeared above the left clavicle. The signs are sufficient to warrant a diagnosis of tumor of the anterior mediastinum, probably sarcoma, which is encroaching upon the right lung and compressing the veins that return the blood from the right pleural cavity.¹

The *aspirated fluid* was of a turbid amber color and frothy. It coagulated upon standing, the clot floating at some distance from the bottom. The circumambient fluid was clear, straw-colored, and not gelatinous. It coagulated into a moist, solid mass upon boiling, had a specific gravity of 1021, was faintly alkaline in reaction, gave the biuret test, was free from sugar, and did not form a precipitate with acetic acid. The amount of albumin present with Esbach's albuminometer was 4.5 per cent. The total solids obtained by evaporating, thoroughly drying in the water oven, and standing for twenty-four hours in a desiccator, were 6.8 per cent.

The *expectorated fluid* was of a turbid amber color, was covered with a layer of froth to the amount of about half the thickness of the fluid itself, and had a thin sediment of blood. It was gelatinous, like white of egg, and coagulated upon boiling into a solid mass. The specific gravity was 1018, the reaction neutral, the biuret test positive. There was no sugar. Chlorides were present, and acetic acid produced a distinct precipitate. The albumin amounted to 3.5 grammes with Esbach's albuminometer; the total solids were 5.84 per cent.

As the terms albuminous, albuminoid, and seroalbuminous imply, the expectoration consists of a richly albuminous or serous fluid. It is viscid,

¹ The chest was aspirated for the third time on December 6, 1901, and more than three pints of fluid were withdrawn. A severe, spasmodic cough ensued, but no albuminous expectoration.

Addendum. The patient died on December 19, 1901, and at the autopsy a sarcoma of the anterior mediastinum was found.

frothy, neutral or faintly alkaline, and of a yellowish or amber color, contains a little blood, and is coagulated upon boiling and by nitric acid. As a rule, it closely resembles in appearance the chest fluid. Acetic acid, as in my own case, usually gives a precipitate, suggesting the presence of mucin. Urea, hæmoglobin, and the salts characteristic of serum are present. Urobilin has also been found.

The expectoration seldom sets in during the paracentesis, but generally after an interval of from a few minutes to half an hour. In rare instances the interval has been prolonged to two hours, and in the case reported by Pepper it was apparently eighteen hours. During the interval the patient generally enjoys a marked feeling of comfort, which is suddenly terminated by dyspnœa and cough. Sometimes the latter begins before the tapping is finished. The cough rapidly becomes more intense, and expectoration increases, the patient at the same time experiencing a terrible sense of oppression, which compels him to sit up in bed. The breathing is intensely labored, the face deeply cyanosed and very anxious, the skin clammy, the pulse rapid and weak. Auscultation reveals the characteristic coarse and fine moist râles of pulmonary œdema.

In mild cases these symptoms subside gradually, the expectoration ceasing after several hours; sometimes, however, not until a day has elapsed. In extreme cases the fluid gushes from the mouth and nose in a constant stream, the patient suffering the most agonizing dyspnœa, and not infrequently dying of asphyxia—drowned, as it were, in his own juices. The amount expectorated varies with the duration and the intensity of the attack; not rarely as much as a pint or even a quart is brought up in the course of one or two hours. Gee's patient expectorated a litre in two hours; Schütz's a total quantity of 1500 c.c.

The duration of an attack is, as a rule, from one to two hours, but in some cases it has been six, twelve, twenty-four, and even forty-eight hours.

On standing, the fluid generally separates into three layers; the upper, whitish and frothy; the middle, opalescent and yellowish; the lower, denser and more viscid and containing a few whitish flocculi. In my own case there were but two layers on standing; an upper, frothy layer, and a lower stratum, twice the thickness of the upper, of opalescent fluid.

ANALYSES OF THE FLUID. I have already detailed the analysis in my own case. For purposes of comparison I give below the few analyses of the pleural and expectorated fluids that I was able to find in the literature. Many are incomplete.

1. Terrillon's case: The pleural fluid contained 1.61 per cent. of albumin; the expectoration, 1.42 per cent.

2. Foucart states that the total solids in the pleural fluids were 7.5

grammes per litre; in the expectoration they were 100.3, the albumin being 93.6 and the mineral salts 6.7.

3. Daremberg and Dujardin-Beaumetz: *a. Pleural Fluid*. Specific gravity, 1020; albumin, 66.88 grammes per litre; urea, 0.58 gramme per litre; hæmatoidin crystals, biliverdin, and a notable quantity of mucosin.

b. Expectoration. Specific gravity, 1010; albumin, 1 gramme per litre; urea, 2.36 grammes per litre; cholesterin and fat.

4. Drivon: *a. Pleural Fluid*. 945.28 parts water, 54.72 parts solids. Of the solids there were albuminoids 48.28, divided as follows: Albumin, 25.69; hydropsin, 22.59; mucosin, a trace; salts, 6.44 per cent.

b. Expectoration. 976.86 parts water, 23.14 parts solids. Of the solids there were 15.87 parts organic and 7.27 parts salts.

5. Laboulbène: *a. Pleural Fluid*. Specific gravity, 1020; total solids, 65 grammes per kilogramme. Of this 0.678 were fibrin; 7.5 mineral matter.

b. Expectoration. The dry residue varied from 16.47 to 24.60 on different days; the mineral matter from 3.53 to 7.8. The albumin was not estimated in the expectoration.

6. Besnier merely states that the quantity of albumin in the expectoration was double that in the effusion.

The analyses, it is seen, show enormous variations among themselves. In some cases the two fluids differed widely; in others they resembled each other very closely in composition. Regarding hydropsin, which, according to Drivon, is a substance characteristic of serous membrane exudates, I was unable to find any reliable data. Its existence is not recognized by physiological chemists of the present day.

Albuminous expectoration occurs especially after tapping in cases of acute effusion, although in my own case the effusion was unquestionably chronic. It has taken place with both left-sided and right-sided exudates and transudates. Of thirty-two cases the effusion was left-sided in eighteen, bilateral in four.

An important point, to which reference will again be made, is that in the majority of instances the amount of fluid withdrawn was inordinately large; and usually, but not invariably, the withdrawal was rapid. In some instances the simple trocar was used; in others, the aspirator. Thus there were withdrawn in the case of Woillez, 5500 c.c.; in that of Marrotte, 5000 c.c.; in Lande's two cases, 3000 c.c. and 1800 c.c., respectively; in Pinault's, 3000 c.c.; in one of Besnier's, 2600 c.c.; in Pepper's, 2400 c.c. Prévost tapped several times in the same case. At the first tapping he withdrew 1000 c.c., and there was no albuminous expectoration; at the second, 3000 c.c. were withdrawn, and the expectoration followed. Subsequent punctures were not attended by any complication. In Schütz's case 2000 c.c. were removed.

In the majority of cases complicating conditions of various kinds existed and probably played a part in the production of the phenomenon. Lande believes that the danger of albuminous expectoration is particularly to be feared when the conditions on the opposite side are such as to lessen the play of the other lung. At *autopsy* the principal change is an œdema of the lung on the affected side; sometimes of both lungs. In cases in which the pleural effusion was of the nature of hydrothorax, heart disease was found. Scriba's case is of interest because at the autopsy a fibrinous coagulum was discovered plugging the bronchi. In the case of Hayem and Tissier there was adherent pericardium.

Various theories have been advanced in explanation of the curious phenomenon of albuminous expectoration. Terrillon classifies them as follows:

1. Perforation of the lung by the trocar.
2. Spontaneous rupture of the lung.
3. Absorption by the lung of fluid remaining after the tapping.
4. Pulmonary congestion, with intense œdema.

The first three assume that the fluid expectorated is a portion of the pleural exudate; the last, that it is a transudate from the bloodvessels of the lung.

I. *Perforation of the Lung.* The identity in appearance of the two fluids and the analogy in composition, which latter, however, is not constant, suggest this view, which was first propounded by Woillez and Marrotte, and was held by Scriba and others. I may also say that when I examined and compared the two fluids in my own case I was at first inclined to this theory; but against such an interpretation are the following weighty arguments:

1. The absence of pneumothorax, which should be present if the lung has been punctured by the trocar.
2. The late onset of the expectoration. If connected with perforation of the lung it ought to set in at once, and not after an interval of half an hour or more from the time of tapping.
3. The absence of hæmoptysis.
4. The fact that albuminous expectoration has occurred especially in cases in which the effusion was very large, and the lung, of necessity, far away from the chest wall.
5. The large amount of expectoration. It is hardly conceivable that a pint or a quart of fluid could, in a short space of time, pass through such a small opening as would be made by the point of a trocar.
6. In some instances the expectoration occurred several times in the same case. Thus the patient whom Behier tapped four times had albuminous expectoration each time; and in the patient of Gee every one of three tapings was followed by albuminous expectoration. It

is unlikely that the lung in these cases was punctured so many times. Moreover, both writers state that, warned by their first experience, they afterward tapped with the greatest care, so as to avoid wounding the lung.

7. In none of the fatal cases was any perforation of the lung found post-mortem. This, to be sure, is not an argument of great force, for it is proverbially difficult to discover perforations in cases of pneumothorax and of empyema rupturing into the lung; but, on the other hand, the argument is not to be held too lightly, since, death being so much more prompt in albuminous expectoration than in pneumothorax or empyema, the chances of finding a perforation should be better.

II. *Spontaneous Perforation of the Lung* is the view defended particularly by Féréol. The excessive rarity of serous effusions perforating into the lung is against this. Furthermore, there is no reason why it should occur just at the end of tapping; and this, together with the non-development of pneumothorax, the failure to find the perforation at autopsy, and the absence of the auscultatory signs of pleurobronchial fistula, is sufficient to dispose of this theory.

III. *Absorption of the Fluid by the Lung*. This is an illogical view, for it must at once be apparent that the absorption or sucking up of the fluid through the stomata of the lung would cause it to enter the lymphatic system and not the bronchi.

IV. *Pulmonary Œdema*. This theory was first advanced by Pinault and was strongly advocated by Behier. It is the one adopted by Pepper, West, and most recent writers. In support of it we have the following arguments:

1. The onset of the expectoration a little while after the tapping is completed.

2. The physical signs of œdema—crepitant and subcrepitant râles—are present.

3. Œdema of the lungs is the chief lesion found in those organs at autopsy.

4. The occasional development of albuminous expectoration in the absence of pleural effusion. Thus Foucart reports a case in which it occurred in a patient with heart disease without pleurisy, and Woillez observed it twice in emphysematous asthma and once in aneurism of the aorta. In his cases the expectoration did not contain any mucin.

5. The occurrence of the phenomenon after tapping in empyema, as in the case of Fraser. The rarity of albuminous expectoration after the evacuation of a pyothorax has been well explained by Curtin, who ascribes it to the fact that, owing to the thickness of the fluid, the abstraction is necessarily slow.

6. The chemical composition of the two fluids tells both for and

against the œdema theory, inasmuch as the fluids sometimes differ and sometimes appear to be almost identical; but, as it is not possible to discover any clinical difference between the two groups of cases, it is fair to assume that, even when the expectorated fluid most closely resembles that from the pleura, its origin is the same as when it differs the most widely.

7. In the majority of cases in which albuminous expectoration occurred the amount withdrawn was excessively large; in many instances, but not in all, the withdrawal was also rapid.

8. As stated by West, theoretical grounds are in favor of this view. The compressed lung is suddenly released from pressure, and there is naturally a great afflux of blood. A congestion follows, for which I should like to propose the name of *congestion by recoil*. It is true that it has been shown that an increase of pressure in the pulmonary circulation is not alone sufficient to produce œdema; but we have in the condition here considered more than mere pressure—we have vessels that, by reason of their prolonged compression and the altered circulatory conditions, have suffered a change in their walls, by reason of which they are rendered more permeable. A transudation occurs into the air vesicles, which finds its way into the bronchi, and is expectorated. It is possible that the negative pressure exerted upon the heart by the expanding lung, intensifying the diastolic relaxation of the organ, and thus embarrassing its action, is a factor in the production of the phenomenon. This negative pressure effect was first adduced by Dr. Mary Putnam-Jacobi in explanation of the sudden syncope sometimes following thoracocentesis. Lande was of the opinion that the œdema was due to the irritation of the air rushing into the expanding lung, while Johnson believed that it was favored by the existence of thrombosis in the vessels, the result of the long-continued compression and stagnation of the blood. This, however, is not tenable, because such a thrombosis would produce infarction, and of the existence of this there is no evidence in cases of albuminous expectoration. Duffin's view of a vasomotor paralysis of the vessels, in consequence of the prolonged compression, is not unreasonable, and helps to account for the congestion; but whether it be through a paralysis or through a nutritional change in the vessel walls, there is, in either case, a relaxation and a sudden afflux of blood to the expanding lung. The subsequent event—the transudation of serum—is best explained on the assumption of an alteration in the walls of the capillaries and small veins, such as Cohnheim has assumed follows prolonged compression.

TREATMENT. The cough is usually so intense that, despite the apparent contraindication of the drug, morphine cannot be dispensed with, at least in the severer cases. It is advantageously combined with atropine. Counter-irritation, in the form of mustard or dry cups,

should also prove of value. Artificial respiration and venesection suggest themselves as theoretically beneficial. The former has had a practical trial in one case, without avail; the latter, so far as I have been able to determine, has not been employed.

CONCLUSIONS. 1. Albuminous expectoration is a very rare complication of thoracocentesis. It is usually serious and sometimes fatal.

2. It consists in the expectoration of a viscid albuminous fluid closely resembling the fluid of serous effusion.

3. The condition is best explained on the basis of an intense congestion and cedema of the lungs (congestion by recoil).

4. The principal cause seems to be either too rapid or too great a withdrawal of fluid.

5. Serious cardiac disease and morbid conditions of the opposite lung, hindering expansion, are predisposing causes.

6. Under all circumstances, but particularly when these complications exist, aspiration should be performed slowly. If the effusion is large the amount withdrawn at any one time should be moderate.

7. In some cases it may be wise to perform several tappings, drawing off a small quantity each time.

8. The treatment consists in counter-irritation, venesection, and artificial respiration, together with the use of morphine if the cough is severe.

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PRIMARY TUBERCULOSIS OF THE LIVER.

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THERE is no organ of the body free from the attacks of the tubercle bacillus, and probably the most frequently involved, if we exclude the pulmonary apparatus, are those of the abdominal cavity. These processes involving the intestinal canal, especially in children, whether it be in the form of primary or secondary ulcerations, have long been recognized and very thoroughly studied. Secondary manifestations are quite common in all organs, and are so frequent that their absence in those dead of tuberculosis is unusual.

Since improvement in our pathological examination, and since we have become more careful in the study of specimens and of cases, with advanced methods of clinical research our knowledge of tubercular lesions has broadened, and many changes thought formerly to be always secondary are now recognized as occurring as primary manifestations in many instances. This applies with especial force to peritoneal tuberculosis and to tuberculosis of those viscera inclosed by the peritoneum. As an example we have the studies of Williams in pelvic disease, his statistics showing that probably 8 per cent. of tubal diseases are due to tubercular infection. The seminal vesicles may in like man-

ner be the starting-point of the disease, the peritoneum or the general system becoming later involved. In some instances the seat of primary infection may be entirely overlooked, or there may be no trace in other instances of the manner in which the organisms have gained entrance into the body.

Although the liver rarely escapes in the secondary or disseminated forms of the disease, observers are a unit as to the infrequency of the disease in this organ as a primary affection; and so true is this that such instances are looked upon as having interest rather from the stand-point of pathologico-anatomic curiosities than any other. The disease is described as occurring in three forms in the liver, though Osler, including the type in which the bile vessels are first attacked, says it may show itself in four ways: (a) General tuberculosis; (b) tuberculosis in the finer bile vessels, which produces tubercles of various sizes, often bile-stained and frequently caseous in the centre, giving the appearance of small abscesses; (c) large, coarse, caseous masses (these being the large tubercular abscesses), and in the form of (d) tubercular cirrhosis.

While it is true that the disease of this organ is rare as a primary affection, and that most frequently organs easily accessible are first attacked, the bacilli may, as stated above, obtain entrance into the blood currents or lymph channels and attack the internal organs, leaving no trace of their mode or place of entrance or primary infection. It has been shown that in the fœtus the liver may, however, be regarded as the primary seat of the affection.

Sabourand, in 1891, showed the liver and spleen of a child, aged eleven days, which were beset with numerous miliary tubercles; the mother suffered from congenital tuberculosis. Another observer has reported a case in a child, aged fifteen days, the disease being, however, so extensive as to preclude post-natal infection. In these cases the infection is probably through the umbilical vein from the placenta, and may be looked upon as primary in character.

Hanot says the disease may be primary, although very rare; and he describes the forms of tubercular hepatitis, viz.: (1) Acute or fatty hypertrophic liver; (2) subacute or fatty cirrhotic; (3) the fat, tuberculous liver, which later is at the limit of latent forms which have a clinical expression, with a slow increase in the size of the organ. The duration of the disease, he says, is from four to twelve months.

In Macaigne and Finet's case of diffuse nodular hepatitis the infection was secondary, the nodular development having its centre in the portal system, through which it was considered that the infection may have occurred.

That this does often occur we cannot doubt when we consider the portal circulation in its connection with the intestinal tract and the

frequency with which the bowel may be the seat of lesions not only secondarily but primarily.

Remembering, then, that the tubercle bacillus, like others, may obtain entrance into the body and leave no trace of its mode or place of introduction; that tubercle bacilli may find their way into the intestinal tract and produce lesions here without disease higher up in the alimentary canal, and that the blood from the intestinal canal passes through the liver by way of the portal circulation, it seems we might find primary hepatic tuberculosis far oftener than we do. Fletcher regards the portal veins as paths of infection, and in the case herewith reported we believe the infection to have taken place in this way, the clinical history and autopsy seeming to bear out the assumption.

The patient, Mr. K., aged forty-four years, was seen in consultation with Dr. Florence Brandeis, first in June, 1901, he having been ill for several months, with almost daily chills, followed by elevation of temperature. This had been diagnosed as malaria, and he was given the usual quinine treatment therefor. The history obtained was about as follows: He had lived in Louisville most of his life, and five years ago moved to New Orleans and engaged in business. During his stay there he had an attack of dysentery, which was quite severe, confining him to bed for two or three months, and from which he did not fully recover until his return to Louisville, about eighteen months ago. He had not been ill before that time, and remained in good health after his return until about five months ago. His family history is good, and there is no history of syphilis or other disease of importance than that above given.

During his present illness he has complained mostly of pain in the abdomen, quite severe at times, especially over the liver; also in the right hypochondrium and in the epigastrium. He has had frequent chills, and fever more or less constant, the temperature range being as high as 103° to 105° F. Profuse night-sweats have been a source of constant annoyance.

At present he is somewhat jaundiced and much emaciated. The abdomen is distended, especially at its lower portion. There is some pain in the upper abdomen, most marked to the right of the left costal margin, about on a line with the eleventh rib. The liver is considerably enlarged, the left lobe extending over to the median line and three fingers' breadth below the border of the ribs; the right lobe extends three inches lower than normal. There is some tenderness over the gall-bladder region; pulse, 110; temperature, 102° F.; skin dry, harsh, slightly jaundiced; stools clayey and odor marked; urine contains bile-pigment; blood examination shows nothing abnormal.

He was sent to the infirmary for observation, we thinking we had to deal with a case of suppuration and adhesions about the gall-bladder, or maybe with a case of hepatic abscess, with localized peritonitis of a slow type. After watching him for several days—the urine and blood being repeatedly examined, although nothing further was thereby discovered—a slight ascites was noticed. We concluded, after the week's observation, that our first diagnosis was incorrect and that the case was probably one of tubercular peritonitis. Exploratory operation was advised and carried out June 15, 1901.

Operation by incision in the right linea semilunaris, at the margin of the liver. The gall-bladder and liver present. There are numerous dense adhesions between the liver and the parietal peritoneum, which are partially separated; the gall-bladder is distended, and there are a few omental adhesions about it; omentum also adherent at the margin of the liver. No pus in the gall-bladder or any stones either in the gall-bladder or ducts. The liver presents numerous small nodules, especially along the lower margin, where the omentum is adherent, which have the characteristic appearance of miliary tubercles. Some enlarged glands are felt posterior to the foramen of Winslow. There is a small cyst on the anterior surface of the liver, about midway between its upper and lower border, which upon aspiration yields a clear fluid. Liver densely adherent to the diaphragm. There is some fluid in the abdominal cavity, and the omentum presents a few tubercles here and there. The intestines are free; no tubercles upon them. No enlarged mesenteric lymphatics. Stomach not adherent. Appendix normal and not removed. All the abdominal organs examined, and nothing abnormal noted.

A portion of the thickened peritoneum over the liver, where it had been adherent, as also a portion of the omentum, was removed for examination by Prof. Carl Weidner, who reported that he found no tubercle, nor were any bacilli found in the fluid from the small cyst which was aspirated. After drying the cavity thoroughly the incision was closed by interrupted buried sutures of catgut, with continuous silk-worm-gut for skin. A post-operative diagnosis of primary tuberculosis of liver was made.

Convalescence normal. Bowels moved on the third day, and since the operation temperature remains normal and sweats have ceased. He expresses himself as feeling decidedly better.

June 29th. Discharged from the infirmary. The next entry in my notes is July 5th, when he was seen at his residence. He has had no sweats or temperature. His appetite has improved, as has his color, and he has gained in strength. He is up and about some each day.

July 29th. I note he is beginning to have some temperature daily; night-sweats returned, abdomen ascitic, feet oedematous; and it is thought he will develop a general tuberculosis.

He remained under the care of Dr. Brandeis, going from bad to worse, until September 19th, when he died of exhaustion, having presented in his later days all the symptoms of a general abdominal tuberculosis. Shortly before death he had several small, hemorrhagic stools, the fecal matter being slightly streaked with blood.

At the autopsy, which was done September 19th, by Dr. L. L. Solomon, the following conditions were noted:

The liver was densely adherent to the parietal peritoneum and to the diaphragm, also much enlarged, measuring sixteen inches in its transverse by twelve inches in its longitudinal diameter. The left lobe was about as large as the right. The organ was congested and full of blood. There are present on the surface as well as throughout the liver, as shown by incision into it, numerous nodules, varying in size from a millet-seed to that of a pea. Many of these nodules have undergone caseation. There is a decided increase in the connective tissue.

The spleen is enlarged, dark red, and full of blood. Studding the surface and throughout the organ are nodules similar to those in the liver.

The kidneys are slightly enlarged and congested.

The omentum is studded with the same variety of nodules noted above.

The intestines are covered with small nodules, especially about the caput coli and the appendix vermiformis. The latter is adherent by its tip at the brim of the pelvis.

The mesenteric glands are enlarged, many of them caseous. In the posterior fold of the foramen Winslowii are several enlarged glands.

There are about three pints of fluid free in the cavity; no other lesions noted.

The process was certainly most advanced in the liver and spleen; next in the glands.

Microscopical examination of the structures—i. e., liver, spleen, glands, and peritoneum, made by Dr. J. E. Hays—showed the nodules to be tubercular and to present various stages of the process, even to caseation.

In reviewing this case and such literature as I have at hand the following conclusions appear to me to be justified:

1. That primary tuberculosis of the liver, though rare, may occur.
2. That the infection may take place by way of the intestine and the portal circulation, the bacilli finding entrance through an ulcer in the bowel (which in this case was at the time of illness some time since), and leave no trace of the place of entrance into the body.
3. That tuberculosis of the liver may infect the other abdominal viscera and also the peritoneum secondarily.
4. That the process may cause a great increase of the connective tissue in the liver (interstitial hepatitis).
5. That the disease results fatally within twelve months.

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ON THE ASSOCIATION OF STONE AND TUMOR OF THE URINARY BLADDER, WITH REPORT OF A CASE.

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DEFINITE reference in the literature to the co-existence of calculus and tumor of the urinary bladder are few indeed. The articles by Rösen, Kolozek, Colley, Swediar, Rochet, and Ohdedar are the only ones in the English, German, and French languages whose titles indicate directly that they are concerned with this subject.

Until recently this subject has received from authors of text-books passing notice only, and even in such works as those of Sir Henry Thompson, Barling, Fenwick, and others, the statements concerning it are very brief. There has been no attempt to show an etiological relationship between the two conditions.

The following is an interesting case of this nature :

Harry F., aged thirty-five years, tailor, American, entered the Cook County Hospital of Chicago, June 9, 1898. The history obtained from his brother is as follows: The patient has had difficulty of micturition nearly all his life. Had scarlet fever two years ago, and has been deaf and dumb ever since. About three months ago he came to his brother complaining of pain in his back and troubled with frequent micturition. Since that time he has lost much flesh and has had constant pain in the back, and during the past month over the pubes.



Vesical calculus, with carcinoma (from above case).

Upon physical examination a large tender mass extending from the pubes half-way to the umbilicus is found. On rectal examination a smooth, firm mass is felt, filling the pelvis. A few cervical glands are palpable. The clinical diagnosis of sarcoma of the bladder was made. The patient died July 8, 1898, thirty days after admission.

Autopsy (Dr. Hektoen) twenty-four hours after death: no changes of special interest are found, with the exception of those in the urinary organs.

The pelvic cavity is filled with a mass composed of urinary bladder and coils of the lower part of the ileum. This mass is adherent to the anterior abdominal wall and covered by adherent omentum. On separating omental adhesions, a soft tumor mass occupying the urinary bladder is broken into. On removing the bladder the entire wall above the trigone is infiltrated with a grayish-white tumor which completely fills the cavity, the mucous surface of which is necrotic, the wall in places being 3 cm. in thickness. In places the wall has been broken through by the tumor tissue, forming subserous nodules, the largest being the size of a hickory-nut. Lying in the lower segment is a large round stone, having upon its surface warty excrescences, and weighing 150 grammes. The mucous membrane of the ureters is slaty in color and roughened. The ureters and pelvis are dilated and contain grayish pus. The kidney substance is atrophied. The capsule is free. The rectum is not involved.

Anatomical Diagnosis. Carcinoma of the urinary bladder. Vesical calculus. Cysto-uretero-pyelonephritis. Adhesive pleuritis. General marasmus.

The microscopical examination of the tumor verifies the anatomical diagnosis.

REMARKS. There are several considerations which lead us to believe that in this case the stone preceded the tumor formation.

1. The much more frequent occurrence of uncomplicated cases of stone than of tumor.

2. The extreme rarity of uncomplicated carcinoma of the urinary bladder at this age.

3. The long-continued urinary disturbances (30 years) and the mildness of the symptoms. It would be unreasonable to suppose that a tumor, particularly a carcinomatous growth, should have existed for so long a period and have given rise to such slight disturbances. According to Albarran, Barling, Hamilton, and others, carcinoma of the urinary bladder proves fatal within three years.

4. The large size and character of the stone. The excellent researches of Pitha show that a phosphate of lime calculus, under the most favorable conditions may increase 20 to 40 grammes (6 to 12 drachms) per year, and a stone of calcium oxalate may, under the most favorable circumstances, increase 4 to 8 grammes (1 to 2 drachms) per year. Thus it would seem that this stone weighing, approximately, 150 grammes (40 drachms), was the cause of the trouble in micturition of which the patient complained for so many years.

From an extensive review of the literature, the following cases of calculus and tumor of the urinary bladder have been collected and tabulated:

STONE AND TUMOR OF THE URINARY BLADDER. 637

| No. | Sex, age, occupation. | Clinical diagnosis. | Kind of calculus. | Kind of tumor. | Other changes. | Seat of tumor. | Authors. ¹ |
|-----|-----------------------|---|---|---|---|------------------------------|-----------------------|
| 1 | M. 42 | Carcinoma vesicae. | Mulberry oxalate (48 gms.) | Carcinoma (squamous-celled). | Left. Hydronephrosis with pyelonephritis; right kidney is atrophic; metastasis in regional lymph glands. | Anterior wall. | Rösen, L. |
| 2 | M. 33 | Urinary calculus. | Mulberry oxalate. | Carcinoma. | Passive hyperemia of kidneys; hypertrophic cirrhosis; adhesive peritonitis about tumor growth. | Anterior wall. | Ibid. |
| 3 | F. 64 | Tumor of the bladder. | "Phosphatic gravel." | Villous cancer. | Right. Pyelonephritis; dilatation of right ureter; muscularis is infiltrated; metastasis in regional lymph glands. | Lower segment. | Watson, P. |
| 4 | M. 51 painter | Stone and tumor of the urinary bladder. | Oxalate of lime coated with phosphates. | Epithelioma. | Left renal calculus with pyelonephritis; metastatic nodules in liver; metastasis in regional lymph glands; pneumonia. | Posterior wall. | Colley, J. N. C. |
| 5 | M. 60 milor. | Stone and tumor of the urinary bladder. | | Carcinoma. | Metastasis in the regional lymph glands. | Anterior wall. | Fenger, F. |
| 6 | M. 65 milor. | Vesical calculus at 14 years. | | Carcinoma. | Right. Chronic nephritis; carcinomatous nodules in right kidney; adhesive pericarditis. | Posterior inferior wall. | Hastington, J. F. |
| 7 | M. 56 | Stone and tumor. | | Squamous epithelioma. | Interstitial nephritis; cirrhosis of liver; cheesy foci in apices of lungs; adhesive pleuritis. | | Hughes, W. E. |
| 8 | M. 47 | | | Carcinoma. | Right. Chronic diffuse nephritis. Left. Kidney substance absorbed; hydronephrosis. | Posterior wall. | Loomis, H. P. |
| 9 | M. 35 | Sarcoma of the bladder. | Weight 150 gms. | Carcinoma. | Cysto-uretero-pyelonephritis; adhesive pleuritis. | Whole interior. | Present case. |
| 10 | M. 50 | | Uric acid size of small apple | Carcinoma. | | Posterior wall. | Birch-Hirschfeld. |
| 11 | M. 30 | Vesical calculus. | | Squamous epithelioma. | Pyelonephritis; metastasis in kidneys. | Lateral inferior wall. | Kolozek. |
| 12 | M. 45 | Large calculus. | | Squamous epithelioma. | | Anterior wall. | Assendelft, E. |
| 13 | M. 47 | | Large stone | Carcinoma. | | Base and prostate. | Ibid. |
| 14 | M. 47 | Large stone | "Huge calculus." | Ulcerative carcinoma. | | Base. | Ibid. |
| 15 | M. 60 | Tumor and stone. | "Phosphatic." | Villous cancer; papilloma removed previously. | Metastases in ileum, head of femur and lung. | $\frac{3}{4}$ inner surface. | Watson, F. S. |
| 16 | M. 54 garden-er. | Tumor of the urinary bladder. | "Phosphatic gravel." | Carcinoma. | Multiple abscesses of kidneys; metastases in regional lymph glands and lungs. | Anterior inferior wall. | Bode, E. |
| 17 | M. 69 hotel-keeper. | Stone and cancer of bladder. | "Several small calculi." | Carcinoma. | Right. Chronic diffuse nephritis; cirrhosis of liver, adhesive pleuritis. | Base and prostate. | MacGowan, G. |
| 18 | M. 35 | Stone and cancer. | Phosphatic | Carcinoma. | | Left inf. wall. | Fenwick, E. H. |
| 19 | M. 79 | | Phosphatic | Carcinoma. | | Base and prostate. | Johnson, R. |
| 20 | M. 55 | Tumor. | Phosphatic | Cancer. | | Anterior wall. | Douglas, J. |

¹ For references see alphabetical list at end of article.

638 STONE AND TUMOR OF THE URINARY BLADDER.

| No. | Sex, age, occupation. | Clinical diagnosis. | Kind of calculus. | Kind of tumor. | Other changes. | Seat of tumor. | Author. ¹ |
|-----|-----------------------|-------------------------------------|---------------------------------|---|---|-------------------------|----------------------|
| 21 | M. 32 | Cancer of bladder. | Phosphatic | Epithelioma (papilloma removed previously). | | Right lateral wall. | MacGowan, G. |
| 22 | M. 57 | | | Carcinoma. | Metastasis in kidneys; double hydronephrosis atheroma of ascending aorta. | Entire wall. | Haake, O. |
| 23 | M. 43 merchant. | Tumor and stone. | "Brownish," wt. 40 gms. | Small r'd-celled sarcoma. | | Inferior wall. | Krause, L. J. |
| 24 | M. 59 | Stone and tumor of urinary bladder. | Uric acid, wt. 20 gms. | Myxosarcoma. | | Diffuse. | Obdedar, M.N. |
| 25 | F. 50 housewife. | Cystitis. | "Gravel and small stones." | Medullary sarcoma. | Sacral lymph glands are enlarged. | | Williams, W.R. |
| 26 | M. 6 | Calculus and tumor. | "Small impacted." | Myxosarcoma. | Double hydronephrosis | Diffuse. | Southan, F. A. |
| 27 | M. 50 clergy. | | "Phosphatic concretions." | Sarcoma. | | Base of bladder. | Skilern, K. R. |
| 28 | M. 67 | Stone and tumor of urinary bladder. | Uric acid. | Papilloma. | | | Thompson, Sir H. |
| 29 | F. 31 | Tumor of bladder. | Phosphate of lime. | Papilloma. | | Base. | Norton, A. |
| 30 | M. 38 laborer. | "Phosphatic gravel." | "Phosphatic gravel." | Papilloma | | Anterior wall. | MacGowan, G. |
| 31 | M. 47 | Stone and tumor of urinary bladder. | | Fibro-papilloma. | | Anterior inferior wall. | Ibid. |
| 32 | M. ... | Tumor. | "Stony concretions." | Papilloma. | | Lateral inferior wall. | Wendell, W. |
| 33 | F. 45 housewife. | Tumor. | "Phosphatic gravel." | Papilloma. | | Left inferior wall. | Vander Veer, A. |
| 34 | M. 29 | Tumor and calculus of bladder. | Oxalate & phosphate of calcium. | Polypus. | | | Thompson, Sir H. |
| 35 | F. 43 | Tumor. | Phosphatic | Polypus. | | Inferior wall. | Hill, B. |
| 36 | M. 56 | Stone and tumor. | "Phosphatic concretions." | "Villous." | | Lateral inferior wall. | Thompson, Sir H. |
| 37 | M. 67 | Stone. | Uric acid, wt. 15 gms. | | | Lateral wall. | Ibid. |
| 38 | M. 67 | Stone of bladder. | "Walnut-sized." | "Pedunculate." | | | Schrey, H. |
| 39 | M. 31 | Ulcer of bladder. | "White stones." | "Ulcerative." | | Posterior wall. | MacGowan, G. |
| 40 | M. 62 weaver. | Stone. | "Gravel." | "Fungus." | Right. pyelonephrosis with eight renal calculi; nodules in liver. | | Douglas, J. |
| 41 | F. 79 | Cystitis. | Phosphatic | "Fungus." | | | Ibid. |
| 42 | | Urinary calculus. | Phosphatic | | | Base. | Anderson, W. |
| 43 | M. 53 | Encysted calculus. | Phosphatic | "Malignant." | | Anterior wall. | Post. |
| 44 | M. 40 | Calculus. | Weight 40 gms. | "Fungus." | | Base. | Swedjar, F. |

¹ For references see alphabetical list at end of article.

In this series of 44 cases, 37 occurred in males; 6 in females; and in one the sex is not given. The average age of the cases is 48 years. The ages range from 6 to 79 years. In two cases the age is not given. The following table shows the cases by decades:

| <i>Age periods.</i> | <i>Carcinomas.</i> | <i>Sarcomas.</i> | <i>Benign tumors.</i> | <i>Total.</i> |
|-------------------------|--------------------|------------------|-----------------------|---------------|
| 6 to 20 years | 0 | 1 | 0 | 1 |
| 21 " 30 " | 1 | 0 | 1 | 2 |
| 31 " 40 " | 4 | 0 | 3 | 7 |
| 41 " 50 " | 6 | 3 | 3 | 12 |
| 51 " 60 " | 7 | 1 | 3 | 11 |
| 61 " 70 " | 3 | 0 | 4 | 7 |
| 71 " 80 " | 1 | 0 | 1 | 2 |
| | <u>22</u> | <u>5</u> | <u>15</u> | <u>42</u> |

The clinical diagnosis was either not made or not given in 8 of the cases. Of the remaining 36, the diagnosis of stone was made 10 times, tumor 10 times, both calculus and tumor 13 times, cystitis twice, and ulcer of the bladder once. The mulberry or oxalate of lime and uric acid calculi each occurred 4 times; the phosphatic calculus 12 times; and in the remaining cases no mention is made of the character of the stone.

A microscopical examination was made in all but three of the cases of carcinoma and sarcoma, and in these the malignancy is verified by metastases. This gives 22 carcinomas; 5 sarcomas; 5 papillomas; 2 polypi, and 2 fungus growths, while the remainder are given as "malignant," "villous," "ulcerative," or undesignated. In this series the tumors are all primary, except possibly four cases, reported respectively by Assenfeldt, Johnson, MacGowan, and Hastings. In the first three, the prostate was simultaneously involved, and in the latter it was doubtful which was primary, the carcinoma of the bladder or that of the right kidney.

A close study of the clinical history and post-mortem findings in these cases shows that the stone was primary in 18 and the tumor in 8 instances, while in the remainder it is impossible to state with any degree of certainty which of the two conditions was primary.

Other lesions besides those in the bladder are given in twenty instances. Of these, ascending uretero-pyelonephritis with dilatation is the most common. Metastases in the neighboring lymph glands occurred in a considerable number of the malignant tumors. Metastatic nodules occurred in two instances each, in the lungs and liver, and three times in the kidneys. It is quite generally known that especially distant metastases in carcinoma of the urinary bladder are not so frequent as in carcinoma elsewhere (Barling), and if it occurs at all it occurs late in the disease.

In 21 cases the seat of the tumor is given as the lower segment; in 9, the anterior wall; in 5, the posterior wall; in 3, the lateral walls; and in 3 as diffuse; while in the remaining cases no mention of the location is made.

What relation is there between calculus and neoplasm of the urinary bladder? Is the combination merely accidental? Does tumor enhance the likelihood of the development of calculus? Or, finally, does calculus by its influence increase the liability to the development of new growth?

The number of cases of calculus and tumor that I have collected seems altogether too large to be regarded as the result of accidental coincidence, and especially so when the rarity of tumors of the urinary bladder as a whole is considered. Küster says that tumors of this organ constitute only one-half per cent. of all tumors. Heilborn and Hasenclever found in the Berlin Pathological Institute only 10 primary carcinomas of the urinary bladder in 20,000 post-mortems from 1859 to 1880. According to the statistics of Ditel, Zuckerkandl, Guyon, Bozy, Israel, Fenwick, Keyes, Hamilton, Podrazki, and others, carcinoma occurs in a little over half of all tumors of the urinary bladder. Hence, since tumor occurs so infrequently, it would seem that the number in which the combination of calculus and tumor exists is too large to be explained as accidental.

The following arguments favor the view that tumor increases the liability to the formation of calculus:

1. In 8 cases of our series there is no question but that the calculus was probably secondary to the tumor.

2. Tumors, especially of the malignant type (Barling, Fenwick, and others), are not infrequently found incrustated with phosphatic material.

3. Villous growths are especially liable to cause calculi, because a poorly nourished projecting villus while yet attached, or when torn off, may afford a nucleus, much the same as a foreign body around which urinary salts are deposited.

The following points may be considered as evidence that vesical calculus is a factor in the etiology of tumors of the bladder, by causing primarily a "mechanical irritation," followed by inflammatory hyperplasia, the "inflammatory tumor," of Leichtenstern, which may be transformed into true neoplasms, as shown by Wendel, Zuckerkandl, and others.

1. In over half of the cases it appears that the stone was primary in point of time.

2. Inflammation of the urinary bladder caused by irritation other than that produced by a calculus has been found to cause an increased liability of new growths. Wendel and Leichtenstern have shown that this not infrequently occurs in individuals who are subject to inflammation of the urinary tract from the constant inhalation of the poisonous vapors from aniline dyes. Albarran, Bernhard, and others report cases in which tumors undoubtedly resulted from chronic hyperplastic inflammation caused by the parasite *Bilharzia hæmatobia*. Störk claims

that in one of his cases a carcinoma developed from a cystitis cystica. Fagge believes that a carcinoma was the result of irritation from the use of the catheter for thirty years. Kaufman, Thoma, Watson, Rösen, Colley, Zuckerkandl, Rochet, and others believe that chronic inflammation is present in the majority of all tumors of the bladder, and my study of reported cases corroborates this view. Hence, it is permissible to conclude that if inflammation caused by chemical, bacterial, and parasitic causes enhances the likelihood of tumor growth in the urinary bladder the mechanical irritation from a calculus may have the same effect. Orth goes so far as to say that papillary growths are the result specially of the chronic inflammation produced by stone.

3. Tumor of the urinary bladder uncomplicated by stone occurs more frequently in the lower segment, (Fenwick, Keyes, Barling, Küster, Haake, Birch-Hirschfeld, Hasenclever, and others). This is also apparent in this series. In the uncomplicated cases the posterior wall is affected more often than the anterior, the favorite site being at or near the orifices of the ureters, (Keyes, Fenwick, and others), while in the cases with calculus we find the anterior wall to be the seat of the tumor in nearly twice as many instances as the posterior wall. In explanation of the more frequent involvement of the lower segment different views are held, one being that the tumor arises from the prostate; but against this is the fact that in this series, as well as in all primary urinary bladder tumors, the more frequent involvement of the lower segment prevails alike in both sexes. A more plausible explanation, and one held almost universally, is the greater irritation of this portion both by chemical and mechanical influences. The more frequent involvement of the anterior wall in this series, where calculus is present, in our opinion, must be ascribed to the influence of the stone.

4. The large number of cases in which the calculus was primary in point of time and the fact that both calculus and tumor occur more frequently in the male sex and nearly at the same age may be taken to indicate that the calculus was not without etiological influence in the development of the tumor.

5. Since in 90 to 95 per cent. of primary carcinoma of the gall-bladder, biliary calculi are present (Tillman, Siegert), analogy would lead us to expect a somewhat similar relation between stone and tumor in the urinary bladder.

Hence, the conclusion may be drawn that, contrary to the views of Neelsen, Busse, and others, an etiological relationship between calculus and tumor is not to be denied altogether, and calculus seems to favor the development of tumor in a larger percentage of cases than tumor favors the development of stone.

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A CASE OF STOMATITIS GANGRENOZA (NOMA).

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THIS disease is frequently called noma, and sometimes cancrum oris. It is characterized by a gangrenous destructive process located on the cheek. Although the left cheek is the favorite site of the disease, it can frequently be found on both cheeks. The writer has met with children suffering from this disease on the right cheek. Girls are more liable to noma than boys. It is usually secondary to some infectious disease, and has been known to follow typhoid fever, smallpox, scarlet fever, measles, and pertussis and allied infectious disorders. We must therefore assume that the infectious diseases are predisposing factors in the development of this disease.

Some authorities claim that noma frequently is a sequel to infectious diseases.

The process usually commences on the gums or the inner portion of the cheek, and spreads very rapidly to the adjacent tissues. Thus it is that it will destroy the inner portion of the cheek and spread to the outside, causing similar destruction to the healthy tissues. From the nature of the method of spreading it appears to be of a specific nature. Whether or not a specific micro-organism causes this disease has not yet been definitely determined. We know, however, that it commences similarly to a diphtheritic process and spreads in the same manner. Weak children, as those above mentioned, that have passed through severe infections, are the ones usually attacked with this disease.

Symptoms. The cheek will appear swollen, hard, and cedematous to the touch, the cedema causing such swelling that frequently the eye of the affected side cannot be opened. There is a decided fetor to the breath, which is often the first symptom noticed. The disease spreads very rapidly from the gums to the cheek. Frequently the teeth will loosen and fall out. The latter is frequently caused by the previous administration of mercury. Thus it is that great care should be used in giving mercury to children.

That it is not an inflammatory disease can be seen by the fact that the temperature is rarely or never above normal. The swelling can best be felt by opening the mouth and grasping the cheek between the thumb and forefinger. The skin over the induration is frequently mottled with purple spots resembling ecchymoses. The appetite is diminished, partly due to the fear of pain caused by chewing.

Some authorities state that children so affected have diarrhoea. Forchheimer believes that hemorrhages rarely occur, owing to the bloodvessels being filled with thrombi.

When this gangrenous mass discharges we will find a dirty, fetid saliva, with threads of broken-down tissue. The cervical glands in the immediate vicinity are always found enlarged. In severe cases it is not rare to have the parts ulcerate and even perforate the cheek after several days. When the disease extends inward, not only does pericostitis occur, but necrosis of the jawbone has been noted. When the disease is as malignant as has just been described, then subnormal temperature, possibly delirium, may complicate the condition. The disease may extend to the lungs, causing a gangrenous infiltration. When the gangrene affects the genitals in girls, then a serious prognosis must be given.

Starr maintains that noma makes its appearance uniformly at one point on the cheek, and is unilateral, which suggests a localized causative lesion. The most natural theory, that of embolism of a large arterial branch, due to weakness of the cardiac muscle or increased coagulability of the blood—effects of the primary disease—is untenable, because, with the given conditions, emboli ought, at least occasionally, to be found in other positions, which does not happen. It is necessary to look rather to the nerves—namely, the trifacial, the facial, or the vasomotors. That the gangrene is due to a lesion of one of these seems to be borne out by experiments. Thus Magendie found that division of the trifacial in dogs caused destruction of the corresponding eyeball, and half of the tongue became dry, brown, and fissured, the gums spongy and hemorrhagic, and the teeth loose. "In animals tenacious of life—the batrachians, for example—the soft portions of the face are cast off in shreds, just as in spontaneous gangrene. After three or four weeks only one-half of the face remains."

A variety of bacteria can be found at the seat of lesion, but their presence has no etiological significance. The body of a child dead from noma has a gangrenous odor and decomposes quickly; the skin is shrivelled and the face and the feet are œdematous. The gangrenous parts are converted into a blackish-brown mass, and the maxillary bones are naked, brownish in color, and brittle. The nerves, when examined microscopically, are yellowish in color but unaltered in structure, and the bloodvessels are thickened and filled with thrombi. In the uninvolved parts of the cheek there is a dense exudation, while the palate, tongue, and tonsils are swollen and covered with black scales and crusts. The lungs are the seat of hemorrhagic infarctions, lobular or metastatic lobar pneumonia, and sometimes gangrene. The intestines are catarrhal. Evidences of the primary disease may also be present; for example, the lesions of typhoid fever or dysentery.

The following case will illustrate the condition described :

Elise G., aged seven years, was seen by me in January, 1900. The child had complained of severe headache for three or four days, and was very feverish. Her mother became alarmed because of persistent vomiting. She stated that the child vomited at least six times in twenty-four hours. She complained of feeling fatigued and had pains in her arms and legs.

Small doses of quinine were given the child, but did not seem to relieve the present condition.

The child was nursed for ten months, and was a strong baby up to this time; dentition commenced at the seventh month; the child's muscles and bones were well developed; there were no evidences of rickets; the first two years were passed without any sickness except an occasional attack of constipation. The child walked at the end of the first year and commenced talking at its fourteenth month. Twenty teeth—"milk teeth"—appeared at the end of two years. The child had measles in its third year, which left a bronchitis; the mother states that this same cough recurs every winter. The child has had whooping-cough, lasting four months, which was so violent that it had epistaxis almost every day for one month. This whooping-cough was so severe that, in addition to the nose-bleed, the child vomited almost continuously. From loss of sleep, in addition to the above-named symptoms, the child commenced to emaciate. This was at the end of her fifth year.

She lost twelve pounds in two months, and the mother states that since that time she has been very puny and delicate. There is also a hernia directly traceable to the violent paroxysms of cough.

The mother suspected the child was suffering from malaria, or possibly an attack of grip. When the child was undressed an eruption was found all over the body, which was that of typical scarlet fever. The throat was filled with evidences of pseudo-membranous patches which were distinctly scarlatinal in character. The temperature was 103.4° F., taken in the rectum; pulse, 128; respiration, 22. The child was put to bed and an expectant plan of treatment ordered, in addition to a very light liquid diet consisting of soup, milk, butter-milk, broth. Nothing else was allowed; no solids were given. For the thirst I ordered orange-juice and apple-sauce. Small doses (wine-glasses) of citrate of magnesia were given for their laxative and diuretic effects.

Desquamation followed in the second week in the usual manner. The urine showed traces of albumin in the second week, which increased until that time—six pro mille, according to Eschbach's albuminometer—hyaline and epithelial casts were found in great numbers. There were also large quantities of blood-corpuscles visible under the microscope. The urine was quite red from the blood that it contained. At the end of the third week there was quite an anuria. This latter condition was relieved by the application of several dry-cups over the region of the kidneys. Five to ten grains of diuretin internally were ordered every four hours. Citrate of potash was given, five-grain doses combined with large quantities of apollinaris and lithia water. After three weeks of patient treatment the child recovered.

The heart sounds were not only very feeble, but thready, and a loud,

blowing, hæmic murmur was audible, which was attributed to the anæmic condition. Iron was given in the form of the syrup of iodide of iron; hypophosphites were also administered as restoratives. Convalescence lasted in all until April, a period of almost three months from the time of the child's first illness. About this time she complained of pain in the gums and on the cheek while chewing. Later, the foul breath attracted attention. At first this condition was attributed to the teeth, but a dentist who saw the child found the teeth and gums healthy. The ulceration, which had now become quite marked, from the size of a silver dollar, spread with remarkable rapidity.



Its color was that of a dirty, blackish-gray, and had purpuric spots scattered around the edges of this ulceration, resembling subcutaneous hemorrhages. On examining it considerable fluid, which was very foul smelling, exuded on pressure. Antiseptic lotion, consisting of 50 per cent. peroxide of hydrogen diluted with water, was ordered as a mouth wash. The child was told to rinse the mouth every half-hour, especially after eating. The gangrene extended to the outside of the cheek, involving, as can be seen by the illustration, almost the whole cheek. The picture was taken after the child had had its mouth and its cheek thoroughly cauterized by using the Paquelin cautery. Ichthyol was applied in the following manner: R.—Ichthyol and lanolin. M. ft. ungt. S.—Apply over the whole of the gangrenous surface by rubbing

the parts thoroughly, the same to be repeated at least three or four times a day. The ichthyol seemed to serve remarkably well in this case. The same was continued for about three weeks, when the child was discharged as cured.

A very instructive case is reported in the *Archiv für Kinderheilkunde*, 1898, p. 245, by Dr. A. Klautsch. Ranke was the first to report micro-organisms in this gangrenous mass; later Schimmelbusch reported a case of noma in the *Deutsche medicinische Wochenschrift*, 1889. This latter author found short rods with rounded edges, frequently in pairs, almost in pure culture, in the necrotic masses and in the necrotic tissue.

Cultures made on agar produced a growth of colonies which resembled porcelain. They also grew in large numbers on coagulated ascites fluid. The size and form of the bacilli depend on the nutrient medium on which they were planted. Their staining was rather difficult. When inoculated with necrotic tissue of pure cultures, rabbits, mice, pigeons, and chickens showed circumscribed necroses about the size of the above, which healed in about three days. In addition to the

above-named bacilli, Nicolaysen's investigations on two cases of noma showed that there are large masses of both bacilli and cocci present. The bacilli, while present in the necrotic tissue, frequently infected also the healthy parts. The cocci were found one centimetre distant from the necrotic tissue and in the lymph channels.

Cultures taken from the gangrenous and adjacent parts showed a coccus which was a non-pathogenic staphylococcus and a polymorphous non-pathogenic bacillus which resembled the diphtheria bacillus. Thus it appears that we are hardly in a position to state that there is a specific micro-organism which is a causative factor of noma. We are rather inclined to the belief that there are several bacteria causing this putrid process. The latter interferes with the nutriment of the parts, causing necrobiosis and consequent destruction of the tissue so invaded.

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AN EXPERIMENTAL STUDY OF THE EFFECTS OF CHANGE OF COLOR UPON PIGMENT BACTERIA.¹

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As is well known, nearly all bacteria are devoid of color, appearing at most when colonized in pure cultures as whitish or yellowish-white masses. There are, however, a number of species which when massed exhibit marked, and, under certain conditions, very beautiful, though quite variable, coloration. These are known as the chromogenic or pigment bacteria, and are divided by Breyerinck and Schroeter into three groups: the "chromoparous," comprising those in which the

¹ Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, at the December, 1901, meeting.

pigment-material consists of numerous color-granules surrounding a colorless germ-cell; the "chromophorous," those in which the protoplasmic matter of the cell is pigmented; and the "parachromatophorous," those in which the color-granules are situated in the cell-wall.

As Fischer says: "In all probability the coloring matter is of importance physiologically only where it is bound up with the protoplasm. In all the chromoparous bacteria the pigment is an excretion merely, and, as might have been expected, chemical and spectroscopic analyses fail to show that it has any connection with assimilation."

Since the special form of germ-material employed in the following experiments has been studied solely from a color-point of view (the author being well aware of its color behavior to differences of environment, etc.¹) the use of a chromogenic basis of terminology is deemed justifiable in this contribution to the color department of ophthalmology.

It is a matter of common observation that some of the species of the genus *chromatium* of the colored forms (the so-called "purpurbacteria") of the sulphur micro-organisms give a reddish tinge to water, particularly during the spring and the autumn months. This tinting is found to be more pronounced in situations where the light-stimulus is the strongest. At first sight, this fact would lead to the belief that light is a necessity for such germs. The existence, however, of the main group of sulphur bacteria, for example, depends, as has been shown by Winogradsky, upon the intaking of sulphuretted hydrogen and oxygen; this particular form of thiobacteria possessing in such situations, through the agency of a color material (known as "bakteriopurpurin"), an additional power of assimilating carbonic acid for both energy-making and metabolism-product formation. Here the pigmented sulphur masses situated in the outer layers of the protoplasm of the bacterium are used for physiological purposes. These conditions are also true for the *bacillus virens*.

Other chromogenic bacteria, notably the *bacillus violaceus*, a species in which the pigment granules are generally found in the walls of the germ cell, lose their characteristic tint and become colorless should oxygen be taken from them.

The *bacillus prodigiosus* offers an excellent example of the chromoparous type of micro-organism. The red tint of its colonies is very pronounced, particularly if they appear upon such culture media as milk.

Being aware of the fact that many of the pigment bacteria colonizations become etiolated and changed in tint when the germs are removed from light stimuli, the author determined, as the result of the accidental

¹ The author has, in a measure, completed a number of studies upon the behavior of aggregations of aerobic and anaerobic bacteria toward fixed prismatic colors, the work being based upon Marshall Ward's classic experiments.

observation of a series of definite differences of coloration exhibited by two similar cultures grown under different color media, to make a number of experiments with the three types of chromogenic bacteria above mentioned. Some two years ago, in association with Dr. Napoleon B. Boston, bacteriologist to the Philadelphia Hospital, he commenced a methodical study of the effects of the interposition of transparent sheets of definitely gauged colored glass upon the coloration of germs that have received their designative names from their characteristic hues. A pure culture of every bacterium that was to be employed was obtained care being taken to select only those colonizations that had been grown under the optimal conditions for the particular micro-organism in question. Well-selected color-sheets of known ratios of pure red, pure green, pure blue, and pure yellow, chosen from definite primary tints in Radde's color-scale, were so arranged over specially contrived incubating boxes that the cultures used (kept at uniform temperatures) were uninterruptedly and yet coetaneously exposed to certain color-values for definite periods of time. Each species of germ experimented with was first studied and sketched after it had been grown in the dark and after it had been cultivated beneath an ordinary transparent area of clear glass. Some of the same germ material was grown separately in different media beneath the variously tinted sheets. Care was taken to avoid any loopholes of uncertainty and to see that the germ material was free from contamination. To be doubly certain of the results, fresh cultures in strange environments were studied with new apparatus and in conflicting and recurrent ways.¹

In this brief communication² just sufficient of the work to be of interest to the ophthalmologist, *i. e.*, a few of the most striking of the color-changes noticed in the series of experiments, has been considered. This has been done with a desire to lessen the length of the article and to place enough of the data of the general findings that may be of interest to the practitioner in ophthalmology in a position where he

¹ The author desires to express his gratitude to Dr. Alexander C. Abbott for his many kind suggestions, and to Dr. Ella A. Hackett for most painstaking research work. He also wishes to thank Messrs. Wall & Ochs for careful selection of proper color-sheets, and Mr. Samuel T. Fox, of Queen & Co., for the loan of especially adapted physical apparatus and a set of Radde's International Color-scales.

To Mr. Frank B. A. Linton, Miss Alice B. Cruice, and Miss Mary A. Koonce credit must be given for exquisite art work, without which much that has been accomplished would have been irretrievably lost. To Coppen Jones' translation of Alfred Fischer's remarkably interesting and fascinating work "*Vorlesungen über Bakterien*," much of the enthusiasm that has constantly pursued the author during the latter part of the researches is due.

² A paper bearing in a new and novel manner upon this subject was to have been read before the June, 1901, meeting of the Section of Ophthalmology of the American Medical Association, but on account of the author's inability to be present at the meeting it was withdrawn. It will, however, be reserved for incorporation in a more extended communication upon the general subject.

will be the more certain of finding them.¹ A system of color nomenclature that can be understood in any part of the civilized world has been consistently employed throughout all of the studies. These results, obtained in this manner, will give the reader an opportunity to have before him possibly some new and not uninteresting facts upon the still many moot points in the question of natural color, normal and subnormal color-perception, etc. At least the author hopes that the reader may have thus given him an inducement for further and more comprehensive study of the question which may be productive of observations which will be advantageous to himself and useful to the world.

A few of the most interesting results are as follows :

1. *Bacillus Prodigiosus*. This chromoparous species of germ grown on agar-agar in the dark gives a tint of carmine-zinnober which is represented in Radde's color-scale by letter h, plate 29, card 10. Grown on the same culture medium under a pure blue represented by letter h, plate 19, card 7 (Radde's color-scale), the growth assumes a purple-violet hue, shown by letter l, plate 24, card 8; while the borders appear carmine-gray in tint, as shown by letter m, plate 42, card 14. The germs grown on agar-agar under a pure red (letter m, plate 1, card 1, of Radde's scale) give a peculiar carmine-purple tint shown by letter d, in plate 27, on card 9 (Radde); and at times the pigment massing becomes nearly colorless. A change of the culture medium to blood serum with the constant exposure of the germ to a pure green or cardinal tone, "grasgrün" (Radde, letter m, plate 13, card 5), has the effect of changing the colonization tint to a purple-violet, shown by letter i, plate 24, of card 8 of Radde.

2. *Bacillus Virens*. This beautiful sap-green species (shown by letter m, plate 14, card 5, in Radde's scale), like the sulphur-bacteria, has the pigment matter located in the protoplasm of the cell. Grown in the same situation and under similar conditions on agar-agar, under pure blue (see tint above), it, at times, becomes so greatly bleached that the germs assume a tint that falls to the letter u in the same plate on the same Radde card. Grown on blood serum and placed under the same pure blue, the characteristic naming tint of the germ disappears, it changing its hue to a light brown, as shown by letter s, plate 33, of Radde's card 11. Grown under red (*vide* tint above), on the same serum, it again becomes tinted deeply green, rising to letter f of plate 14, of Radde's fifth card.

¹ The author is arranging a series of papers upon the relative virulencies of the differently tinted colonies of the same bacterium; the seasonal, the altitudinal, and the climatic differences of coloration in the chromogenic forms of bacteria; and the selective color-points of greatest and least development, growth, and sporulation of paratrophic and metatrophic bacteria, etc.

3. *Micrococcus Rosans*. This little rose-colored sphere grown in the dark has its coloration degree indicated by letter m, plate 30, card 10 (carmine-zinnober), in Radde's color-scale. Grown on the same culture medium under pure red exposure (*vide* tint above), it quite frequently loses nearly all of its color characteristics, passing to q in the same plate of the Radde color series. The change made when the germ is grown on agar-agar under pure blue (*vide* tint above) is so slight at times as to be almost unnoticeable. Not infrequently, after the germ has grown on agar-agar under pure blue and pure green exposures, and even in the dark, it becomes deeper in tint than when it is grown under pure red.¹

4. *Bacillus Indicans*. This short capsulated rod, which, as shown by Alvarez, produces indigo by fermentation with the glucoside indican, appearing in its characteristic tint when grown on agar-agar in the dark, becomes tinted to a delicate orange-zinnober hue (letter s, plate 3, card 1, of Radde) when it is grown on blood serum under a constant exposure to pure red.

5. *Bacillus Pyocyaneus*. The characteristic aggregation of this germ when grown in the dark is markedly changed when the micro-organism is placed in different media and is given varying color conditions. Two examples, both taken from blood serum cultures, illustrate this very well. Grown under the pure red of the series, the pigment becomes orange-yellow (letter m, plate 6, card 2, Radde) in tint; and grown under the pure blue of the series it fades to v of the same color-plate.

6. *Sarcina Lutea*. This greenish-golden species of the genus sarcina, with its curious bale-shaped colonies of cells, obtains its characteristic coloration (letter q, plate 8, card 3, Radde series) when it is grown in the dark, and bleaches to a light green-yellow (letter u, plate 9, card 3, Radde) when it is grown under the same conditions on blood serum. Similar though very delicate differences of color intensity, which are almost imperceptible at times, also manifest themselves when the germ is grown on agar-agar under pure red, pure blue, and pure green, these changes being even less noticeable when blood serum is used.

In addition to these series, which are of sufficient number to illustrate the points to be evolved in this communication, the author has made a great number of experiments upon the behavior of the bacillus janthinus,² pink yeast, the staphylococcus aureus, etc., obtaining not only both positive and negative color-changes, but other results which at first seemed irreconcilably curious and bizarre.

¹ These apparently diverse results were later accounted for by a study of the germ's food-stuff. This study will be the subject of a further and more extended communication upon bacterial coloration.

² This germ gives the most remarkable difference of coloration when it is grown upon different media.

CONCLUSIONS. 1. Color-changes both of kind and of intensity take place in and around many chromogenic bacteria when such micro-organisms are placed under different color-conditions.

2. Rehabilitation of chromogenic bacteria into old color environments after having later obtained a new color-value under any particular color-condition, is quite frequently accompanied, sooner or later, with a return of the germ's color-equivalent to that which it primarily held while in its original situation.

3. Differences of color-conditions in pigment-bacteria most probably signify in part relative changes in the various methods of obtainance of sustenance, peculiarities in the kinds and the ratios of foodstuffs, and irregularities in the character of resulting excreta; each species of color-bacterium exhibiting its chromogenic change in a typical and relevant manner, a behavior that has its determining effect upon the very life of the germ itself.

4. The naming of bacteria by specific coloration is only of value when the actual habitat of the micro-organism is understood.

5. As a postulate conclusion based upon these facts, it is most certain that all living faunal and floral color-changes of true objective type are expressions of biochemical peculiarities taking place in and around such organisms—a mere difference of molecular motion as it were in part dependent upon the relationship existing between active life force and co-existing conditions.

ON THE EFFECT OF SEVERING AND OF MECHANICALLY IRRITATING THE VAGI.

BY GEORGE W. CRILE, M.D.,
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PRELIMINARY REMARKS. In operations on the neck not infrequently the dissection is carried to the vagus, and occasionally, especially in a case of malignant disease, the question of removing a portion of this nerve or subjecting it to injury arises. In the literature of this subject the clinical reports of the effects of mechanically irritating, dividing, or resecting are much at variance.

REVIEW OF LITERATURE. Fontana resected a portion of the pneumogastric on twelve rabbits, three of which died.

Longet found that dogs usually survived this operation. He found that roughening of the voice followed and an increase in the frequency of respiration. There was emaciation and repeated vomiting, with diminution rather than increase of appetite.

Fano reported a case of the accidental resection of the right vagus

in a case of malignant disease adherent to the larynx. During the operation the patient was inclined to cough. In the evening after the operation nothing in particular was observed except that the voice was a little muffled. The following day there was difficulty in respiration and a cough. On the third day there was an increase in the cough and the voice had become more muffled. Five days after the operation there was chill, followed by fever and delirium. Respiration was normal. Upon auscultation no congestion of the lung was discovered. Coma succeeded the delirium; the pulse was full and frequent. During this time the dressings were torn from the wound, causing hemorrhage. Death occurred upon the sixth day. At the autopsy no metastatic abscess was found. Both lungs were a little engorged at the base and posteriorly. The stomach was of normal volume. The vagus on the right side had been removed in the course of the operation. The right hemisphere of the brain presented a vascularization more pronounced than the left. On the right side of the larynx there were some portions of the cancer that had not been removed. The exact cause of death is not stated.

Keppler reported the following case: H. K., a weaver, aged thirty years; diagnosis, sarcoma of the neck. Some difficulty was experienced in removing the tumor from its capsule, involving by extension considerable healthy tissue. Lying external to the common carotid artery a strand, about the thickness of the vagus nerve, whose position could not be ascertained owing to displacement of the parts as a result of the growth, was observed. This was carefully dissected away, and proved to be the obliterated jugular vein. After removal of the growth search was made for the vagus, which was found to have been severed. One hour after the operation the pulse was 112, regular; respiration, 36; temperature, 38° C. At 5 P.M., seven hours after the operation, the pulse was 128; respiration, 24. Eleven hours after the operation the pulse was 112; respiration, 30. The following day the pulse was 96; respiration, 24 to 26. Patient made a recovery without any untoward symptoms. Laryngoscopic examination showed epiglottis and aryteno-epiglottidean folds to be slightly red, but free movement of the epiglottis returned four weeks after the operation. The pulse and respiration showed nothing anomalous.

Arthand and Butte severed the vagi in seven experiments, and report as a consequence there was increased respiratory action, followed by a return to normal condition.

Langendorff experimented on this subject, showing that severing one vagus caused long and deep respiration.

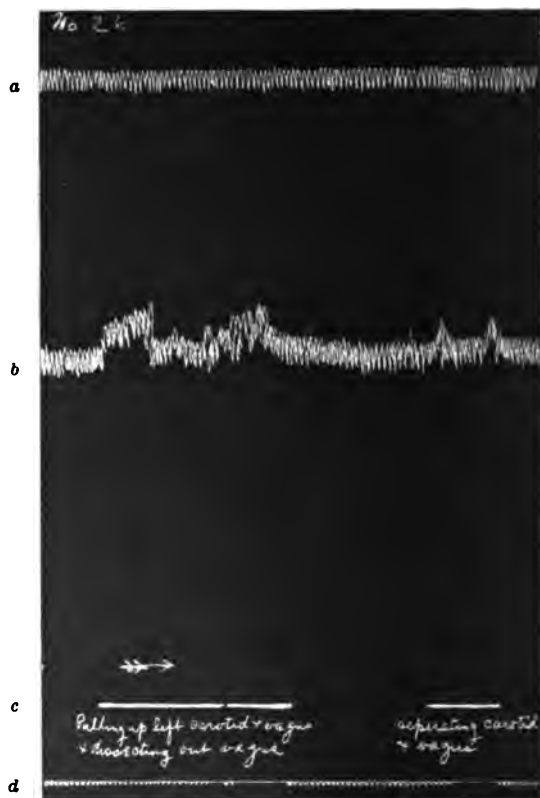
Cruenhogan claims that the section of one vagus either produces no change in respiratory movements, or, if so, the movement is but slightly lessened in frequency.

Widmer, in nineteen cases of one-sided vagus sections, states that in eight cases there was no change in respiratory movement; in one there

was a slight change; in another feeble respiration, and an increase in an already cyanotic condition.

Shevla found in a series of operations that the section of one vagus slows the respiration with deep inspiratory action, and that after several days respiration became more rapid until it reached its original frequency.

FIG. 1.



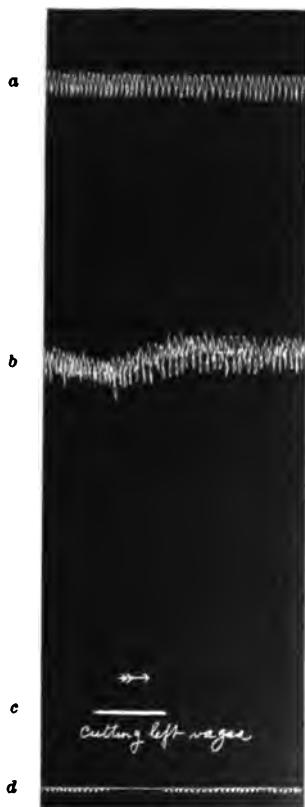
Vagal Experiment. *a*, respiration; *b*, blood-pressure; *c*, signal; *d*, seconds. Note the variation in the blood-pressure during raising and dissecting the vagus and the carotid. The second signal designates the dissection of the nerve from the artery. Note the irregular blood-pressure curve.

von Anrep, experimenting with warm-blooded animals, found that they could not withstand bilateral vagotomy. The animals died of pulmonary complications.

EXPERIMENTAL RESEARCH. *Protocols.* 1. *Vagal Experiment.* February 14, 1899. Cur dog; weight, twenty-six pounds; ether anaesthesia. While washing out the clot some magnesium sulphate solution ran into the carotid. The dog immediately had several convulsions. The blood-pressure dropped to the abscissa line and respira-

tion ceased. Artificial respiration was immediately instituted, and one-thirtieth grain of strychnine sulphate injected into the external jugular. After the lapse of a few minutes shallow respirations began, which gradually became stronger, the blood-pressure correspondingly rising. On manipulation of the left vagus there was a great temporary fall in the blood-pressure, followed by a slight rise.

FIG. 2.



Vagal Experiment. *a*, respiration; *b*, heart's action; *c*, time of experiment; *d*, seconds. Left vagus severed with a snip of sharp scissors. Note the slowed respiratory action and gradual rise in the blood-pressure.

2. *Vagal Experiment.* February 15, 1899. Canula in femoral artery. Brown cur dog; weight, twenty-seven pounds; ether anaesthesia. Pulling up and dissecting out of the left vagus and carotid caused a rise in blood-pressure with irregularity of the strokes (Fig. 1). Separating the vagus from the carotid caused a further slight rise in the blood-pressure and additional irregularity of the heart's action. On making traction of the left vagus respirations immediately slowed, the blood-pressure rose, and the strokes became irregular. On severing the left vagus the blood-pressure immediately rose, and respirations became slow. On severing the right vagus respirations became deep

and slow, showing quite an interval at the end of each expiration. The heart's action during this time was rapid and the blood-pressure became slightly elevated.

3. *Vagal Experiment.* February 16, 1899. Black cur dog; weight, eighteen pounds; ether anæsthesia. While separating the right carotid from the vagus by sharp dissection a slight rise in the blood-pressure was noticed. This dissection, together with other manipulation, caused a marked rise in the blood-pressure and slowing of the respiration. Severing the vagus caused a rise with no perceptible change in the respiration. The same experiment on the opposite vagus showed a like result as far as manipulation was concerned, but on severing the vagus the respirations immediately became slow.

4. *Vagal Experiment.* February 17, 1899. Yellow cur dog; weight, twenty-one pounds; ether anæsthesia. Canula in right carotid. Pulling and manipulating the right vagus caused an immediate rise in the blood-pressure and a slowing of the respiration. After one-half minute the blood-pressure returned to the point it occupied previous to the stimulation. After four minutes of manipulation of the carotid and vagus, similar to that produced by dissecting out a tumor, there was a slight rise in the blood-pressure, but no change in the respiration. On severing the left vagus there was an immediate gradual rise (Fig. 2) and a slowing of the respiration. Severing the right vagus caused an increased rapidity of the heart's action, producing a slight rise in the blood-pressure, with a slowing of respiration. There was a long pause at the end of each expiration.

5. *Vagal Experiment.* February 17, 1899. Mongrel dog; weight, twenty-six pounds; ether anæsthesia. Canula in right femoral artery. Dissecting the left vagus from the artery caused a slight fall in blood-pressure, but no change in respiration. Separating the right vagus from the carotid produced an immediate slight rise in blood-pressure. The experiment was made as nearly alike as possible on both sides. Severing the right vagus caused a rise in blood-pressure and a slowing of respiration. On severing the left vagus an additional rise in blood-pressure followed, and there was marked slowing of the respiration.

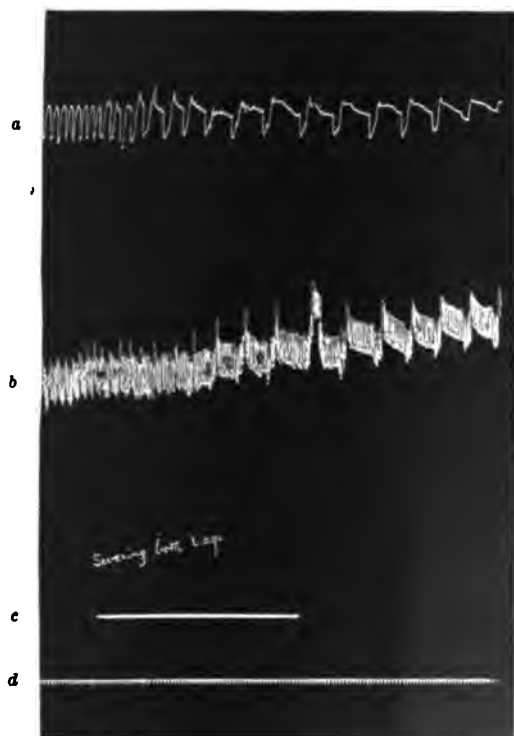
6. *Vagal Experiment.* February 18, 1899. Black mangy cur dog; weight, thirty-two pounds; ether anæsthesia. Canula in right carotid. Separating the right vagus from the carotid produced a rise in the blood-pressure. Both vagi severed by quick snips of a scissors. Respiration was much slowed and soon ceased. Heart-beats increased in frequency, and the blood-pressure gradually rose. (See Fig. 3.)

7. *Vagal Experiment.* February 18, 1899. Long-haired yellow mongrel dog, fair condition; weight, sixteen pounds; ether anæsthesia. Canula in right femoral. On pulling up the right carotid and vagus and roughly manipulating them there was a slight rise in the blood-pressure, which fell to the control as soon as manipulation ceased. The same was repeated several times with like result. On roughly manipulating the left vagus there was an immediate rise in the blood-pressure. (See Fig. 4.)

8. *Vagal Experiment.* February 20, 1899. Black mongrel, good condition; weight, twenty-two pounds; ether anæsthesia. Delicate manipulation of left side of neck caused a slight rise in the blood-pressure. Dog not entirely under anæsthetic. Repeating the same for about fifteen seconds again caused a slight rise. Applying vigorous

traction and rubbing the left vagus with artery forceps caused a marked irregular rise in blood-pressure, and a slowing of respiration, with a lengthening of the amplitude of the respiratory excursions. On cessation of manipulation the blood-pressure remained high, but the respiratory amplitude was lessened. Repeating the experiment caused an additional steady rise in the blood-pressure and a marked slowing of respiration. Upon a repetition of manipulation the pressure readily

FIG. 3.



Vagal Experiment. a, respiration; b, heart's action; c, time of experiment; d, seconds. Severing both vagi. Note the slowed respiration and the gradually rising blood-pressure. The respiratory effect upon the blood-pressure is well marked.

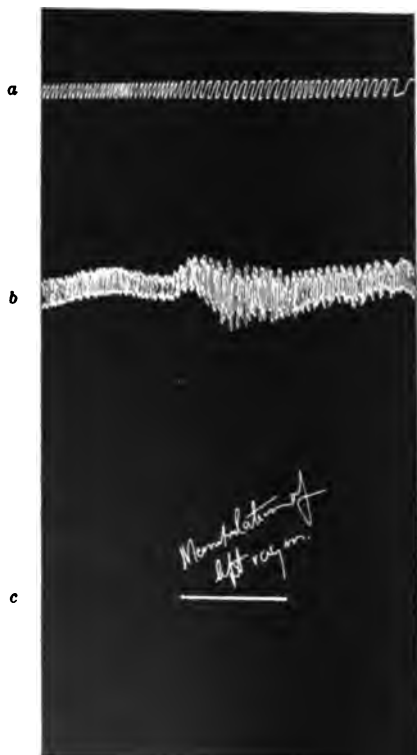
fell and the respiration remained slow. When the blood-pressure had reached the level of the control, the respiration improved, after which there was a slight rise. Roughly manipulating the right vagus caused a marked rise in the pressure, but not so marked as in the left. Respirations were not appreciably altered.

9. *Vagal Experiment.* February 20, 1899. Fox terrier, fair condition; weight, fourteen pounds; ether anæsthesia. On rubbing the left vagus between the thumb and finger an immediate rise in the blood-pressure occurred, and the respiration was much slowed. Dissecting out and vigorously rubbing the right vagus between the fingers caused a rise in the blood-pressure, but no alteration in the respiration. This

was repeated several times with like results. On roughly manipulating the trunk of the superior laryngeal, the blood-pressure fell to the abscissa line; the heart was reflexly paralyzed. (See Fig. 4.)

SUMMARY OF EXPERIMENTAL EVIDENCE. In the dog the sympathetic fibres run in a common trunk with the vagus proper. Therefore this nerve has been designated the vago-sympathetic, so that allowance must be made for the sympathetic factor. Picking up the artery and nerve together and bringing them up in the wound, making

FIG. 4.



Vagal Experiment. a, respiration; b, heart's action; c, time of experiment. Tracing showing the effect of mechanical nutation of the vagus. Note the slowed respiration during and following nutation, also the slowed heart-beat and irregular blood-pressure curve.

an ordinary blunt dissection and separating these structures from their surrounding tissues, produced slight effect upon the circulation and respiration. On separating the nerve from the artery by means of blunt dissection, comparatively slight effects were noted. Grasping the nerve with the forceps produced, in most instances, a rise in the blood-pressure and a slowing of the respiration. Dragging down upon the nerve produced a marked effect. Picking up the nerve and rubbing it

up and down, between the fingers, producing as much irritation as possible, caused a very distinct slowing of the respiration, and in most instances a rise in the blood-pressure. Occasionally, however, there was a temporary fall with a marked increase of the length of the heart strokes, indicating an inhibitory effect. In no instance was the heart completely inhibited. Grasping the nerve with two hemostatic forceps and so manipulating and irritating it, by sliding the forceps up and down, as to finally wear the nerve in two, produced in most instances a rise in the blood-pressure, and usually a very decided slowing in the respiration. Not only was the respiration slowed, but the amplitude was diminished. It required some time before the normal rapidity and amplitude were regained. No amount of mechanical irritation, even to the extent of mechanically destroying the vagi, produced an arrest of the heart's action. Severing one vagus was attended by comparatively little effect upon either respiration or circulation. Usually there was a slight rise in the blood-pressure, and a slight decrease in the frequency of respiration, with an increase in the amplitude. The blood-pressure curve remained regular, and no particular effect upon the amplitude of the excursion of the manometer was noted. However, on severing both vagi the blood-pressure rose considerably, ran an uneven course, the frequency of the heart's action was much increased and the length of the stroke shortened. Respirations were greatly decreased in frequency, and the amplitude of the respiratory excursion was markedly increased, so that, as nearly as could be estimated, the loss occasioned by the respiratory action was about counterbalanced by the increased amplitude of the excursion. The respiratory mechanism was much more affected than the circulatory and exhibited early signs of exhaustion.

CLINICAL. CASE I.—*Excision of right vagus, together with all the venous and arterial trunks on that side, in operation for malignant disease; respiratory failure after the operation; recovery.* John W., aged thirty years and eleven months, had had a very large median epithelioma of the lower lip removed. He returned to the hospital because of tumors on both sides of the neck; the glands were extensively involved; those on the left side were removed at a separate operation. The dissection was extensive, involving the removal of the external jugular vein, the external carotid artery, the submaxillary and parotid glands, exposure by close dissection of the common carotid artery, and the vagus nerve. Two weeks later the right side was operated upon. A chain of glands along the internal jugular vein was so extensively involved that on reaching this point it was found necessary to sacrifice both jugular veins, the common carotid artery, and its branches. The artery was separated from the vagus, but on carrying the dissection higher it was found that the vagus was so directly involved in the growth that it was necessary to sacrifice it. A consultant made a pulse-count both before and after severing the nerve. The heart became accelerated four beats per minute. No other effect was noted. Respirations were

not affected. A hypodermic injection of $\frac{1}{100}$ grain of atropine had been previously given. The dissection was then carried up behind the angle of the jaw, including the submaxillary and the parotid glands, the facial artery and nerve, and all the soft parts. The vagus was then severed just below the point at which the superior laryngeal is given off. This nerve and the lingual were laid bare in the dissection. The dissection was carried far behind the angle of the jaw, and the vessels were so deeply severed as to leave an insufficient length for applying a ligature. By means of a small curved needle, armed with silk, the deep structures around these vessels were picked up, forming a close purse-string, which, when tied, closed them. During the latter part of the operation the respirations were considerably slowed and the patient became somewhat cyanotic. The wound was then closed after completely arresting the hemorrhage. The sympathetic nerve was laid bare throughout most of its course, and the superior cervical ganglion lying upon the floor of the wound was wholly exposed. After closing the wound by bringing together as far as possible its deeper portions by means of catgut sutures the patient was sent to the ward. Directly afterward he was reported to be in a critical condition due to respiratory failure. Upon arriving at the bedside, I found the house surgeon maintaining artificial respiration. While observing the patient I noticed fresh blood on the dressings, indicating a dangerous hemorrhage. Hastily cutting away the dressings and laying open the wound, I thrust my hand into the upper angle from which the hemorrhage came. Although this was quickly done, there was a great loss of blood. With my hand grasping his throat he was replaced upon the ward ambulance and taken to the operating-room, the house surgeon maintaining, as well as he could, artificial respiration on the way. On returning to the operating-room the large vessels were clamped and the forceps allowed to remain. The patient was removed from the ward to the operating-room on account of want of light. Ice and heat were alternately applied, which helped to restore the respiratory action. He was given subcutaneous injections of saline solution and $\frac{1}{100}$ grain of strychnine every half-hour. At the end of twenty-four hours the forceps were removed, and at no time after recovery from immediate effects of the operation were there observed any unusual respiratory or circulatory symptoms. The patient made a good recovery. The microscopical examination of the growth showed a large number of inclusion cells, indicating a marked malignancy. There was considerable hoarseness for two weeks, after which it gradually disappeared. The respiratory failure in this case was in full accord with the experimental evidence.

CASE II.—(Abstract.) In removing glandular metastases following total laryngectomy, the left carotid artery and vagus were involved. The pulse and respirations were observed during the excision and after it. A hypodermic injection of $\frac{1}{100}$ of a grain of atropine was given twenty minutes before the operation was begun. The nerve was severed by a quick snip of the scissors. No effect upon the circulation or respiration was noted. Patient made a good recovery.

CASE III.—(Abstract.) In this patient the vagus was resected. One $\frac{1}{100}$ of a grain of atropine was administered prior to the operation. No immediate effects were noted.

CASE IV.—(Abstract.) A full charge from a shot-gun whose muzzle was within a few inches of the patient's neck when discharged, entered

the neck in the upper part of the carotid triangle. The artery was torn off and the nerve ulcerated. The wadding and shot were firmly packed upon and driven into the torn nerve and other structures of the neck. The pulse was reduced to forty-two beats per minute. Respirations were slow, exhibiting quickened respiratory action with lengthened pause and prolonged expiratory phase. The slow pulse continued more than two hours, after which the "vagal" mechanism went into resolution, and an extremely rapid cardiac action followed.

CASE V.—(Abstract.) In removing a mixed tumor of the parotid the dissection was carried for some distance upon the carotid and the vagus. While freeing the nerve near the level of the tip of the styloid process the pulse dropped from 90 to 56. The wound was almost bloodless, exhibiting the various structures in plain sight. A 4 per cent. solution of cocaine was applied upon a piece of cotton and the dissection carried elsewhere. After three minutes the dissection of the vagus was again resumed. Meanwhile, the pulse had returned to 86. During the remainder of the resection of the vagus there was no appreciable alteration in the heart's action, in spite of a more severe manipulation than had before been given. The depth of the tumor not having been anticipated, no atropine had been administered. This case illustrated the effect of stimulation of the upper portion of the vagus, notably near the point at which the superior laryngeal is given off. This nerve trunk, though not seen at the time, might have contributed to the cardiac phenomena from indirect violence.

CASE VI.—(Abstract.) *Emergency removal, without anæsthesia, of a large goitre during unconsciousness from asphyxia; vagus clamped; recovery.* Preliminary administration of one $\frac{1}{100}$ grain of atropine; inclusion of vagus nerve in large forceps; no cardiac inhibition; artificial respiration.

Female, aged sixty-four years; had had a goitre twelve years, which during the six months preceding the operation had rapidly developed. The tumor was large, quite firm, deeply situated in the neck, extending well down behind the sternum and clavicle, displacing the apex of the lung and producing a compression of the trachea against the vertebral column. The tumor was so firmly fixed as to be scarcely movable. During two months she had been obliged to make use of the extraordinary muscles of respiration. The development of the platysma was remarkable. In each inspiratory effort the contraction of the muscle was so powerful as to draw down the angles of the mouth, lower lip, and the integument of the lower part of her face, to throw into horizontal folds the entire surface of her neck and upper thorax, and to entirely change the aspect of the neck and face. The lower jaw, meanwhile, was carried fixedly forward and upward. By this means she was able to so slightly relieve the pressure upon the trachea as to accomplish a stridulous though scant exchange of air. It was planned to expose the tumor under local anæsthesia, then try to relieve the obstruction by elevating the tumor while administering general anæsthesia. She was placed upon the table, but was absolutely unable to breathe in a lying posture. She struggled for breath, sprang up into a sitting posture, then gasping, turned cyanotic, became unconscious, and fell apparently lifeless on the table. At a single stroke with a scalpel the tumor was laid bare, then literally torn out, while the blood from the many torn vessels flooded her face and neck. Artificial respiration was in the

meantime begun. The hemorrhage was first controlled by firm gauze packing, then with a large forceps the common carotid artery, jugular vein, and vagus nerve were grasped *en masse* below and likewise above, completely controlling the hemorrhage. The operation was performed in about forty-five seconds. The patient had been given $\frac{1}{160}$ grain of atropine previous to the operation, which prevented any cardiac inhibition, although the vagus was crushed at the point of clamping. The operation was completed without her knowledge. She said afterward that she believed she was dying and was conscious that something was done, but had no pain. Patient made a good recovery.

AN EXPERIMENTAL INVESTIGATION INTO THE CAUSES AND THE TREATMENT OF DIABETES MELLITUS.¹

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In the years 1890 to 1892 Lépine, Arthus, Krauss, and others published a series of investigations on the self-destruction of the blood-sugar; they postulated the existence of a sugar-splitting (glycolytic) ferment, and formulated a number of more or less hypothetical views in regard to the rôle of this substance in the pathogenesis of diabetes.

Some of the experiments of these authors I have repeated and amplified.

I. *The Sugar-destroying Action of the Blood.*

Experimental Series 1. 100 c.c. (3½ oz.) of blood were removed, under aseptic precautions, from the median vein of a healthy adult man, rapidly defibrinated, and allowed to stand in a sterile vessel at body temperature. Samples of this blood were removed at varying intervals, and the sugar determined by titration. The method employed was that of Röhrmann, modified by Arthus² (it consists essentially in removing all of the albumin and titrating the sugar in the filtrates according to Fehling). The following table will show the progressive loss of sugar:

¹ Read, by invitation, before a meeting of the Section on General Medicine of the College of Physicians of Philadelphia, November 11, 1901.

² 20 c.c. of blood are poured into from 7 to 8 volumes of boiling water that is slightly acidulated with acetic acid filtered, the coagulate expressed, washed repeatedly with hot acidulated water, the filtrate and the washings concentrated to about 200 c.c., filtered again, freed from the last traces of albumin with sodium-acetate and chloride of iron; the coagulate filtered off, washed, washings and filtrate evaporated to about 150 c.c. and carefully neutralized. In this fluid the sugar is determined according to Fehling with a modification suggested by Causse, viz., the addition of 2.5 g. of potassium ferrocyanide for the purpose of keeping the cuprous oxide in solution; the disappearance of the blue color of the solution is the index.

| | |
|---|-----------------|
| Blood immediately after removal from the vein at 39° C. | 0.179 per cent. |
| " $\frac{1}{2}$ hour after removal | 0.177 " |
| " 1 hour after | 0.169 " |
| " $1\frac{1}{2}$ hours after removal | 0.148 " |

The temperature at which the blood is kept seems to exercise a considerable influence on the intensity of the process; the temperature optimum is the temperature of the body; below 10° C. no glycolytic action is manifest; heating the blood to 60° C. destroys the power. The statement made by Lépine that the presence or absence of oxygen exercises an effect could not be verified; a series of experiments performed as above in an atmosphere of oxygen, nitrogen, hydrogen, and carbon dioxide revealed no appreciable differences.

The lymph, too, possesses glycolytic powers, as shown by the following:

Experimental Series 2. Lymph removed from the thoracic fistula of a dog under aseptic precautions, and allowed to stand in a sterile vessel as above, will gradually destroy its own sugar; the glycolytic power of the lymph is, however, considerably greater than that of the blood; the sugar destruction proceeds more rapidly and is more radical.

Experimental Series 3. If a stated quantity of blood or of lymph is allowed to flow, under aseptic precautions, into a sterile vessel containing a sterile sugar solution, and the sugar in the mixture is determined at varying intervals, it will be found that a considerable quantity of the sugar is destroyed; the lymph destroys more sugar than the blood in a given time; as much as 4 per cent. of sugar are lost after one hour and as much as 7 per cent. after two hours, with blood; 8 and 12 per cent. in the corresponding time, with lymph.

The conclusions from the experimental series 1, 2, and 3 are that the blood and the lymph incorporate an agent that can destroy sugar.

II. *The Glycolytic Ferment.*

In order to determine the source and character of the sugar-destroying substance in the blood, the following experiments were undertaken:

Experimental Series 4. 250 c.c. (8½ oz.) of blood were defibrinated and centrifuged for two hours at the rate of about 2400 revolutions a minute.

(a) *The serum* was decanted and examined for its glycolytic power and for the amount of sugar it contained; it was found that the serum contained nearly all the blood sugar and possessed no glycolytic power whatever.

(b) *The corpuscles* were mixed with a volume of cold physiological salt solution equal to the amount of serum decanted; the mixture was centrifuged, and the process of decantation and centrifuging repeated a number of times; in this manner almost all traces of serum were removed. It was found that the blood-corpuscles that remained behind incorporated no sugar and possessed no glycolytic power.

(c) *The washings.* The first washing contained nearly all the serum that was adherent to the corpuscles, and consequently, some sugar and

albumin; it had also acquired some glycolytic power. The second washing contained only traces of sugar and albumin, but had considerable glycolytic power. The third washing finally contained no sugar, only a very small trace of albumin (probably some hæmoglobin that had been dissolved out of disintegrating red blood-corpuscles), but possessed a still greater glycolytic power. The washings after this were indifferent, still contained some albumin in traces, their glycolytic power decreased successively, and finally disappeared.

From the experiments of series 4 we draw the conclusion that the sugar-destroying agent of the blood is contained in the corpuscles and not in the serum; it is soluble in cold physiological salt solution, and is consequently unorganized and not dependent on the life of the cell; in other words, the glycolytic power of the blood is not a vital process, but a chemical one, and due to the action of a "ferment."

In order to determine whether the glycolytic ferment is bound to the white or the red blood cells, the following experiments were undertaken:

Experimental Series 5. Ligatures were placed around the jugular vein of a cow, about four inches apart, and the piece of vein between the two ligatures excised and suspended for twenty-four hours in a vertical position; in this manner contact of the blood-corpuscles with "vital" tissue was insured and the coagulation of the blood prevented; at the same time the corpuscles could follow the law of gravity and settle at the dependent portion of the vein; it was determined in the course of these experiments that it is possible to bring about a distinct separation of the red and the white corpuscles in this manner, the red ones naturally occupying a lower position than the white ones. By introducing an aspirating needle into the lowest portion of the vein red blood-corpuscles mixed with a little serum are obtained; a little higher up white corpuscles mixed with serum are aspirated, and near the top of the vein pure serum is drawn.

The serum and the white and the red blood-corpuscles were placed into separate portions of sterile sugar solutions of known titer, and allowed to stand in the incubator for twenty-four hours; it was found that the serum and the red blood-corpuscles possessed no glycolytic power, but that the white cells were capable of destroying a large proportion of the sugar.

It must be added that in all these experiments with sugar solutions every effort was made to insure absolute sterility; bacteriological examinations of all the specimens were made, and those samples that were found to contain any bacterial growth whatever (despite the precautions taken, including the addition of thymol to all the specimens) were considered not available, and thrown out.

Experimental Series 6. If blood is removed from the vein of an animal and allowed to flow into a sterile vessel, and kept at body temperature as in Experimental Series 1, and the sugar determined every ten minutes, it will be found that the decrease of sugar is almost *nil* for the first half hour; that the sugar destruction progresses more rapidly

during the second half-hour, still more rapidly during the third, and that after that time it gradually decreases. (See table.)

If stained blood specimens are made during the first to the fourth half-hour, it will be found that glycolytic action seems to begin as soon as the first degenerative changes are noticeable within the leucocytes, and to progress *pari passu* with the increasing degeneration.

Experimental Series 7. The finding chronicled above induced us to attempt a more rapid artificial degeneration of the white cells; this was brought about by the addition to the blood of certain chemotactic substances, viz., turpentine and powdered animal charcoal; the blood was also rapidly cooled by immersion of the vessel into ice-water immediately after removal from the vein, and then placed into the incubator, after mixing with the sugar solution as above; these procedures, in fact, caused a more rapid glycolytic action during the first half-hour, a maximum during the second half-hour, and a marked decrease again during the third half-hour. (See table.)

TABLE TO EXPERIMENTAL SERIES 6 AND 7.

| | <i>Blood alone.</i> | <i>With turpentine.</i> | <i>Charcoal.</i> | <i>Cooling.</i> |
|------------------------|---------------------|-------------------------|------------------|-----------------|
| Blood on removal . . . | 0.164 | 0.171 | 0.169 | 0.170 |
| First half-hour . . . | 0.160 | 0.168 | 0.150 | 0.151 |
| Second half-hour . . . | 0.148 | 0.148 | 0.142 | 0.140 |
| Third half-hour . . . | 0.129 | 0.144 | 0.140 | 0.137 |
| Fourth half-hour . . . | 0.118 | | 0.138 | 0.136 |

Experimental Series 8. In Experimental Series 4 we showed that the first decantation of physiological salt solution removed from the corpuscles that had been separated from the serum by centrifuging possessed a smaller glycolytic power than the second decantation, and that the third, fourth, and fifth decantations showed decreasing sugar-destroying powers.

If, after removal of the serum, the corpuscles are shaken with a few drops of turpentine, or are mixed with charcoal and shaken, or are rapidly cooled off, and are then extracted with physiological salt solution, it will be found that the first decantation possesses stronger glycolytic powers than the second, and that the second, third, fourth, and the remaining corpuscles possess none at all.

Experimental Series 9. If, after removal of the serum as above, the corpuscles are mixed with glass powder and rubbed into a fine pulp in a mortar until microscopically no formed elements can be discovered, and if the mass is then extracted with a physiological salt solution, it will be found that the glycolytic power is greater than in any of the extracts obtained in the other experiments.

The conclusions from the experimental series 5, 6, 7, 8, and 9 are that the glycolytic ferment of the blood is present in the white cells (whether in the leucocytes or the plaques is not determined) and degeneration of the white cells must occur before the ferment can develop its powers.

While examining the blood serum for its glycolytic power (with negative result, see above) a peculiar phenomenon was observed that has a bearing on the question discussed in the preceding paragraphs, and may be mentioned in this place. The serum contains all the blood

sugar in solution; after being allowed to stand in the incubator for half an hour it was found in some instances that not only had no sugar been lost, but that, on the contrary, an increase of sugar had occurred! This can only be explained by the fact that a certain amount of glycogen was present in the serum which became converted into dextrose, in this way increasing the titer for sugar. This fact must be taken into consideration when interpreting the small glycolytic power of the blood during the first half-hour after removal from the body of an animal; the process at that time is quite complicated, viz., new sugar is being formed from the glycogen, the disintegration of the white cells is slowly beginning, the ferment is being liberated and, by osmosis, is gradually diffusing through the fluid, finally causing the destruction of the sugar.

III. *The Rôle of the Pancreas.*

According to Lépine, the glycolytic power of the blood is considerably reduced after removal of the pancreas; a number of investigators after him denied this. In order to clear up this uncertainty the following experiments were undertaken:

Experimental Series 10. The pancreas was removed from two large dogs; the animals received no nourishment until their death, which ensued sixty and sixty-eight hours after the operation; eight and twenty hours after removal of the pancreas 30 c.c. (about 102 oz.) of blood were removed from each animal and allowed to flow into a sterile vessel containing 3 mg. ($\frac{1}{20}$ gr.) of oxalate of sodium (in order to prevent the coagulation of the blood); the four specimens were placed in the incubator at body temperature; sugar determinations were performed immediately after removal of the blood from the vein and every half-hour thereafter for one hour and a half. (See table.)

TABLE TO EXPERIMENTAL SERIES 10.

| | 8 hours after operation. | | 20 hours after operation. | | Normal dog. Compare table to Exp. Series 6 and 7. |
|--------------------------------|--------------------------|--------|---------------------------|--------|---|
| | Dog 1. | Dog 2. | Dog 1. | Dog 2. | |
| Blood at once . . | 0.321 | 0.336 | 0.347 | 0.354 | 0.164 |
| After $\frac{1}{2}$ hour . . | 0.313 | 0.329 | 0.339 | | 0.160 |
| After 1 hour . . | 0.304 | | 0.331 | 0.340 | 0.148 |
| After $1\frac{1}{2}$ hours . . | 0.297 | 0.312 | 0.326 | 0.331 | 0.129 |
| Percentile loss . . | 7.47 | 7.11 | 6.05 | 6.49 | 21.3 |
| Absolute loss . . | 24 ‰ | 24 ‰ | 21 ‰ | 22 ‰ | 35 ‰ |

It will be seen that the absolute loss of sugar as compared to the absolute loss in the blood of a normal dog (the pancreas of which was not removed) is only slightly smaller (viz., 21–24 *pro mille* as against 35 *pro mille*), but that the percentile loss is very much smaller (viz., 6.05–7.47 per cent. as against 21.3 per cent.). It is difficult to draw any definite conclusions from these figures in regard to the decrease of the sugar-destroying powers of the blood after removal of the pancreas; for it might be argued that, in view of the abnormally large quantities

of sugar found in the blood (see table) after removal of the pancreas, a decrease in the percentile loss would be self-evident, even though the sugar-destroying powers remain normal; that, in fact, the only criterion for a loss of glycolytic power would be a marked decrease in the absolute quantity of sugar destroyed. It is true that less sugar is absolutely destroyed after extirpation of the pancreas, according to our tables (about one-third less), but the difference seems too small to warrant far-reaching conclusions.

A second series of experiments carried out in a different manner, however, definitely decide this question.

*Experimental Series 11.*¹ The object of these experiments was to increase the quantity of blood-sugar in a dog whose pancreas was not removed and to study the percentile and the absolute loss of sugar under the same conditions and at the same intervals as in Experimental Series 10. For this purpose the ureters of a dog were occluded by temporary ligatures and 12 g. (3 dr.) of dextrose dissolved in 30 c.c. (1 oz.) of sterile water allowed to flow slowly into the right jugular vein. In this case (see table) it was found that the absolute loss of sugar was far greater than normal and the percentile loss approximately normal.

TABLE TO EXPERIMENTAL SERIES 11.

| | <i>Dog of Exp. Series 11.</i> | <i>Normal dog.</i> |
|---------------------------|-------------------------------|--------------------|
| Blood at once | 0.462 | 0.164 |
| After ½ hour | 0.431 | 0.160 |
| After 1 hour | 0.408 | 0.148 |
| After 1½ hours | 0.381 | 0.129 |
| Percentile loss | 17.6 | 21.3 |
| Absolute loss | 81 ‰ | 35 ‰ |

In comparing, therefore, the loss of sugar in two dogs whose blood-sugar is increased above normal—the one animal with a pancreas, the other without—it will be found that the former animal destroys a far greater quantity of sugar than the latter; in other words, we arrive at the conclusion from the Experimental Series 10 and 11 that the removal of the pancreas decreases the glycolytic power of the blood.

From the conclusions arrived at so far it seemed *a priori* probable that the pancreas was in some way concerned in the manufacture of the glycolytic ferment—a supposition that a number of investigators, notably Lépine, the father of the glycolytic ferment, had expressed. Many attempts have, in fact, been made to isolate such a substance from the pancreas, heretofore, however, without success.

The known ferments of the pancreas placed into sugar solutions do not possess the power of destroying it. I could determine that trypsin, amylapsin, steapsin, and a variety of preparations of pancreatin may

¹ In the animals treated in this series it was found that alcohol, oxybutyric, lactic, and diacetic acid and acetone were formed in the blood; a report on this interesting and significant finding is reserved for future publication.

remain in contact with sugar solutions of varying concentration for many months without causing any loss of sugar.

In the course of some experiments on the destructive metabolism of hæmoglobin the following discovery was made: It was found that a watery extract of liver pulp always contained considerable quantities of trypsin, and it occurred to me that possibly this ferment was concerned in the formation of bile-pigment from hæmoglobin within the liver. In order to determine this point an attempt was made to artificially create conditions *in vitro* as nearly similar as possible to those obtaining within the liver. For this purpose a few drops of a dilute trypsin solution (with all due precautions in regard to antisepsis) were added to a dilute solution of hæmoglobin containing a small quantity of dextrose or glycogen, and the mixture kept at room temperature for a week. At the end of this time the hæmoglobin was found to be destroyed, the liquid was colorless, and contained bile-acids, while bile-pigment was found in the sediment. At the same time it was discovered that if sugar was omitted from the mixture—in other words, if trypsin was simply added to a dilute solution of hæmoglobin alone—the destruction of hæmoglobin proceeded along altogether different channels, i. e., neither bile-pigment nor bile-acids were generated.¹ The sugar, therefore, played an important rôle in this conversion. A determination of the sugar before and after showed that a considerable quantity of the sugar had been destroyed in the process; in other words, trypsin in the presence of hæmoglobin possesses glycolytic powers. Here, then, was found a glycolytic ferment of pancreatic origin.

If trypsin is the glycolytic ferment that we have been discussing, then it should also be present in the blood, and, basing on our previous conclusions, should be present in the leucocytes and liberated from them when they disintegrated; or, inversely, the glycolytic ferment of our previous discussions should possess the properties of trypsin.

Both these postulates I have found to be correct.

In order to determine the character of the glycolytic ferment of the blood and to prove or disprove its identity with trypsin it was necessary to manufacture, if possible, some of the substance from the blood. We saw that the glycolytic ferment is liberated only after the disintegration of the leucocytes, so that the task set, theoretically, was to procure large quantities of leucocytes from normal blood, to cause their disintegration, and then to extract the ferment. Practically such an undertaking is nearly impossible; we know of no adequate method of procuring large quantities of leucocytes from the blood, nor was it probable that after disintegration only the ferment we were looking for would go into

¹ The results of this investigation have been recently published. See "Some Experiments on the Formation of Bile Pigment," etc. Philadelphia Medical Journal, January 11 and 18, 1902.

solution. As a matter of fact, a variety of substances have been described in leucocytic extracts. In view of these difficulties we had to content ourselves with a determination of the properties in general of the salt-water *extract* described above.

For the purpose of comparison with trypsin this is sufficient, because in the case of this ferment we are confronted with like difficulties. No one, so far, has determined the chemical character of trypsin, nor has anyone in all probability ever manufactured pure trypsin. Its presence in different solutions and products is revealed to us merely from its manifestations; in other words, from its ferment action on stated substances under stated conditions.

The properties of trypsin that we are familiar with are briefly the following: In an alkaline medium it digests proteid in such a manner that certain well-characterized products of proteolysis are generated. Brought in contact with certain coagulable solutions it is capable of inducing coagulation. Injected into the veins of an animal it produces widespread congestions, and is said to cause the death of the animal by forming thrombooses. Chemically, it gives several proteid reactions (see footnote on page 671), and it is precipitable by dilute acetic acid (Hammarsten). It is coagulated by absolute alcohol, but is again soluble in water or in physiological salt solution, even after having remained in contact with alcohol for many weeks. Finally, according to an investigation that I have recently published (*Philadelphia Medical Journal*, January 11 and 18, 1902), it is capable of decomposing hæmoglobin and forming from it bile-pigment and bile-acids, only, however, in the presence of dextrose or of glycogen, and at the same time of destroying some of the sugar.

The solution of the glycolytic ferment from the blood possesses these properties of trypsin; this is shown by the following experiments:

Experimental Series 12. If fibrin flakes are placed into the ferment solution, and the latter is slightly alkalized and kept at 38° C. for forty-eight hours, it will be found that a partial solution of the fibrin flakes has occurred and that albumoses, peptone, etc., in small quantities can be discovered in the solution.

Experimental Series 13. If the leucocyte extract is brought in contact with coagulable liquids it produces coagulation; this fact had been determined previously by Schmidt, Lilienfeld, and others; the former and his pupils attribute this action to the presence of a substance they call the fibrin ferment,¹ the latter to nucleo-histon; both substances are

¹ Wright (Journ. Path. and Bact., June, 1893). "There does not at present exist any test of any kind, chemical or physiological, of the existence of such a substance as the fibrin ferment. We may, therefore, omit it from our catalogue of coagulative substances."

Halliburton and Brodie (Journ. of Physiol., vol. vii. p. 143). "Whether any relation exists between the fibrin ferment and . . . nucleo-proteids is a matter that demands renewed research."

chemically more or less undefined, and have probably not been procured in a pure shape, so that it is uncertain what the active final principle is that causes coagulation.

Experimental Series 14. If a small quantity of the solution is injected into the veins of a dog it produces instant death in some cases; on post-mortem examination thromboses will be found (see below). This fact, too, had been established by others.

Experimental Series 15. In order to test the chemical properties of our ferment it seemed imperative to manufacture a product that was more pure than the extract employed above; for this purpose a method was employed identical to that used to obtain the purest preparation of trypsin known; the blood (instead of the pancreas) was allowed to stand for half an hour at room temperature, and then poured into absolute alcohol and allowed to remain in contact with alcohol for four weeks; the alcohol was then removed by filtration and expression, and the residue (a brick-red, amorphous powder) extracted with normal salt solution; in contradistinction to the extract described above, this extract was colorless, and contained no hæmoglobin whatever (spectroscopic examination was altogether negative); on evaporation only traces of a dry residue were obtained showing that very little substance goes into solution; at the same time this extract possessed all the properties of the other one, and gave the chemical reactions of trypsin.

Experimental Series 16. The extract described in the preceding experiment, mixed with a dilute solution of hæmoglobin containing about 0.6 per cent. of dextrose, destroyed the hæmoglobin in the course of four to ten days; the fluid became colorless and, at the same time, bile-acids and bile-pigment were formed.

Experimental Series 17. The extract of Experimental Series 15 placed into sugar solution possessed no glycolytic power. The presence of hæmoglobin (or other proteid?) was necessary. It is probable that the glycolytic power of the extract described in Experimental Series 4 was due to the presence of minute quantities of hæmoglobin in the solution.

Experimental Series 18. Trypsin is the only one of the soluble ferments of the human organism that is never found in the urine; pepsin, rennet, diastase, and ptyalin have all been discovered there under certain conditions; the glycolytic ferment, too, is normally absent from the urine. It is further known that the urine destroys the power of trypsin; urine added to the extract described above destroys its glycolytic power.

To summarize, therefore, the actions of solutions of trypsin and solutions of our glycolytic ferment, we can state that they both can digest fibrin in alkaline solutions; that they can coagulate certain coagulable solutions; that they can produce thrombosis; that they give certain proteid reactions; that they are precipitable by dilute acetic acid; that they are precipitable by alcohol; that they are soluble in water after contact with alcohol; that they can form bile-pigment and acids from hæmoglobin in the presence of small quantities of dextrose; that they lose this power if dextrose is not present; that they possess glycolytic power in the presence of hæmoglobin; that they lose this power in the absence of hæmoglobin; that they are rendered inactive by con-

tact with urine, so that neither is normally excreted in an active form via the kidneys.

The conclusions from the Experimental Series 12 to 18 are that the solutions of the glycolytic ferment and solutions of trypsin are, if not identical, so similar that they cannot be distinguished by known methods. Until proof to the contrary is forthcoming, we are justified in declaring the glycolytic ferment of the blood and tissues to be none other than trypsin¹.

IV. *Pathogenesis.*

Diabetes is a symptom-complex, properly speaking, of a variety of pathological conditions. Glycosuria never occurs without hyperglycæmia; in other words, the sugar circulating in the blood must be increased considerably above normal before the kidneys excrete it. Hyperglycæmia may be due to an increased formation or assimilation of sugar, or to a decreased destruction, or to both.

In order to understand the pathology of a disease, it is necessary to understand the function or functions a perversion of which it represents. Normal sugar catabolism, the function perverted in diabetes, probably proceeds as follows:

Immaterial what the source of the sugar, whether it comes from the food or from the fats or proteids of the body, it is finally oxidized to carbon dioxide and water, and eliminated from the body in this form. The question arises (and applies with equal strength to the fats and the proteids), How can a substance that, outside of the body, requires very high degrees of temperature for its combustion be oxidized at a temperature not above 38° C.? The answer is given in the fermentative destruction of the sugar molecule by certain bacteria and other organized and unorganized ferments; as a preliminary step the sugar molecule must be split into simpler substances like alcohol or lactic acid that possess a greater affinity for oxygen than sugar. Even after these preliminary transformations the action of some oxygen-carrier must be brought into play in order to cause the union of these bodies with oxygen in the body.

We have demonstrated that a sugar-splitting substance, a or "the" glycolytic ferment, exists in the body. We have shown that it is present in the blood and the tissues, and that it is probably none other than trypsin. We have further elucidated the process by which sugar is destroyed, and have shown that it is by a process in the formation of bile-pigments and bile-acids that the disintegration of the sugar molecule can occur anywhere within the body where hæmoglobin is liberated in the presence of trypsin. A perversion of this glycolytic function

¹ Whether or not the solutions of trypsin and of the glycolytic ferment contain a *tertium quid* that is the real ferment, chemical analysis cannot determine. I have shown in another publication that as far as can be determined the purest trypsin is a "dentero-albumose."

must lead to hyperglycæmia and glycosuria; we are justified, therefore, in seeing one of the causes, if not the only cause, of diabetes in a reduction of glycolysis. The question is hereby moved one step backward: What are the causes that pervert glycolysis and reduce the sugar-destroying powers of the body?

It is hardly probable that a uniform cause for this perversion exists. In many cases we have no explanation that is based on facts, and however seductive the various hypotheses in regard to the rôle of certain functional nervous disorders, infections, auto-intoxications, etc., may be, we are not justified in accepting explanations that are merely evolved by *à priori* reasoning.

A few words, however, may not be amiss in regard to the rôle of the pancreas. We have seen what an important part trypsin plays in the destruction of the sugar under stated conditions; and we have shown, as far as that is possible, that trypsin is identical with the glycolytic ferment; no wonder, therefore, that removal of the pancreas causes the accumulation of sugar in the blood; if trypsin is absent, the destruction of hæmoglobin proceeds along abnormal channels, and no sugar is destroyed; consequently, it accumulates; in addition, the products of abnormal hæmoglobin catabolism (albumoses, etc.) must needs be harmful, and, circulating in the blood, produce many of the toxogenic symptoms of diabetes; they probably constitute protoplasmic poisons; finally, the accumulation of excessive quantities of sugar in the blood leads to a variety of perversions of metabolism that will be discussed at length in a monograph mentioned in a foot-note previously; it may be stated in this place that the generation of large quantities of acetone, diacetic acid, and of oxybutyric acid *may* be caused by the presence of abnormally large quantities of sugar alone.

The occasional occurrence of bronzed diabetes ("cirrhose pigmentaire") is suggestive, in view of the intimate connection we have shown to exist between the formation of bile-pigment and sugar catabolism.

In the present state of our knowledge we are not justified in assuming pancreatic disease in all cases of diabetes; it is possible that functional diseases of the organ may exist during life that leave no anatomical trace; but we do not know that this is the case. Recent discoveries by Opie, however, in regard to the frequent occurrence of degenerative changes in the islands of Langerhans of the pancreas, give a certain experimental fundament to such an hypothesis.

V. *Therapeutic Suggestions.*

What we have learned from our experiments seems to point the way to a rational therapeusis of hyperglycæmia, and therewith of glycosuria, and of diabetes.

The indication is to increase glycolysis. To do this the glycolytic ferment must be supplied. We know that such a ferment is present in the chyle, the blood, in leucocyte extracts of different kinds; we know, further, that trypsin, under certain conditions, possesses glycolytic powers, and that, finally, there are unorganized glycolytic ferments in certain plants—i. e., the glycolytic ferment of yeast (made by Buchner).

The following measures, therefore, promise some success:

1. The infusion of chyle or of blood from a healthy animal.
2. The injection of leucocyte extracts.
3. The injection of trypsin.
4. The injection of vegetable glycolytic ferments.

I wish to append, as a preliminary report, the results of the employment of some of these methods on animals that had been rendered diabetic by the extirpation of the pancreas.

Experimental Series 19. The pancreas was removed from two female dogs; the animals received no nourishment until their death. Twelve hours after the operation the first analysis of the urine was made, and every four hours thereafter; the urine was withdrawn by catheterization; the different specimens contained the following quantities of sugar:

TABLE TO EXPERIMENTAL SERIES 19.

| | <i>Bitch 1.</i> | <i>Bitch 2.</i> |
|-----------------|-----------------|-----------------|
| 7 A.M. | 0.62 | 0.77 |
| 11 A.M. | 1.27 | 1.24 |
| 3 P.M. | 1.82 | 1.96 |
| 7 P.M. | 2.02 | 2.21 |

A progressive increase of the sugar excretion will be noted.

After the last determination of sugar about 20 c.c. (about 6 dr.) of lymph, removed under sterile precautions from the thoracic duct of a healthy bitch of approximately the same age as the dogs whose pancreas was removed, were allowed to flow into the jugular vein, and the excretion of sugar determined every hour thereafter. (These experiments on the two dogs were performed under identical conditions at different times; for clearness sake the results obtained are tabulated together.)

TABLE II. TO EXPERIMENTAL SERIES 19.

| | <i>Bitch 1.</i> | <i>Bitch 2.</i> |
|-----------------|-----------------|-----------------|
| 8 P.M. | 1.97 | 2.14 |
| 9 P.M. | 1.64 | 1.70 |
| 10 P.M. | 0.34 | 0.40 |
| 11 P.M. | 0.82 | 1.13 |

It will be seen that a decrease of the sugar excretion occurred within an hour after the injection of the lymph; that it reached a still lower point two hours after the injection; reached its lowest point three hours after the injection, and then slowly increased again until it finally

reached a point as high as before the injection. One of the dogs died during the night; the other one survived nearly two days. No subjective signs of improvement in their condition could be observed during the period of decreased sugar excretion. Lépine performed a similar series of experiments and arrived at similar conclusions.

We interpret the peculiar variations in the sugar excretion as follows: The leucocytes of the lymph survived for a time in the blood-stream, and did not begin to degenerate to any appreciable extent for some time after the injection; this corresponds to their behavior outside of the organism when kept at body temperature; as a result, the glycolytic ferment was not liberated until the end of the first hour; at that time glycolytic action began, and continued for another hour; as the ferment was liberated it was again removed from the circulation, so that after three hours all the glycolytic ferment contained in the cells of the chyle injected had disappeared, and, no new ferment being formed, glycolysis was again reduced. Result: An increased excretion of sugar.

Experimental Series 20. A healthy dog was treated as in the above experiment; instead of injecting lymph, however, blood from a healthy animal was transfused. The comparative sugar excretion was as follows:

TABLE TO EXPERIMENTAL SERIES 19.

| | <i>Before transfusion.</i> | <i>After transfusion.</i> |
|-----------------------|--------------------------------|-------------------------------|
| First hour | 0.51 | 1.71 |
| Second hour | 1.07 | 1.57 |
| Third hour | 1.66 | 1.41 |
| Fourth hour | 1.91 | 1.87 |

It will be seen that similar fluctuations in the secretion of sugar occurred, though within narrower limits than in Experimental Series 19.

It was observed in the course of some of these and other experiments in which the pancreas was extirpated that in those animals that died from the operation prior to any other interference, and that developed symptoms of peritonitis or of sepsis (and there was a goodly number of such failures), a decrease in the initial great excretion of sugar occurred in several instances. V. Mehring, the discoverer of pancreatic diabetes, reports similar findings. We are inclined to the belief that in these cases the decrease of sugar was due, in part at least, to the phagocytosis that naturally occurred in the accidents mentioned and to the disintegration of leucocytes that resulted.

The following clinical case, moreover, that came to my notice almost has the value of an experiment:

P. E., aged forty-nine years, diabetic; average sugar excretion during one month, 2.11 per cent.; develops chill and sudden rise of temperature; lobar pneumonia of lower left lobe; sputum analysis reveals pneumobacillus; during the course of the disease, which terminated by

crisis in the night from the seventh to ninth day, a great reduction of the sugar excretion was observed; the average for the seven days was 0.61 per cent.; the lowest point, 0.39 per cent., was reached just prior to the crisis; it is uncertain whether the leucocytes played a rôle here, or whether the reduction of the sugar was due to a specific action of the pneumobacillus (the latter, it is stated, possesses glycolytic properties).

Similar observations are reported in the literature. It is my intention to study the effect of intercurrent infections on the course of experimental diabetes in order to clear up this question.

Experimental Series 21. After removal of the pancreas and determination of the sugar excretion as above, leucocyte extract—both the salt-water extract and the solution of precipitated blood—was injected into the jugular vein: one dog died suddenly while the fluid was still flowing into the vein, and another animal died twenty minutes after the operation. In the first case an embolism was found in the precentral artery, and a large thrombus in the crural vein; in the second dog a thrombus was seen in the first part of the femoral artery, and another one in one of the branches of the pulmonary artery of the left side; the blood removed from both dogs did not coagulate. The extracts, therefore, incorporated either the fibrin ferment or Lilienfeld's nucleo-histon, both chemically rather undefined substances; it has never, so far, been determined whether either of these bodies (?) possesses glycolytic powers—i. e., whether they are identical with the glycolytic ferment or not.

The injection of vegetable ferments with glycolytic powers has so far not been attempted.

The injections with trypsin and their results will be published later; for the present it can be stated that injections of doses larger than a milligramme are attended with serious symptoms of intoxication, and generally lead to the death of the animal in a very short time from various causes; at the same time the extreme toxicity of trypsin proves its great power of altering the course of normal processes; the same applies to many of our most useful remedial agencies; it is possible that when the correct dosage of trypsin is found, this substance may prove of value in the treatment of diabetes; it must be remembered that the glycolytic ferment is present in very minute quantities only.

The disastrous results obtained in animal experiments with leucocyte extracts and with trypsin have necessarily debarred us so far from attempting any of these measures on human subjects. From our animal experiments, however, it seems rational to attempt the infusion of lymph and the transfusion of blood in diabetes.

PRESENT METHODS OF TREATING URETERS SEVERED
DURING ABDOMINAL OPERATIONS.BY WILLIAM R. NICHOLSON, PH.B., M.D.,
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THE attempt to treat the ureter severed during abdominal operations is of relatively recent development. Indeed, until within the last few years the repair of injuries to these tubes was a sealed book, it being the general opinion that any injury demanded either a nephrectomy or at least a ligation of the lower end of the upper fragment. Thus, Herman Thompson,¹ of Odessa, writing as recently as eight years ago, advised nephrectomy as giving the only chance of recovery in any case in which the ureter was cut. The dangers, however, incident to the operation of nephrectomy caused abdominal surgeons much concern, and various methods have of late been proposed to avoid this mutilating operation. These procedures may be divided into three classes: First, anastomoses made with other viscera in the abdominal cavity, as into the bladder and bowel; second, reunion of the severed ends of the cut ureter or uretero-ureteral anastomosis; and, third, anastomosis with the external surface of the body, as upon the skin or into the vagina and urethra.

While injuries to the ureters are considered to be one of the rare accidents which occur during the course of abdominal operations, it is quite probable, as remarked by B. B. Davis,² that, if the truth were known, a percentage of deaths ascribed to shock and faulty anesthetization would be found in reality to be cases in which ureteral injury was the actual cause of the fatality. Injuries to the ureter may arise during the performance of operations in various ways. They may be caused by the inclusion of the ureter by a ligature at some point in its course, or by cutting or tearing it in the enucleation of tumors or inflammatory masses. The most usual situation of the traumatism is in the bottom of the pelvis near the base of the broad ligaments. The success attained in the repair of these structures has enabled operators to conscientiously sacrifice them in the removal of malignant neoplastic growths, and has even emboldened some enthusiasts to advise the extirpation of the whole bladder in cases of either a primary malignancy of the bladder or a malignancy of this organ secondary to carcinoma primarily in the uterus. Early in the serious study of this question the bladder anastomosis appealed to many men as theoretically the

¹ Zeitschrift f. Geburtshülfe u. Gynäk., 1893.² Journal of the American Medical Association, December 29, 1900.

proper method for the treatment of many of these cases. Hegar and also Paoli and Busachi¹ made various experimental anastomoses between the ureter and the bladder; and Bauman, in 1892, Novaro,² in 1893, and Penrose³ and Krug,⁴ in 1894, performed this operation upon human beings. The method here used was to pass a suture threaded at each end through the wall of the upper end of the ureter, the same having been slit for a short distance in order to prevent stricture. These needles are then passed into the bladder through a slit in its wall made at the point at which the anastomosis will produce the least amount of tension, and are then pushed through the wall, from within outward, a short distance from the edge of the bladder wound. Traction exerted by means of these sutures thus draws the ureteral end within the cavity of the bladder. The suture is then tied and the mucous membrane of the bladder above and below the ureter sutured with catgut, the connective tissue and peritoneal surfaces being closed after the manner of any ordinary bladder laceration. It is always to be remembered that the distal end of the ureter must be tied to prevent a backward flow of urine through the normal ureteral orifice, as this complication has been met with in a certain number of cases reported by Modlinsky⁵ and also by Pozzi.⁶ As to the indications for this form of operation, it will be found that quite a number of men advise the bladder implantation in all cases occurring below the pelvic brim, *i. e.*, where the distance from the point of injury to the bladder is not so great as to preclude the possibility of an anastomosis between them. Among this number may be mentioned Penrose, Baldy,⁷ and Krug. Kelly,⁸ on the other hand, while believing that the operation is a valuable one, thinks that it should not be allowed to invade the province of uretero-ureteral anastomosis. The last-mentioned operator would perform uretero-ureteral anastomosis in cases where both the severed ends of the ureter are easily accessible, when no obstruction is present between the lower end of the ureter and the bladder, and when there is no carcinomatous trouble necessitating the sacrifice of the lower portion. If, however, any of these complications were present he, too, would perform a uretero-cystostomy. One of the strongest advocates of uretero-cystostomy is Baldy, who has seen three cases in which this bladder anastomosis was employed, in every case

¹ Emmet. *American Journal of Obstetrics*, 1896.

² *Centralblatt für Chirurgie*, 1893, vol. xxvii.

³ Penrose. *University of Pennsylvania Medical Magazine*, April, 1894.

⁴ Krug. *Journal of Obstetrics and Gynecology*, 1894.

⁵ *Centralblatt für die Krankheiten des Harn-Sexual Organs*, vol. v. 1894.

⁶ Pozzi. *Annales des Maladies des Organes Genito-Urinaires*, 1891.

⁷ Baldy. *American Journal of Obstetrics*, vol. xxxiii., 1896; and *New York Journal of Gynecology and Obstetrics*, November, 1894.

⁸ Kelly. *Bulletin of the Johns Hopkins Hospital*, October, 1893, and February, 1895; *Annals of Surgery*, January, 1894; *Journal of the American Medical Association*, October 6, 1900.

with a satisfactory result. This operator claims that the limits of ureteral destruction, as set by some men, beyond which they do not advise the performance of the bladder anastomosis, are too narrow. He, with others, claims that this operation may be done with success at any point of the pelvic portion of the ureter. In his own case the ureter was cut above the iliopectineal line, but by passing a catgut stitch through the wall of the bladder, and thus attaching it to the stump of the ovarian artery, he was able to avoid tension on the ureteral stitches at the point of anastomosis. Kelly in a similar case of high ureteral injury dissected the bladder loose from its pubic attachments and dropped it back into the pelvis, thus producing an apparent lengthening of the ureter amply sufficient for the operation. Baldy calls attention to the fact that the majority of ureteral injuries occur in the lower portion, where most men are agreed that a uretero-ureteral anastomosis is a very difficult operation. He calls attention likewise to the apparent increase in ureteral length resulting from its dissection from beneath the peritoneum, by which its markedly curved course is converted into a straight line. The majority of those who have used the operation under discussion favor the technique of Van Hook,¹ or what may be called the free-hand method, in contradistinction to the method by the use of anastomosis forceps. This is the method which has been given above in brief. Kelly, however, at one time advised that Sanger's forceps, passed into the bladder by the urethra, and then through an incision in its wall, should be employed. The same author has more recently called attention to a ureteral guide, which he introduces into the bladder through a small incision in the wall and then passes out through the usual bladder incision. Upon the bar of this instrument thus passed the upper fragment of the ureter is threaded, and then is easily drawn into the bladder and the usual stitches applied. While this instrument does not have the objection of Dudley's forceps in that it is not passed by the urethra, it nevertheless necessitates the making of an extra incision in the bladder wall, which seems an unnecessary traumatism, since on the basis of the cases reported as done by the free-hand method the operation has not presented any marked difficulty. Among the objections urged against the operation of uretero-cystostomy there are some which bring interesting points into discussion. Thus Emmet² claims that the chance of infection in bladder anastomosis is greater than in the case of uretero-ureteral anastomosis, on the ground that in the former the normal orifice of the ureter is not preserved. Later studies have, however, shown that the so-called ureteral valve is probably non-existent, and that the real safeguard against infection is the presence of uninjured

¹ Journal of the American Medical Association, 1898.

² Loc. cit.

vesical mucous membrane. The absence of infection in the cases reported also offers strong evidence in support of this last-mentioned belief. As further evidence, it may be mentioned that in the experiments to be referred to later, in which the ureters were implanted in the bowel, it was found that the mere implantation of the ureteral orifices alone did not prevent infection, while if a portion of surrounding mucous membrane were transplanted uninjured with the ureteral orifice the chance of infection was greatly reduced. Bovée¹ is of the opinion that one of the possible dangers of the bladder implantation is that either a constriction of the ureter will be caused, with its resulting evils of slow hydronephrosis, or that a too free opening, with the absence of the valve, may permit a backward flow of urine, with the same result. These complications, however, have not, as far as our knowledge goes, been seen in practice, and theoretically it would seem unlikely that the tendency to stricture after uretero-cystostomy would be as great as in the case of either a uretero-ureteral anastomosis of the oblique, the end-to-end, or the end-in-end methods, since healing in all these takes place in a circular direction; while in the uretero-cystostomy, on the other hand, it occurs in an ellipse, and so may be considered comparable to the method of healing as observed in the end-in-side method of uretero-ureteral anastomosis. The backward flow of urine in any case of uretero-cystostomy, which is certainly even theoretically only problematical, will in practice be prevented by the high point in the bladder wall at which the anastomosis is made and by the use of the catheter at suitable intervals after the operation. It is, of course, true that the ureteral muscle-bundles running in the normal bladder from the ureteral orifice downward cannot be imitated by this operation, and likewise that the course of the anastomosed ureter is not oblique with reference to the muscular wall of the bladder, but perpendicular. While, however, these may be theoretical objections, they have not apparently given rise to any practical difficulties in the cases which have been reported as operated upon by this method.

The other method which has recently received the greatest amount of attention is the rejoining of the severed ureteral ends, or uretero-ureteral anastomosis. One of the chief drawbacks of this method of repair is the inherent difficulty of the operation, which Kelly² likens to the most difficult of those done upon the eye. The flaccidity of the walls and minuteness of structure of the ureter, as well as the depth at which most of these injuries occur, which necessitates that the repair be carried out in the bottom of the pelvis, have constrained Baldy³ to urge that bladder anastomosis be done whenever practicable. Kelly⁴ does not feel that these operations—i. e., bladder anastomosis and

¹ *Annals of Surgery*, 1897.

² *Loc. cit.*

³ *Loc. cit.*

⁴ *Loc. cit.*

uretero-ureteral anastomosis—are to be considered as rivals, but thinks that there are indications for the employment of each. He would prefer to do a uretero-ureteral anastomosis when both ends are accessible, when no obstruction is present between the lower end and bladder, and when the lower end does not have to be sacrificed to meet the indication of malignancy. If the above conditions are not fulfilled he would do a uretero-cystostomy. Kelly¹ advocates the use of his ureteral guide, previously mentioned, as an aid to the performance of the operation of uretero-ureteral anastomosis.

Different investigators have advocated different methods of uretero-ureteral anastomosis, and these may be grouped into four classes, as follows:

1. Lateral implantations, or the end-in-side method. Originated by Van Hook.
2. Transverse end-to-end approximation.
3. Oblique end-to-end approximation. Bovée's method.
4. End-in-end. Done by various experimenters, and once in a human case, the result in the latter being a success.

The dangers of these methods of anastomosis may be considered in the main to be those of urinary leakage, fistulæ, and constriction. These dangers are particularly threatening, as may be imagined, in the case of the end-to-end approximation—i. e., without invagination—and the end-in-end, with invagination. Bovée's method has only been used once in a human being, and there has been no experimentation carried out in animals, so there is not enough evidence upon which to base a verdict. Van Hook's method, on the other hand, originated in 1893, as a development of the experimentation of Poggi,² published six years previously, seems, on the authority of his own animal experimentation and also upon that of others, to be free from these objections to a great extent. Kelly³ was the first to use this method in a patient. His operation was performed in 1893, and Emmet⁴ followed with the second case in 1894.

Bovée,⁵ in 1897, was able to collect twelve cases of anastomosis of the divided ends of the ureter. Of these seven were done in Europe and five in America. Among these twelve cases three of the European cases died, but in no one of them could the death be attributed to the ureteral complication. The American cases all lived. In the series there was only one case which showed a leakage, and this soon ceased. But four of these twelve cases were drained, and the author is emphatically against the use of drainage for the ureteral injury *per se*, only advising it if pus be present. Three of the cases in the series reported

¹ Loc. cit.

² Loc. cit.

³ *Riforma Medica*, 1897.

⁴ Loc. cit.

⁵ Loc. cit.

were done by Van Hook's method. All of these recovered, but no positive statement is to be found regarding the development or absence of constriction as a late result. One case done by Poggi's method—the end-in-end invagination—was a success. Seven of this series were end-to-end approximations; and, although three died, there was no leakage in any of the seven, and the deaths were not due to the ureteral condition, as far as can be ascertained. The author's case was the twelfth, and was performed by making an oblique end-to-end anastomosis. It was successful.

As to the actual method of performing the operation of uretero-ureteral anastomosis, it is, first of all, as Kelly insists, of importance to determine the presence of any anomalies, and also to find out whether the ureter is "alive," or, in other words, patulous. Kelly was able to collect five cases from the literature in which the ureter was so occluded by disease that no urine could pass, and the operation, of course, was not indicated. If on palpation the kidney be found atrophic or sclerotic the operation is useless. It may be noted that the soiling of the peritoneum by the escape of urine is an accident of no importance, provided the latter is normal; but as abnormalities may readily be present it is well to try to avoid the soiling of the peritoneum in all cases by the use of pads during the suturing. The use of Kelly's guide is self-explanatory, the transverse bar being introduced into the lower fragment through an incision, and passed out of the ureter through the upper end of the lower fragment and into the lower end of the upper fragment, which is then invaginated into the lower and the suture inserted. The use of this guide enables one to have the aid proposed by Pawlik without the disadvantages of the catheter. Pawlik advised the retention of the catheter to assist drainage, but this is not advisable. In Van Hook's method the lower end of the ureter is ligated, and an incision is made into the wall a quarter of an inch below the point tied. This incision should be twice as long as the diameter of the ureter. By the application of traction sutures the upper end of the ureter is then drawn within the incision and the sutures are applied.

Early in the study of this subject the possibility of intestinal anastomosis presented itself to investigators. As early as 1851 it was performed in a case of exstrophy of the bladder by Simon, upon the suggestion of Roux. This case died in a year with marked kidney changes. Chaput and Leet were more successful, the former having one and the latter two cases which recovered. It is very easy to understand the desire of investigators to prove the applicability of intestinal anastomosis, as if feasible the operation would be much easier of accomplishment than uretero-ureteral anastomosis, on account of the size of the tube into which the ureter is introduced, and also because there would be a complete avoidance of the dangerous traction, which has been

one of the causes of many of the failures of the latter method of operation.

Notwithstanding the encouragement met by some investigators in this field, there seems to be one factor which is insurmountable, namely, the ascending infection, which involves the vast majority of all ureters thus anastomosed. Aside from this difficulty, there seems little reason why the intestines should not be thus utilized, as it has been found, as mentioned by Peterson,¹ that the large bowel in the dog can assume the functions of a urinary reservoir, and that sphincteric control will be maintained. It is to be noted also that while it seems to be the current belief that the mere presence of the urine in the intestines will cause a diarrhoea, there does not seem to be a unanimity in this opinion among many of the men who have had experience in both the human and the experimental cases in which this form of operation has been done. Thus Martin² expresses himself as not in accord with the view that urinary absorption is a great danger, and in quite a number of experimental and also of human cases it will be found that nothing more than a rather increased frequency in evacuations was caused, and that the incontinence of the bowels, so much feared by many, has not, as a rule, been observed.

Unfortunately, however, the question of the liability of ascending infection is one which no one as yet has been able to surmount. By this, of course, is not meant that no case has ever recovered from such an operation and lived for a long period, but that the percentage of cases exhibiting this form of infection is so high that the operation cannot be at present considered as justifiable. Of all the papers published on this subject, that by Peterson³ is the most thorough. The author is rather enthusiastic on the results of his method, which is a modification of Maydl's operation, by which the trigone is transplanted as a whole, thus avoiding any injury to the ureteral orifices, a point upon which Tuffier first insisted, with the idea that infection was prevented by the presence of the supposed ureteral valves. Later investigation seems to show, however, that, as is claimed by Peterson and others, the ureteral muscle in its sphincteric action has no preventive power as regards the ascending infection, and that there is no demonstrable valve at the ureteral orifices; but that the mucous membrane in the immediate vicinity of the orifices has the selective power. Peterson places the mortality of experimental intestinal implantations of the ureter as 61 per cent. in the case of the unilateral and 85 per cent. in the bilateral, and was unable to find a case in which kidney

¹ American Gynecological and Obstetrical Journal, 1900.

² Journal of the American Medical Association, January 28, 1899.

³ Ibid., February 16, 1901.

involvement did not occur sooner or later. The deaths in Peterson's series were due to peritonitis from giving way of the stitches or from uræmia. He collected a series of thirty-three operations by different men in human beings, and found a primary mortality of 33 per cent., with a final mortality still higher. He is able to show one case of unilateral implantation living after eight years, and one of double implantation alive and well after three and a half years. Of the twenty-two cases recovering from the immediate operation there were three subsequent deaths from pyelonephritis and two from uræmia. The author says he is unwilling to call any operation a success which will not bear the test of the double implantation. The above statistics apply to what the author calls the simple anastomosis—*i. e.*, an anastomosis in which the ureters were cut and the free ends inserted into the bowel. In forming his conclusions on the subject, Peterson made three series of experiments. The first of these comprises twenty-eight bilateral anastomoses, without preservation of the trigone. Of these dogs twenty-three died and five recovered, and among these recoveries there was no case which did not show gross lesions of kidney infection. In but one of the five, indeed, was the ureter not patent; but in four there was a distinct enlargement of the lumen, showing that a constriction was present. One of the most interesting facts brought out by this paper is that in one of the five recoveries it was found that the kidneys had been able to overcome the infection, and that they were sterile. The same was true in another of the five cases, in which instance the kidney was at the time of the examination recovering from a pyelonephritis, thus showing that immunity to the colon bacillus was being established. Of course, the cost of the development of such immunity was the production of contracted kidneys.

The second series of experiments comprised sixteen lateral uretero-intestinal anastomoses. Of this number there were but three recoveries. Twelve died from peritonitis because of leakage through stitch holes, and one from hemorrhage. The three recoveries were simply operative, as death followed in all after a short time from kidney infection and pyæmia.

His third group of experiments are those devoted to the implantation in the intestinal wall of the trigone of the bladder, and, while the results obtained are not particularly good, he explains a considerable number of the failures of his earlier operations as due to the fact that, inadvertently, the blood-supply of the trigonal area was destroyed, he believing that it was obtained from the ureteral arteries instead of the superior vesicals, as is really the case. This fault in technique he claims as the cause of the deaths in twelve of the cases of the series. He performed the experimental operation in twenty-one dogs, and reports the results as follows: Twelve deaths from peritonitis, due to

elaborated experimentally by Peterson, may in the future give better results; but at present, while his results seem more favorable than those of any other experimenter, they are not sufficiently encouraging to render their adoption justifiable in the human patient. There seems to be a certain amount of evidence to show that some cases may develop an immunity to infection, as in some of the human cases of anastomosis reported there was history of back pains, gradually disappearing; and among the experiments of some of the authors mentioned cases were noted at autopsy in which there was found a condition of contracted kidneys, and also in one case of Peterson's the dog's kidneys were found to be recovering from a pyelonephritis. Moreover, it is a well-known fact that the ureteral orifices, which in cases of exstrophy of the bladder are often liable to the contact of septic material, because of faulty care in diapering, sometimes resist infection for long periods. Reasoning from analogy, there seems some evidence also, as it is a well-known fact that in birds the urine and feces are deposited in a common receptacle or cloaca. This bird analogy has been advanced by Martin, among others, in support of his position, and he assumes that the production of this immunity to kidney infection may not be a matter of hereditary cycles, but that by improved methods of operating and after-treatment we may in the future be able to surmount the dangers of ureteral infection as we have already done in respect to infection of the peritoneal cavity. Van Hook, on the other hand, denies the applicability of the bird analogy, taking the ground that heredity plays an essential part in the production of such immunity, and, moreover, because of the difference between the character of the excrement voided by birds and that voided by human beings, the urine of the former being much less liquid than is the case with man, and therefore not as likely to carry an infection upward. Of course, arguments by analogy are rather dangerous until all of the facts of the case are in our possession; but it certainly seems a very questionable procedure to admit, as an operation of election, the anastomosis of the delicate ureter—perhaps the most carefully guarded excretory duct in the human economy—into an excretory system which, without exception, is always actively septic.

With regard to the remaining sites of ureteral implantation but little need be said, as from a practical stand-point none is of sufficient value to make its detailed consideration worth while. Though Bovée considered skin implantation about on a par with that into the bowel, and Martin has reported two cases of implantation upon the surface of the body, other men have determined that from the stand-point of infection it is even less trustworthy. Aside from this, the fact that a patient is debarred from all social duties, and becomes personally disgusting, would be sufficient to exclude this procedure from the list of legitimate

operations. The propositions of Van Hook, Nussbaum, and Rydygier, to connect the severed ends of the ureter by implanting each in the skin, and then, by plastic operations, to construct in various ways a channel between these ends, are, while interesting as experiments, of no value practically, as either bladder, uretero-ureteral, or bowel anastomosis are far preferable. Kelly considers skin implantation in the same class with nephrectomy.¹

The vagina as a site for ureteral implantation has the advantage of reservoir space, but this is the only possible advantage which it possesses, although Bovée has considered it to be preferable to the bowel, and, indeed, places it immediately after the method of anastomosis with the bladder. He considers that uretero-ureteral anastomosis is the best method of all. It is to be remembered that he considered the simple bowel implantation only, and not the trigonal implantation, which had not at that time been brought to the attention of the profession by Peterson.

Among the curiosities of the subject is the case reported by Boari² (case operated on by Nichaus), in which an anastomosis was made between the ureter and the urethra. This man recovered from the operation. Adolfo and Schwarz report anastomoses made in animals between the same structures, in which after a time a new bladder was formed from the urethra, and continence maintained by the development of a vesical sphincter from its muscle. Finally, there are two methods of dealing with a severed ureter to which consideration should be given, in order, in the one case, to unreservedly condemn, and in the other to advise the limitation of its application to the case alone in which the condition of the patient is so precarious that there is no time for a formal operation of anastomosis. The procedure to be condemned is nephrectomy, done first by Simon, in 1871, to meet the indications of ureteral fistulæ. While formerly there was an excuse for recourse to this operation, at the present time none can be offered. The other procedure is the aseptic ligature of the severed ureter—an operation hardly less dangerous than nephrectomy, and whose only claim for recognition rests upon the fact of the short time necessary for its performance. It is, of course, of paramount importance to be very careful that the ligature be done under the most rigid aseptic precautions, as otherwise a pyonephritis or peritonitis may result. As soon after ligature as the extravascular urinary pressure equals the intravascular blood-pressure

¹ Of interest but of no practical application to this question are the researches of Rosenberg and Bardenheuer on the subject of the metamorphosis of the epithellum of transplanted mucous membranes, upon which Van Hook based his supposition that the skin surface used as a link would finally exhibit the characteristics of the ureteral membrane.

² *Annales de Maladies des Organes Genito-Urinaires*, vol. xiv., Paris, 1896.

the function of the organ will be abolished, and atrophy of the kidney will in the majority of cases follow. The organ at first enlarges; but if the ligature has been well applied, thus insuring a sudden cessation of excretion, there will be, according to Orth, but little sacculation. Indeed, the danger of hydronephrosis is less in the case of complete ligature of the ureter than in many cases of uretero-ureteral and other forms of anastomosis. Guyon, who advised the use of the ligature in cases demanding speed, supports this claim, as do also Cohnheim, Straus, Albarran, Germont, and Byron Robinson. Wladimiroff,¹ however, while finding his results, as a rule, in accord with those of the investigators just mentioned, reports two cases of pyonephrosis in a series of nineteen ligations. Of course, this may be looked upon as due to some failure in technique; but notwithstanding the rather favorable results as reported by some men on the basis of animal experimentation, the operation should be considered as only applicable to the few cases in which the time element is of paramount importance.

In addition to the references given in the article the papers by the following authors have aided in the compilation of this review:

- Reynolds. *Boston Medical and Surgical Journal*, January 24, 1901.
 Schopf. *Allgemein Wiener medicinische Zeitung*, 1886, No. 31.
 Cushing. *Annals of Gynecology and Pediatrics*, February, 1898, vol. vi.
 Bloodgood. *Johns Hopkins Hospital Bulletin*, 1898, vol. iv. p. 89.
 Reed. *Journal of the American Medical Association*, 1896.
 Smith. *Philadelphia Medical Journal*, October 19, 1901.

¹ *Zeitschrift für Geburtshilfe und Gynäkologie*, 1898.

REVIEWS.

A TEXT-BOOK ON DISEASES OF THE EAR, NOSE, AND THROAT. By CHARLES H. BURNETT, M.D., E. FLETCHER INGALS, M.D., and JAMES E. NEWCOMB, M.D. Philadelphia and London: J. B. Lippincott Co., 1901.

In the preface of this book the statement is made that the close relation between diseases of the ear, nose, and throat renders it desirable that there should be a conjoint text-book on those diseases and their treatment. The project is not a new one, as most of the text-books on the ear also contain sections dealing with the relation of diseases of the nose and throat to that organ. So far, however, as we know this is the first endeavor to present a book written by three acknowledged authorities on the different topics under discussion and treating each of those topics as of equal importance with the others. The aim is a commendable one, and its execution has been admirably achieved. The trio of authors are all distinguished in the special lines whereof they treat, and each section of the work may be regarded as an exposition of the views of an authority in that branch. The only way in which such a book can be correctly considered is to take the different sections and consider them as distinct entities, each one constituting a small text-book in itself.

The section on diseases of the ear is written with a very exceptional lucidity and conciseness; matters of importance are emphasized and non-essential details are eliminated. Care is taken to emphasize the dangers attached to the use of certain measures which are very apt to prove misleading to those who do not make a special study of the science of otology. Thus the danger of recklessly incising furuncles in the external auditory canal, thereby providing a lodgement for growths of staphylococci, the dangers attendant upon the use of phono massage and vibratory massage, both of which are capable of producing a kind of boiler-maker's deafness, and the results which may follow attempts at the removal of foreign bodies from the ear, are all pointed out. The indications for the mastoid operation are considered, and the proper operation to be performed for each condition is described. Possibly one might ask for a fuller description of the various operations upon the mastoid with the different technique followed in each; but as this is readily obtainable in the larger works upon the ear and, after all, is of rather minor importance, such elaboration is not seriously missed. Some years ago Dr. Burnett substituted the removal of the incus for progressive deafness, in lieu of removal of the entire membrane and ossicular chain. The operation has certainly been successful in a large number of cases in which he has used it, relieving the tinnitus and vertigo and not increasing the deafness, although seldom improving the hearing to any extent. In the hands of others the operation has of late been used with similar gratifying results, and it undoubtedly has a large field of usefulness. A very valuable section of Dr. Burnett's work is devoted to chronic puru-

lent otitis media occurring in young children without external symptoms. The subject has of late assumed the greatest importance not only to the otologist, but to the general practitioner, and it is becoming more and more essential that all physicians should be familiar with the dangers arising from such a source.

The section on diseases of the nose and nasopharynx is written by Dr. Ingals in his usual interesting style. The arrangement of the chapters is somewhat peculiar, in that acute rhinitis and hay fever are embraced in the same chapter (III.), which is followed by a series of chapters on various subjects, before we come to Chapter VIII., in which the treatment of chronic rhinitis is taken up. Dr. Ingals is an advocate of the galvanocautery. In the removal of nasal tumors Dr. Ingals injects cocaine directly into the growth by means of a hypodermic syringe with a long silver nozzle; for the removal of adenoids he prefers forceps and general anæsthesia. The descriptions of the methods to be pursued in the various operations upon the nose are most excellent, and likewise the portions of the section devoted to the pathology of the various nasal diseases.

Dr. Newcomb's part of the work forms a fitting supplement to its predecessors. While concisely written and condensed, owing to limitations of space, it presents an excellent description of the various pathological conditions of the pharynx and larynx, with a summary of the most recent methods utilized in their treatment. The discussions of tuberculosis and syphilis of the larynx are very fair presentations of the present status of professional opinion on those subjects.

This book as a whole is one of the most useful contributions that has yet been made to the literature of laryngology, rhinology, and otology in this country. It possesses all the advantages of a book of composite authorship, while at the same time it is not a mere hodge-podge of conflicting views and statements. Although written expressly for students, no specialist in these subjects can afford to be without the book. The very brevity of the divisions renders their concise statements of facts particularly valuable. The book is handsomely got up, although, as seems inevitably the case in text-books of this nature, the illustrations are many of them old friends.

F. R. P.

DISEASES OF THE DIGESTIVE ORGANS IN INFANCY AND CHILDHOOD, WITH CHAPTERS ON THE DIET AND GENERAL MANAGEMENT OF CHILDREN AND MASSAGE IN PEDIATRICS. By LOUIS STARR, M.D., late Clinical Professor of Diseases of Children in the Hospital of the University of Pennsylvania; Consulting Pediatricist to the Maternity Hospital, Philadelphia, etc. Third edition, rewritten and enlarged. Illustrated. Philadelphia: P. Blakiston's Son & Co., 1901.

THE present volume has been changed in the following particulars: The forty-page chapter on the investigation of disease, appearing in the second edition, has been entirely cut out. Portions have been left out of the articles on the Eruption of the Temporary Teeth, from the Etiology of Enterocolitis, Cholera Infantum (including a clause relating to the differential diagnosis of this condition from sunstroke), a clause relating to the etiological relationship of dentition to gastric catarrh,

and a clause in the latter condition relating to dentition. Under the symptoms of *tænia nana* the paragraph relating to its similarity to mucous disease has been omitted, and two paragraphs under the treatment of intestinal parasites have been dropped. From the etiology and symptoms of *tabes mesenterica* and the chapter on Fatty Liver some of the old material has been dropped, and in the chapter on Suppurative Hepatitis the report of an illustrative case has been omitted. Numerous additions have been made to most of the articles in the book.

The following diseases are described for the first time in this edition: Rhachitis, scorbutus, lithæmia, Bednar's aphthæ, syphilitic stomatitis, membranous stomatitis, nasopharyngeal adenoid hypertrophy, proctitis, infectious follicular tonsillitis, brief descriptions of bothrioccephalus, and the *tænia cucumerina* and tuberculosis of the intestines. The article on Dysentery has been entirely rewritten, and the article on Typhlitis and Perityphlitis, under the heading of Appendicitis, has been extensively reconstructed. The chapter on Feeding has been altered in minor details, and been added to by a description of the method of milk modification initiated by Dr. Rotch, and here described as "Laboratory or Percentage Milk Feeding." It would probably have been better to have omitted the term laboratory milk for two reasons: In the first place, the method is as readily and perhaps as extensively used out of laboratories as in them. In the second place, the use of this term in the restricted sense of a milk "composed of centrifugal cream of 16 per cent. fat strength (usually) separated milk from which practically all fat has been removed by the centrifugation of the cream, 20 per cent. sugar of milk solution, sterilized lime-water and distilled water," is something of an injustice to the milk laboratories. The object of these institutions is to modify milk according to the physician's formula. They do not restrict themselves to the production of a milk composed of the ingredients above referred to. For instance, whole milk and gravity cream are very extensively used in some of the laboratories, while milk, cream, and whey mixtures are commonly used in all of them. In short, any combination which the physician uses in his home modifications can and will be prepared by any of the laboratories in compliance with the physician's directions.

Dr. Starr's results in feeding from the laboratory, after having "thoroughly tested" the method, lead him to make the statement that he has "never seen an infant from two to ten months old able to satisfactorily digest a laboratory mixture of stronger proteid percentage than 1.50, and has often seen cases of two months and more unable to digest a percentage of 0.50." A statement of this character coming from a man in Dr. Starr's position and one of his vast experience in the feeding of infants cannot lightly be set aside. It is a matter of some importance, however, that the vast majority of pediatricists in this country, some of them of even wider experience in this particular line, have had such directly opposite results, and have learned to look upon percentage feeding as done both in and out of the laboratory as one of the greatest advances in modern medicine. These facts would seem to far outweigh this individual opinion and to greatly lessen its value. The theory which Dr. Starr advances to account for his results is "that in its composition (percentage milk) all of the fat is removed by a separator, and the fat as prepared for the infant is a recombination of this fat and an alkaline solution of the proteids and sugar.

In a word, the natural emulsion is destroyed." This he thinks in some way lessens the digestibility of the proteids. If this is the case it is difficult to understand why any recombination of these ingredients, no matter how low the proteid percentage required, should prove digestible, and yet Dr. Starr recommends percentage feeding for premature infants and infants up to the age of two months. One would scarcely expect the digestions of immature and young infants to be able to cope with problems that those of older infants are incapable of solving. It is something of a question in the mind of the reviewer if the natural emulsion of milk cannot be said to be destroyed in any milk as soon as the cream is separated, even by gravity. If such be the case, any recombination of milk and cream must produce an unnatural emulsion. Aside from this, practically all the cream produced by the best dairies is a centrifugal cream; therefore, according to the author's view, most of the milk modifications in common use are artificial indigestible combinations, a view which can scarcely be supported by common experience.

The advisability of describing the nutritional disorders in a work on the diseases of the digestive organs may be questioned, but the articles on simple atrophy, scorbutus, and rhachitis are so excellent that anyone purchasing this work will feel deeply indebted to the author for having included them. Of special merit are the latter two. Indeed, the reviewer knows no better text-book description of these diseases.

Modern research has pretty clearly demonstrated Murchison's view of the condition which he described as lithæmia to be incorrect. Some of the recent text-books have dropped the term; others have retained it, but abandoned his idea of its pathology. It is somewhat surprising, therefore, to find among the new material in this edition a chapter on Lithæmia in which Murchison's original views are retained. It is unquestionably true that as the result of defective metabolism there are stored up in the economy various products of retrograde metamorphosis which give rise to numerous and varied symptoms. The process of metabolism is so complicated that it is reasonable to suppose that the innumerable symptoms which are described under the term lithæmia are probably dependent upon a variety of toxic products due to defects in more than one of the organs involved in the process, and are, therefore, not any more capable of being described by any one term than would be the symptoms of the various fevers by the general term fever. Under the heading Lithæmia, however, Dr. Starr has given a most interesting and comprehensive description of those symptoms which we commonly see, but concerning the origin, nature, and treatment of which we know so little.

S. M. H.

PEDIATRICS: THE HYGIENIC AND MEDICAL TREATMENT OF CHILDREN.

By THOMAS MORGAN ROTCH, M.D., Professor of the Diseases of Children, Harvard University. Third edition; rearranged and rewritten. Pp. xxi., 1021. Philadelphia and London: J. B. Lippincott Co., 1901.

THE latest edition of Dr. Rotch's *Pediatrics*, which, according to the title page, is technically the third edition, rearranged and rewritten, is a striking illustration of the importance of mere formal perfection in the

art of book-writing and book-making. The first edition of this really valuable treatise was a distinct disappointment to the many friends of Dr. Rotch, who had looked forward to the appearance of his *magnum opus* as an event of signal importance in the field of study in which its author had so brilliantly distinguished himself. Apart from its chapter on Infant Feeding, which alone would have marked an epoch in pediatric literature, there was much ground for criticism in the cumbersome lecture-room style, the lack of systematic classification and arrangement, and the undue prominence and discursive treatment of several divisions of the subject-matter, notably in those sections devoted to nervous diseases and the blood, which together occupied almost one-fourth of the whole book. A wealth of material, however, had been gathered together, and we have no doubt that Dr. Rotch realized the necessity for promptly revising the form in which it had been presented, even before his friendly critics had pointed it out.

The work of revision has been thoroughly carried out. The new edition contains just about one hundred pages less than the original volume, while nothing of importance has been sacrificed and considerable new matter has been added. This saving of space has been effected by eliminating unnecessary verbiage and quotation of cases, and by omitting certain of the least important illustrations.

The section on Feeding, which embodies the author's most notable contribution to pediatric literature, has been to some extent remodelled, and, if possible by slight changes, improved. We are pleased to see that Dr. Rotch has given much greater prominence than in his first edition to the subject of home modification on a percentage basis, and has added a section on the Theory of Percentage Modification, for which he has largely drawn upon the material of recent writers upon this subject.

In the line of additional material mention must be made of the excellent chapter on Typhoid Fever, which seems to have been altogether overlooked in the first edition; of a new section on Epidemic Cerebro-spinal Meningitis, which is thoroughly abreast of our present knowledge of this disease, and of a complete systematized article on Tuberculosis, which now for the first time receives treatment in a unified form. Cholera Infantum is properly classified under the general heading of Infectious Diseases, where, too, Influenza and Parotitis now find their proper place. A new section on Diseases of Nutrition gives a greatly improved, fuller, and better arranged section on Rhachitis than was found in the original text, while Osteomalacia, Scurvy, and Infantile Atrophy also appear under this heading in a new dress. The very unsatisfactory, because unsystematized, chapters on the Blood have been replaced by a well-arranged section on the Blood, the Lymph Nodes, and the Ductless Glands, which is entirely adequate. Finally, it remains to commend the satisfactory manner in which the original material covering Nervous Diseases has been condensed into a systematic review of the subject, occupying less than half the space previously devoted to it.

The only cause for regret is that lithæmia in childhood has not received attention in this new edition. This affection of children of the better classes is daily becoming more and more important in practice, and it deserves adequate consideration in the complete text-book of to-day.

Altogether, this new edition of Rotch's *Pediatrics* is practically a new book, well balanced, well arranged, and beautifully printed, which preserves all the excellencies of the old book and eliminates all its defects.

T. S. W.

A HANDBOOK OF MATERIA MEDICA, PHARMACY, AND THERAPEUTICS. BY SAMUEL O. L. POTTER, A.M., M.D., M.R.C.P. LOND., formerly Professor of the Principles and Practice of Medicine in the Cooper Medical College of San Francisco. Eighth edition, revised and enlarged. Philadelphia: P. Blakiston's Son & Co., 1901.

DR. POTTER's text-book has so long been esteemed an authoritative reference book on everything that pertains to the knowledge of drugs and their uses, that it seems difficult at first thought to realize that the seventh edition no longer was abreast of the times. A careful comparison of this with the latest revision, however, fully confirms the wisdom of the publishers in arranging for a new edition of this successful work.

Dr. Potter's unusual opportunities for study during his sojourn in the Philippines, among a strange people, and face to face with the little-known diseases of the tropics, has served to add much to his already able mastery of the subject of therapeutics, which earlier editions of his book have so fully attested. The text throughout has again been thoroughly revised, largely rewritten, and considerably increased by the addition of new material, which, however, has partly taken the place of material rejected as obsolete, so that the increased size of the book over the previous edition is only twenty pages.

A judicious conservatism has governed the admission of new drugs to a deserved place in the list of materia medica, only thirty-eight new articles having been added, from which those on urgentamin, argonin, creasotal, eucaïne, heroin, holocaine, orthoform, protargol, and urotropin may be selected as specimens of the class.

Many other changes in arrangement of articles and in addition of new materials to the older text can only be referred to in passing. We can recommend this new edition as in every way superior to its predecessors, which is, perhaps, the most substantial praise that could be accorded it.

T. S. W.

A LABORATORY COURSE IN BACTERIOLOGY, FOR THE USE OF MEDICAL, AGRICULTURAL, AND INDUSTRIAL STUDENTS. By FREDERIC P. GORHAM, A.M., Associate Professor of Biology, Brown University; Bacteriologist, Health Department, Providence, R. I. With 97 illustrations. Philadelphia and London: W. B. Saunders & Co, 1901.

PROFESSOR GORHAM has undoubtedly produced one of the best students' laboratory guides to the study of bacteriology on the market. While avoiding unnecessary detail, he has so arranged his material as to give a comprehensive, methodical survey of the entire subject. The tabular method which the author has adopted in dealing with the general technique, characteristics, and classification of the different groups of

bacteria is not only commendable from a logical stand-point, but also enables the student, and especially the student of medicine, whose time must be divided among so many important studies, to gain with the least amount of effort a general understanding of what otherwise would be an extremely difficult subject to master. The illustrations are good and well chosen, not only of apparatus, culture, etc., but also of the bacteria themselves—the latter being for the most part reproductions from photomicrographs. The whole work shows throughout a painstaking, judicious selection and condensation of material, which is highly creditable to the author, and which will be very grateful to the student, who would otherwise be compelled to wade and flounder through some one of the numerous larger text-books on this intricate and bewildering subject, much to his own and his instructor's disgust and despair. The technique is thoroughly modern, amply sufficient for all practical purposes, and so arranged that the student will have no trouble in following directions even to the minutest detail.

H. H. C.

CLINICAL HÆMATOLOGY: A PRACTICAL GUIDE TO THE EXAMINATION OF THE BLOOD, WITH REFERENCE TO DIAGNOSIS. By JOHN C. DA COSTA, JR., M.D., Assistant Demonstrator of Clinical Medicine, Jefferson Medical College; Hæmatologist to the German Hospital, etc. Containing 8 full-page colored plates, 8 charts, and 48 other illustrations. 8 vo., pp. 450. Philadelphia: P. Blakiston's Son & Co., 1901.

THE preface to this book states that it is "designed as a practical guide to the examination of the blood by methods adapted to routine clinical work, and represents an endeavor to record the salient facts of hæmatology as they are understood at the present time. . . . A minimum amount of theoretical discussion has been introduced in the sections dealing with the physiology and pathology of the whole blood and the cellular elements." The first four sections of the book, covering about two hundred pages, detail the examination of the blood by clinical methods, the study of the blood as a whole, the detailed study of the hæmoglobin, erythrocytes, and the leucocytes. In the first section the description of the use of the various instruments of precision is excellent. The writer gives preference to the use of the Oliver apparatus over the von Fleischl, though he recognizes the clumsiness of the Oliver instrument as compared with the one in more common use. The reviewer has had some experience with the Oliver apparatus, but has abandoned it in preference to Dare's hæmoglobinometer, which he has found to answer very well the requirements of hospital use. The sections upon the erythrocytes and the leucocytes show a considerable research of the literature upon these subjects, and perhaps bring up to date the most commonly accepted views upon the now much discussed subject of the value of leucocytosis. Diseases of the blood, chlorosis, pernicious anæmia, the leukæmias, Hodgkin's disease, etc., occupy but seventy-five pages. The plates accompanying this section are well drawn, though none of the blood plates seem to have the accurate coloring of those in Cabot's work. Splenic anæmia is classed among the essential anæmias, and appears under this section. Some of the recent writers on splenic

anæmia and anæmia associated with splenomegaly have reached the conclusion that this disease should be classed among the secondary anæmias. There is a special chapter on the anæmias of infancy and childhood, which is good. In this section von Jaksch's disease, the anæmia infantum pseudoleukæmia, is described, and quite properly to the reviewer's mind, not "as a separate clinical entity, but rather as a form of secondary anæmia associated with marked leucocytosis and splenic enlargement." Section VII., the last section of the book, discusses general hæmatology, and includes the author's experience as hæmatologist to the German Hospital as well as in hospital and private work, and also the statistics of other hæmatologists.

As a whole, the book does not carry out the interpretation of its title. Blood examinations cannot well rest upon clinical experience, but should have for their rational basis a consideration of the pathological conditions which produce them, and these are, in large part, wanting in the book. As a manual for practical work it is trustworthy.

J. A. S.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. For Practitioners and Students. A Complete Dictionary of the Terms used in Medicine, Surgery, Dentistry, Pharmacy, Chemistry, and the kindred branches, including much collateral information of an encyclopædic character, together with new and elaborate tables of Arteries, Muscles, Nerves, Veins, etc.; of Bacilli, Bacteria, Micrococci, Streptococci; Eponymic Tables of Diseases, Operations, Signs and Symptoms, Stains, Tests, Methods of Treatment, etc. BY W. A. NEWMAN DORLAND, A.M., M.D. Second edition, revised. 8vo. pp. 770 Philadelphia and London: W. B. Saunders & Co., 1901.

A SECOND edition of Dr. Dorland's work has been called for within a year, partly because of the popularity of the first, and partly in order that the author might fulfil his claim that the dictionary is up to date in every particular. Each twelve months at the present time adds a large quota of new medical words to the already overburdened list. Fortunately the inexhaustible stores of the Latin and Greek tongues are at the command of their makers, and the manufacture of new words can be continued indefinitely.

This dictionary is especially to be commended for its convenience as a work of reference. Although it contains 770 pages, the paper upon which it is printed is so light that the total weight of the book is very small. The definitions are concisely written and yet sufficiently explanatory. Special attention has been paid to the subject of pronunciation, and it would seem in many instances that the indications for pronunciation partook somewhat of the nature of a work of supererogation. Thus, in the case of ordinary English words of one syllable, such as flat, chalk, cord, and fit, it would hardly seem necessary to indicate the correct pronunciation, such indications merely serving to occupy space which might have been put to better use.

A number of excellent illustrations, rather above the average dictionary pictures, accompany the book, and tables, which serve in many

instances to simplify subjects and render them easy of grasp, are inserted. The book possesses especial value to the medical student or nurse who may desire a compendious dictionary in a handy form.

F. R. P.

FIRST AID TO THE INJURED AND SICK; AN AMBULANCE HANDBOOK.

By F. J. WARWICK, B.A., M.B. CANTAB., M.R.C.S., L.S.A., and A. J. TUNSTALL, M.D., F.R.C.S. EDIN. Philadelphia and London: W. B. Saunders & Co.

A WORK ON "First Aid" is, of course, not intended in any sense as instruction for the physician, but for the laity in general, and in particular for nurses, railroad men, hospital corps of militia, etc., so that of necessity it must be elementary in character and technical terms avoided as much as possible. The authors of this little book have succeeded admirably in producing a work of this description. No one, of course, doubts the importance of first aid in saving life and limb and the desirability of educating the public to look after such emergency cases till proper professional help arrives. The opening chapters are devoted to anatomy and physiology, and do not go very deeply into the subject. The chapters on Bandaging, Treatment of Hemorrhage, Sprains, Fractures, Poison Cases, etc., are well and plainly written. The concluding articles on the transportation of sick and wounded will be of especial interest to the medical officers in the army and militia and to their hospital corps. While the methods differ very little from those in general use by military men, and which are set forth in the manual issued by the Government for the drill and instruction of the hospital corps, in this work they are much more clearly expressed. On the whole it is a very useful little book, and one to be highly commended.

G. M. C.

A TREATISE ON PHARMACY, FOR STUDENTS AND PHARMACISTS. By CHARLES CASPARI, JR., Ph.D., Professor of the Theory and Practice of Pharmacy in the Maryland College of Pharmacy. Second edition, revised and enlarged. Pp. xiii., 766. Philadelphia and New York: Lea Brothers & Co., 1901.

THIS profusely illustrated book appeals to us not only by the evidence of scholarly thoroughness which it presents, but as well by the importance of the subject. That it has been written by a practical pharmacist there is ample evidence, and that the matter is intelligently presented the success which the book has attained bears testimony. In the first part, devoted to General Pharmacy, we find chapters on Pharmacopœias, Weights and Measures, Specific Gravity, Heat, Collection and Preservation of Crude Drugs, Mechanical Subdivision of Drugs, Solution, Percolation, Separation, Volatile and Non-Volatile Matter, Crystallization, and Classification of Natural Products Used in Pharmacy. Each one of these subjects is exhaustively presented and discussed.

The second part—Practical Pharmacy—takes up the various classes of medicines, washes, decoctions, extracts, etc., and gives their method of preparation, composition, with frequent notes of special precautions or a hint for advantageous work. Lastly, Pharmaceutical Chemistry takes up *seriatim* the various chemical classes, both organic and inorganic.

As we have read this book we have become more and more impressed with the importance of pharmacy and its claims to be recognized as a profession. We realize how much the advancement of the physician may depend on the concomitant progress of the pharmacist. While medicine continues to be practised drugs must play an important rôle in the treatment of disease, and subscription to this statement implies neither ignorance of nor the ignoring of the results obtained by so-called physio-therapy. The time is opportune for the undergraduate schools to insist that the student shall have a practical acquaintance with some of his armamentarium. A course in practical pharmacy supplementing the practical materia medica would lessen the mnemonic burden of the second year by giving him an object to which a name would adhere in mental image. Failing this, the recent graduate with this work in hand can readily acquire information which will be of practical value throughout his career. The author has done his work well; let us see his reward not only in better pharmacists, but as well in better informed and more useful physicians.

R. W. W.

ESSENTIALS OF PHYSIOLOGY, PREPARED ESPECIALLY FOR STUDENTS OF MEDICINE. By SIDNEY P. BUDGETT, M.D., Professor of Physiology in the Medical Department of Washington University, St. Louis. Arranged with questions following each chapter. Illustrated. Philadelphia and London: W. B. Saunders & Co., 1901.

THIS little work is intended to be used in conjunction with a textbook, not as a substitute for a more elaborate treatise on physiology. It admirably fulfils its purpose. A sufficient amount of space is given to the chemical aspects of physiology, and the author has succeeded in presenting the rudimentary essentials of this difficult and consequently much-neglected subject in a manner that should make it clear and comprehensive to the dullest student. Such modern subjects as the physiological action of the adrenals, the thyroid and the hypophysis, and the effects following removal of these glands, are conservatively treated. A number of tables diagrammatically explain the digestion of proteids, starch, and fats; they are well constructed and should prove helpful in memorizing the intermediate stages and the end-products of digestive dissimulation.

The full-page, half-tone illustrations of the fibre tracts of the spinal cord and their connections, of the cranial nerves, of the cerebral cortex, and of the structures concerned in the innervation of the cardiovascular system greatly enhance the value of this compend. The questions following each chapter are well selected and constitute a careful summary of the whole subject-matter treated.

A. C. C.

VORLESUNGEN UEBER DIE PATHOLOGISCHE ANATOMIE DES RUCKENMARKS.
Unter Mitwirkung von DR. SIEGFRIED SACKI, Nervenarzt in München,
Herausgegeben von DR. HANS SCHMAUS, A.O., Professor u. I. Assistant
am pathologische Institut in München. Wiesbaden: J. F. Bergmann.

IN many ways this is a remarkable book. It is the work of two authors seeking an ultimate knowledge, the correct understanding of the lesions underlying morbid states, and this end has been reached in an admirable manner by the joint efforts of an able clinician and an expert pathologist. We can cordially commend the idea to those laboratory workers who deal with dead-house material, and have no adequate conception of the clinical course of the disease which induces the lesions studied.

It is not too much to say that these lectures are the first in any language to set forth amply and thoroughly the correlation of anatomical structure and alteration with the various diseases of the spinal cord, and it is a pleasure to record that they not only constitute a marked contribution to our knowledge of the pathology of this portion of the nervous system, but they offer many excellent observations to the clinician as well.

With the recent marvellous studies on the anatomy of the nervous system, the intricacy of its structure is made appallingly definite; and whereas, heretofore, our knowledge of the diseases of these tissues has been almost entirely derived from the clinician, it is a sign of the times that anatomical research should step in and make clear many obscure points in spinal-cord pathology. Especially is this made apparent in this volume in the chapter on myelitis, a chapter which makes a veritable new outlook from the older stand-point.

The illustrative feature in the work calls for more than passing mention; it is a model of the highest type. The work is an invaluable one to the workers in this field.

S. E. J.

THE FOUR EPOCHS OF WOMAN'S LIFE. A STUDY IN HYGIENE. By
ANNA M. GALBRAITH, M.D., Fellow of the New York Academy of Medicine; Attending Physician to the Neurological Department of the New York Orthopedic Hospital and Dispensary, etc. Philadelphia and London: W. B. Saunders & Co.

THIS compact volume of 193 pages, including the most excellent glossary, aims to throw light upon the various periods of the life of woman. There is but little that merits adverse criticism in the subject-matter of the book. Indeed, the only important point to be called in question is the interpretation of hemorrhage during the period of the menopause. In a popular book of this nature it would have been certainly wiser to have impressed the reader with the idea that all such manifestations were to be considered as of a most dangerous nature, as it is safest to consider all aberrant bleeding at this period of life as due to carcinoma until proved otherwise.

The question, however, presents itself, as it always does in considering a book of this class, as to whether its publication subserves a good end. In other words, whether the harm possible of accomplishment when

placed in the hands of the young woman does not outweigh the intended good. While we do not wish to be considered as casting any reflections upon the intention of the author, we are nevertheless compelled to say very positively that we should be very averse to placing the volume in its present form in the hands of any young girl. W. R. N.

A TEXT-BOOK OF MEDICINE, FOR STUDENTS AND PRACTITIONERS. By DR. ADOLPH STRÜMPFEL, Professor and Director of the Medical Clinique at the University of Erlangen. Third American Edition, translated by permission from the Thirteenth German Edition. By HERMAN F. VICKERY, A.B., M.D., Instructor in Clinical Medicine, Harvard University; Visiting Physician to the Massachusetts General Hospital; Member of the Association of American Physicians; Fellow of the Massachusetts Medical Society, etc., and PHILIP COOMBS KNAPP, A.M., M.D., ex-President of the American Neurological Association; Clinical Instructor in Diseases of the Nervous System, Harvard University; Physician for Diseases of the Nervous System, Boston City Hospital; Fellow of the Massachusetts Medical Society, etc. With Editorial Notes, by FREDERICK C. SHATTUCK, A.M., M.D., Jackson Professor of Clinical Medicine, Harvard University; Visiting Physician to the Massachusetts General Hospital; Member of the Association of American Physicians; Fellow of the Massachusetts Medical Society, etc. 1242 pages, with 185 illustrations in the text, and 1 plate. New York: D. Appleton & Co., 1901.

WHEN a book has reached its thirteenth edition in its original German dress, and has been translated into eight languages—English, French, Italian, Spanish, Russian, modern Greek, Turkish, and Japanese—the author is certainly justified “in the assumption that it has exercised some little influence upon the professional thought and practice of numerous medical readers.” Of such a book it is pleasant to record the appearance of a third American edition. It is a book that not only reflects the best of contemporary German medicine, but it possesses the added charm of reflecting the personality of the author—for the book in many respects is a personal book. In an endeavor “to impart to the reader an insight into the origin and relation of the various morbid phenomena,” the author has “brought the facts of clinical experience into the closest possible relation with the data of pathological anatomy and of general pathology, and” has “endeavored, also, in discussing therapeutics, to deduce from the nature of the symptoms a basis for rational medical opinion and treatment, although” he “has not undervalued the importance of simple experience.” In the new edition a considerable portion of the book has been entirely rewritten—in particular, the whole doctrine of gastric diseases and several chapters in other sections, including gallstones, intestinal parasites, etc. We commend to the especial attention of the reader not only these chapters, but also chapters on the Diseases of the Myocardium, and the section of Diseases of the Nervous System, which for a long time has been recognized as a standard in this branch of internal medicine. The translators and the editor have performed their work well, and they have

added not only many notes throughout the book, but also special articles on the Plague, Yellow Fever, Dengue, etc., so as to adapt the work fully to the requirements of English-speaking practitioners. The book, as a whole, is to be most heartily commended. A. O. J. K.

A MANUAL OF CHEMISTRY. A Guide to Lectures and Laboratory Work for Beginners in Chemistry, Especially Adapted for Students of Medicine, Pharmacy, and Dentistry. By W. SIMON, Ph.D., M.D., Professor of Chemistry in the College of Physicians and Surgeons of Baltimore, in the Maryland College of Pharmacy, and in the Baltimore College of Dental Surgery. Seventh edition, thoroughly revised and much enlarged. In one octavo volume of 613 pages, with 66 engravings, 1 colored spectral plate, and 8 colored plates, representing 64 of the most important chemical reactions. Philadelphia and New York: Lea Brothers & Co., 1901.

THE seventh edition (1901) of this old and well-known text-book for medical students has just appeared, and continues as before a most excellent hand-book for the student of chemistry, whether from the medical stand-point or not. The book possesses several unique features. The most striking of these is the use of colored plates representing very accurately the appearance of 64 different tests and portraying faithfully the exact colors of each. No other text-book, as far as we know, has attempted this. It makes them very striking to the student, and will, without doubt, be of much aid in remembering certain important reactions.

The book is of 613 pages, and is divided into seven parts. Parts I. and II., "Chemical Physics and Principles of Chemistry," treat of the main laws that underlie the science and the basis on which it rests. Part III. takes up a study of the non-metals and Part IV. of the metals. The principal compounds of each are described, particularly those used in medicine. Both the chemical and the pharmaceutical nomenclatures are used. Part V. is devoted to a study of "Qualitative and Quantitative Analysis," including the detection and determination of the ordinary metals and acid radicals. The ordinary standard methods of analysis are given. In Part VI. we have a very excellent and concise summary of "Organic Chemistry." Many reactions are given, and a number of displayed or graphic formulæ of the benzene derivatives are introduced. Part VII., the last and perhaps the most important section of the book, is devoted to "Physiological Chemistry," including a study of the chemical changes in plants and animals, a study of the animal fluids and tissues (milk, urine, etc.), and a particularly good chapter on digestion, mainly from the chemical stand-point.

The book is to be welcomed to the shelves of advanced students as an old friend in up-to-date form, and will be of much value to the young student, who is always asking of the older men what book on chemistry can be recommended as a reference book.

It can be strongly recommended as a *precis* of much useful and valuable information, arranged in a manner both clear and lucid.

E. A. C.

SYPHILIS: ITS DIAGNOSIS AND TREATMENT. BY WILLIAM F. GOTTHEIL, M.D., Professor of Dermatology and Syphilography, New York School of Clinical Medicine; Dermatologist to the Lebanon and Beth-Israel Hospitals, the West Side German Dispensary, etc. Profusely illustrated, 216 pages. Chicago: G. P. Englehard & Co., 1901.

THIS small work presents to the general practitioner in a most compact form all the information upon the subject that he needs. In his classification of the secondary cutaneous manifestations of the disease the author has been most wise in avoiding the confusing and complex sub-divisions generally found in larger works on syphilis. While it seems to be the proper thing to add to all books treating of syphilis numerous plates intended to show the various cutaneous lesions, as a matter of fact these cuts rarely furnish any real information regarding the appearance of the lesions. The present work is no exception to this rule; the plates indicating secondary lesions might represent almost anything, those, however, showing tertiary lesions are decidedly better, some of them showing the actual appearance very well. While nothing new concerning treatment is suggested, the chapter devoted to this subject expresses in a concise form the views of the best syphilographers of the day. We are sure that the author's little work will prove of great value to those desirous of obtaining the most information upon the subject with a minimum amount of reading.

H. M. C.

DOSE-BOOK AND MANUAL OF PRESCRIPTION-WRITING. By E. Q. THORNTON, M.D., Ph.G., Demonstrator of Therapeutics in Jefferson Medical College of Philadelphia. Second edition, revised and enlarged. Pp. 362. Philadelphia and London: W. B. Saunders & Co., 1901.

WE note with pleasure the appearance of the second edition of this very convenient manual, presented in a more attractive form and increased to the extent of nearly thirty pages. The additions are chiefly in Prescription-writing, Incompatibilities, and chapters devoted to Poisons and their Antidotes, and to Synonyms in the appendix. Various new remedies of approved value are noticed and commented upon, but which are not to be found in the first edition.

No one who reads current American literature can fail to be impressed by the uncouth abbreviations, mixture of old and new terminology, combinations of alleged Latin and its vernacular, which are to be found under the guise of prescriptions. To remedy this such a book is useful, and especially when we may congratulate the author upon his scrupulous adherence to official and classical nomenclature.

We have read it carefully, and beyond a few typographical errors, as Felix mas, p. 347, find but little to criticise. It has demonstrated its usefulness, and we trust its popularity will increase even more rapidly.

R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

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ASSOCIATE PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

The Blood in Tuberculosis.—APPELBAUM (*Berliner klin. Wochenschrift*, 1901, vol. xxxix., p. 7) has studied the blood in cases of tuberculosis in Gerhardt's clinic with the following result: He divides the patients into those in the first, second and third stages of the disease. Patients in the first stage of tuberculosis may be considered in two groups. The first includes those in whom the disease has been of very gradual onset, patients who often have suffered from tuberculous affections (*scrofula*) since childhood. These are often tall, thin individuals, with characteristic phthisical chests, frequently with a family history of tuberculosis. In these cases the changes in the blood appear long before physical or bacteriological signs allow of a diagnosis. The second group are patients who appear in perfect physical health, well nourished, with well-shaped chests and without hereditary predisposition.

In the first of these classes there is well-marked anæmia, the red blood-corpuscles averaging about 3,800,000; hæmoglobin, 69 per cent.; leucocytes, 6500; specific gravity, 1049; solid residue, 19.5 per cent.

In the second class the blood examination is absolutely normal.

During the second stage of the disease, however, with the development of fever, night-sweats, and expectoration, the red blood corpuscles are often normal in number, 5,000,000 to 5,500,000; hæmoglobin, 90 to 95 per cent.; leucocytes, 8000 to 10,000; specific gravity, 1058 to 1060; solid residue, 22 to 24 per cent. This improvement the author justly considers to be only apparent, and due to the actual concentration of the blood.

In the third stage well-marked anæmia develops. The average number of red blood-corpuscles is from 2,000,000 to 2,500,000; hæmoglobin, 40 to 45 per cent.; specific gravity, 1040 to 1042; solid residue, 15 to 18.5 per cent. There is usually a leucocytosis of from 15,000 to 20,000.

The differential count of the leucocytes in the first stage, in those patients showing the picture of an anæmia, shows a relatively normal number of polymorphonuclear cells and a slight increase of eosinophiles at the expense of the small mononuclear elements. In the second stage the eosinophiles become less numerous, the transitional forms rather more numerous; the relative percentages of the lymphocytes and polymorphonuclear cells are but little changed.

In the third stage there is a well-marked relative increase in the percentage of polymorphonuclear cells. The eosinophiles are diminished and the transitional forms relatively increased in number.

[These observations serve to emphasize an interesting clinical fact upon which the reviewer has often insisted, namely, that during much of the course of pulmonary tuberculosis, though the face may be pale, a careful examination of the lips and mucous membranes will commonly reveal a color even equal to that of the normal individual, while examination of the blood shows a normal percentage of corpuscles and hæmoglobin.—W. S. T.]

Influence of Bile Retention on the Secretory Activity of the Stomach.—SIMNITZKY (*Berliner klin. Wochenschrift*, 1901, vol. xxxviii., p. 1077) states that the observations in literature upon the condition of the gastric juice in jaundice from various causes differ greatly. Some observers have noted superacidity, others subacidity, others nothing remarkable. Simnitzky, in Botkin's clinic, examined 12 patients with jaundice, 7 catarrhal jaundice, 1 Weil's disease with hepatic colic, 3 hypertrophic cirrhosis, and 1 obstructive jaundice following pancreatic cancer. In all of these cases there was a superacidity depending entirely upon the free hydrochloric acid and the total quantity of chlorides. This superacidity disappeared in every instance with the disappearance of the jaundice. In one instance, where there was a relapse of the jaundice, the superacidity returned. "The increase in the secretory function of the stomach is then clearly dependent upon bile retention."

In a series of experiments on dogs in which the gall-ducts had been tied after œsophagotomy, the quantity of HCl was found to be nearly 50 per cent. above normal.

In a second series, in which an isolated ventriculus had been prepared according to the method of Heidenhain-Pavlov, biliary obstruction was followed by a marked increase of hydrochloric acid. Further experiment showed that this superacidity appeared to depend upon an increased irritability of the gastric mucous membrane, which reacted with undue vigor to the normal stimulus, but which became equally rapidly exhausted. Thus, in dogs repeatedly fed with small quantities, the secretion of the gastric glands is in the end diminished.

Influence of Alcohol upon the Natural Resistance Against Infection.—GOLDBERG (*Centralblatt für Bakteriologie und Parasitenkunde*, 1 Abth., 1901, vol. xxx., pp. 696, 731) states that as a result of an interesting series of experiments made in the laboratory of Professor Chistovich (Tschistowitsch) the author arrives at the following conclusions:

1. Doves which are naturally immune against anthrax become subject to

infection after moderate doses (2 to 8 cm.) of 40 per cent. brandy, which produce only transient alcoholic intoxication but do not result in the death of the animal.

2. Chronic alcoholic intoxication diminishes the natural resistance of doves against anthrax.

3. Small doses of alcohol repeatedly given to doves infected with fatal doses of a culture of anthrax do not save the life of the bird, and only exceptionally prolong their lives in comparison with control doves; sometimes they cause apparently earlier death of the bird.

[These observations are in part confirmatory of those of Abbott made upon rabbits.—*Transactions of the Association of American Physicians*, 1895, vol. xi., p. 421.—W. S. T.]

The Origin and Prevention of Deposits of Oxalates in the Urine.—

Bearing in mind the fact that from 30 to 50 per cent. of all renal calculi consist mainly of oxalates, KLEMPERER and TRITSCHLER (*Berliner klin. Wochenschrift*, 1901, vol. xxxviii., p. 1289) have made some interesting observations. If, after an ordinary meal of spinach, about 100 mgr. of soluble oxalic acid is set free in the gastric juice, about 15 mgr. appear in the urine. The rest is, in greater part, destroyed by bacteria and ferments in the intestine. Small quantities of oxalate of sodium and oxalate of lime introduced hypodermically appear in toto in the urine, showing apparently that whatever amounts of oxalates enter into the fluids of the human body pass unchanged through the kidneys. But in human beings or animals fed upon a diet entirely free from oxalic acid, oxalates may yet appear in the urine. This they have shown to be due to the fact that oxalic acid may appear as a result of the digestion of kreatin, glyocol, or glycocholic acid. Thus, in a starving dog, oxalic acid appearing in the urine doubtless develops from the kreatin of the breaking-down muscle.

In order then to make the urine entirely free from oxalic acid, it will be necessary that the nourishment shall contain neither oxalic acid nor kreatin; in other words, no vegetables or meat, and yet sufficient nourishment to prevent loss of body albumin. This will scarcely be possible; but, as the authors point out, the question is not how to prevent the formation of oxalic acid, for oxalic acid, when in solution, is of no harm, but how to prevent the precipitation of oxalate of lime. As a result of a number of analyses the authors have concluded that the solubility of oxalate of lime is proportionate to the quantity of magnesia and in inverse proportion to the quantity of lime in the urine. The best diet for a patient with a tendency toward oxaluria should then be one in which milk, eggs, tea, cocoa, and many vegetables are forbidden, while meat, fat, bread, grains, rice, and the legumens, as well as apples and pears, are allowed. Klemperer does not recommend the absolute prohibition of vegetables, but only spinach and the various forms of cabbage, on account of the abundant quantity of lime which they contain. Moderate quantities of other vegetables should be allowed. The patient should be encouraged to drink much water; there is no especial contraindication for alcohol or coffee.

The quantity of magnesia in the urine may also be influenced by the administration of 2 grammes of sulphate of magnesium a day.

Degeneration of the Islands of Langerhans of the Pancreas in Diabetes Mellitus.—WRIGHT and JOSLIN (*The Journal of Medical Research*, vol. vi., No. 2, November, 1901, p. 360) refer to the work of Lagnesse, Schäfer, Diamare, Ssobolew, and Opie on the degeneration of the islands of Langerhans of the pancreas in diabetes mellitus and the probable disturbance of carbohydrate metabolism resulting from this lesion.

Among fourteen cases of chronic interstitial pancreatitis examined by Opie, the islands of Langerhans were shown to be the seat of hyaline degeneration in four. In three of these four cases diabetes had been present, but had been absent in the others. In the fourth case the islands were only slightly affected. What is more important, he found in two cases of diabetes mellitus in which there was no increase in the interstitial connective tissue of the gland, that these islands were extensively transformed into hyaline material. In one case the degeneration was strictly limited to the islands, while in the other it extended beyond their limits. As a result of his observations Opie concluded that in pancreatic diabetes the lesion is of such a character as to destroy the islands of Langerhans, and that where, although the organ is diseased, diabetes is absent, the interacinar islands are relatively unaffected.

Wright and Mallory examined sections from the pancreas in nine cases of diabetes mellitus and found the islands of Langerhans degenerated in two. In one of the cases the degeneration was very marked in these islands and practically confined to them. In the other case the islands were less markedly involved, and the gland also showed fat necroses. The remaining seven glands were practically normal. It is well to note, however, that most of the material examined was in the form of stock sections in the laboratory, which had not, of course, been specially stained with the object of demonstrating this lesion.

The writers think, nevertheless, that their results are strongly in favor of the hypothesis that lesions of these islands are important factors in the pathology of this disease.

Bacteriolysis and Typhoid Immunity.—MARK W. RICHARDSON (*The Journal of Medical Research*, vol. vi., No. 1, p. 187), in the course of some interesting laboratory experiments, has made some observations which are most suggestive and seem to have an important bearing on the question of natural immunity and of immunity after typhoid fever. During the course of typhoid fever, if a quantity of fresh blood serum be added to an equal quantity of a bouillon culture of the typhoid bacillus, there is, in the vast majority of cases, not destruction but abundant multiplication of the typhoid organisms. If, however, the mixture of serum and bacilli be introduced into the peritoneal cavity of a normal guinea-pig we get the so-called "Pfeiffer's phenomenon," that is, there is not only a complete absence of multiplication of the bacilli, but there is absolute destruction and disappearance of the organisms. The essentials for the reaction are the specific typhoid or immune serum and also the tissues or fluids of a normal living animal. Richardson proceeded to ascertain whether results similar to the above could not be obtained with typhoid bacilli and sera studied by the hanging-drop process. In this he was successful. It was found that if a

loop of typhoid blood serum were added to a loop of a twenty-four-hour culture of the typhoid bacillus and observed in the hanging-drop under the microscope, there was inhibition of the growth of the bacilli, although they still multiplied slightly, but there was absolutely no evidence of destruction of the bacilli. If, however, a loop of normal serum were added to the mixture of typhoid serum and bacilli, it was observed that the typhoid bacilli were almost completely destroyed within an hour. In other words, the addition of the normal serum had the power of producing a bacteriolysis. Thus the observation of Bordet, that Pfeiffer's phenomenon could be observed under the microscope, was confirmed. It was at first found that this reaction was not constant, and it was afterward ascertained that the result was always constant if the three ingredients were added in a definite order. The two sera must not be mixed before being brought into contact with the bacilli. The bacilli must be mixed with one of the sera first before the other serum is added.

Observations were made to see what effect the serum of typhoid patients had at various stages of the disease. In the middle of the disease typhoid serum had no influence in causing bacteriolysis. The addition of normal blood serum caused rapid bacterial destruction. On the other hand, with the fall in temperature, the blood serum of the typhoid patient possessed definite bacteriolytic power. Thus, in the late stages, it acquired properties similar to those produced by the addition of normal serum. The practical importance of these experiments is that they throw some light on the unsatisfactory results obtained so far from serum therapy in typhoid. They indicate that by administering the curative serum there has been added something that the blood already contains in abundance. There is wanting, however, a factor which seems to be only supplied by normal blood serum. The writer seems to have demonstrated that in serum therapy in the future normal serum must also be administered in addition to the curative serum.

On the Occurrence of *Strongyloides Intestinalis* in the United States.

—THAYER (*The Journal of Experimental Medicine*, vol. vi., No. 1, Nov. 29, 1901) reports three cases of infection with *strongyloides intestinalis* observed in the Johns Hopkins Hospital during a period of three years. These are the first cases that have been reported in this country.

The report of the cases is preceded by a detailed account of the life-history of the parasite and the methods of development of the larvæ into the sex-ripe generation. The parasite is the recognized cause of the so-called Cochin-China diarrhoea, and was first discovered by Normad, a French naval surgeon, in the dejects of individuals who had contracted severe diarrhoea in Cochin-China. Bavay, who carefully studied this parasite, gave it the name *anguillula stercoralis*. The following year Bavay found another small nematode worm in the small intestines of patients suffering from Cochin-China diarrhoea in association with the *anguillula stercoralis*. To the new parasite he suggested the name *anguillula intestinalis*. It was later decided that the two parasites were identical. In 1878 and 1879 Grassi and Parona discovered the parasite at a number of autopsies in Pavia, and in the latter year Grassi placed the worm in a special genus closely

allied to the strongylus, which he termed the strongyloides, and gave it the specific term *strongyloides intestinalis*. This classification is generally accepted. In the early eighties the *strongyloides intestinalis* was frequently found in association with the *anchylostoma duodenale* in the intestinal tract of workers in the St. Gothard tunnel. Until the report of Thayer's three cases the parasite had not been observed in the United States. The infection is recognized by the presence in the stools usually of the actively motile embryo, which in Bavay's case measured 0.33 millimetre in length, and 0.022 millimetre in width. The length of the full-grown female was about 1 millimetre; its breadth about 0.04 millimetre. The male was about one-fifth smaller than the female. A characteristic feature of the parasite is the occurrence of two bulbous enlargements of the œsophagus, the posterior of which is armed with three chitinous teeth.

The parasite apparently does not produce intestinal ulceration excepting in very rare instances. The injurious effects of the worm are supposed to be purely mechanical, although Calmette suspects that the parasite may give rise to substances acting as chemical irritants. The clinical manifestations of the disease are mainly those of a chronic diarrhœa and a comparatively mild anæmia. There are usually three or four pasty stools a day. Doses of thymol followed by a purge have given best results.

The first of Thayer's three cases had amœbic dysentery and intestinal infection with *trichomonas intestinalis*. The patient also had an hepatic abscess, and the case terminated fatally. The infection in this case may have occurred in Austria. The infections in the two other cases must have taken place either in Maryland or in Virginia.

The writer expressed the probability that infections with *anchylostomiasis* also occurred in this country. While the article was in press such an infection was found by J. L. Yates at an autopsy in the almshouse, Baltimore. A strong plea for thorough systematic examinations of the feces is made.

On the Toxicity of the Cerebro-spinal Fluid in Epileptics.—Bellisari has shown that the cerebro-spinal fluid of individuals suffering from general paralysis is more toxic than in normal individuals, and that this toxicity is at its maximum after an epileptiform seizure. PELLAGRINI (*La Riforma Medica*, Rome, 1901, Ann. xvii., vol. ii. p. 638) has continued these researches with a view to determining the relative toxicity of the cerebro-spinal fluid in epileptics. In his researches the puncture was made between the third and fourth and fourth and fifth lumbar vertebræ. The quantity of fluid contained varied from 10 to 15 c.cm. Punctures were made in six cases of epilepsy. As a result of the inoculation of these fluids into guinea-pigs the author arrives at the following conclusions:

1. That the cerebro-spinal fluid of epileptics is possessed of a markedly toxic power.

2. That, injected into guinea-pigs, it results always in grave and intense convulsive phenomena, so much so that in some instances a status epilepticus is produced; it has, therefore, a convulsive power.

3. That cerebro-spinal fluid, extracted immediately after the convulsion, has a toxic and convulsive power considerably greater than that obtained at periods far removed from the paroxysm.

4. That the so-called anti-epileptic drugs exercise no influence upon the toxicity of the cerebro-spinal fluid.

5. That cultures upon broth and gelatin from the cerebro-spinal fluid of epileptics remain sterile. Lumbar puncture appears to have no influence upon the course of the disease.

Gangrene of the Extremities after Scarlet Fever and Other Infectious Diseases.—EICHHORST (*Deutsche Arch. f. klin. Med.*, 1901, lxx., 519) reports a case of gangrene of the left leg occurring during a very severe case of scarlet fever in a child, aged four years. On the sixteenth day of the disease, during desquamation, the child complained of pain in both legs, with tenderness on pressure in the muscles. During the night and on the morning of the fourth day after this the lower half of the left leg and foot were found to be pale and cold. This gradually went on to gangrene, for which, after two weeks, amputation was performed, the child making a satisfactory recovery.

The popliteal artery was found to be clear above, but the lumen appeared narrow and the wall was somewhat thickened. About 1 cm. above the point of division of the popliteal the vessel was entirely closed by a thrombus which extended about 1 cm. down into the two branches. In microscopical sections it was shown that the thrombus started from a very small area of endarteritis. Just above and below this point the arterial walls were quite free from change. No bacteria were found in the vessel wall or in the thrombus.

Gangrene of the extremities in scarlet fever is a very unusual complication. Eichhorst has found but two similar cases in the literature. In one of these instances, that of Pearson and Littlewood, the scarlatinal process was extremely mild. In all three cases the thrombosis occurred during the period of desquamation, though in one instance it was as early as the ninth day. The onset of the occlusion was sudden in all cases. In two the pain in the legs was complained of for a short time beforehand, while in one discoloration of the skin was the first striking change. Eichhorst points out, in an interesting manner, that the sudden occlusion of an artery is by no means an evidence of embolism. Not a single one of Eichhorst's cases of cerebral thrombosis has had other than a sudden onset, similar to that seen in embolism or hemorrhage. A sudden onset appears to be the rule, also, in thrombosis of the arteries in the extremities. The author reports a case of autochthonous thrombosis of the right axillary artery occurring in influenza with apoplectiform onset.

Eichhorst has collected and tabulated 166 observations of arterial thrombosis in the extremities during acute infectious diseases; 42 of these occurred in typhus, 40 in typhoid fever, 19 in influenza, 14 during the puerperium, and 10 in pneumonia. The commonest seat of the thrombosis was in the legs.

Under all circumstances, arterial thrombosis in the extremity is a very serious complication. Death occurred in 39 per cent. of these cases. In many of these cases proper pathological investigations are wanting, but in 65 the thrombus in the artery was demonstrated. Satisfactory reports as to the condition of the heart are, however, wanting. In the majority of cases,

however, the onset was not as acute as in the author's instance, and he is inclined to believe that in most instances the condition was secondary to an endarteritis rather than to embolism.

An Analysis of Nine Hundred and Forty-nine Cases of Pneumonia.
—SEARS and LARRABEE (*The Medical and Surgical Reports of the Boston City Hospital*, Twelfth Series, December 1, 1901) have made an elaborate analysis of 949 cases of lobar pneumonia which came under treatment in the second and third medical services of the Boston City Hospital, from the years 1895 to 1900, inclusive. Cases in children under fifteen years of age were excluded. The disparity in the number of cases in the two sexes, 714 males and 235 females, was in part attributed to the greater hospital accommodation for the former sex, but mainly to the more frequent abuse of alcohol by men. In 660 cases the disease began suddenly; in 466 with a chill. In 238 cases in which the presence or absence of abdominal symptoms was noted, nausea or vomiting occurred 45 times, diarrhoea 20 times, both nausea and diarrhoea 12 times, a total of 77 (nearly 33 per cent.) Pain below the costal margin was frequently present, and in several cases appendicitis was simulated, especially when the pain was associated with muscle spasm. The right lung was affected nearly twice as often as the left. Their statistics do not strongly support the statement, so generally made, that the disease is more fatal or even more severe when it attacks the upper lobe. The temperature fell by crisis (i. e., reached normal within forty-eight hours from the commencement of the drop) in 424 cases. The earliest date upon which it occurred was the second day, and the latest the nineteenth. By far the largest proportion of crises occurred between the sixth and ninth days. Death occurred in 29 cases after crisis. The average day for complete deferescence in the cases with lysis was 12.37, in those with crisis it was 9.45, showing that crisis shortens the course of the fever relatively by nearly three days. Of the 949 cases, 341 (35.9 per cent.) died. Of these 91 were moribund on admission. If these be excluded there still remains a mortality of 29.1 per cent., which the writers say leaves much to be hoped for. The percentage of deaths among males and females was 36.5 and 34 respectively. The harmful effects of alcohol were clearly shown. For total abstainers the death-rate was only 25 per cent., whereas 45.5 per cent. of the cases occurring among hard-drinkers ended fatally. The death-rate bore a direct ratio with the amount of albumin in the urine. Albumin was found in 624 out of 799 cases in which it was tested for. The leucocyte count showed that the mortality was much greater when the leucocytes were below 10,000 than when they were above this figure. The peculiar tendency of pneumonia to recur was shown by the fact that 86 patients had had one or more previous attacks. The highest number was 7; three was not very unusual. Autopsies were obtained in only 72 cases. In 36 of these the pneumococcus was isolated either alone or with other organisms from the blood. No ante-mortem blood cultures were taken. Pleurisy with definite signs of effusion occurred in 53 cases. In 34, of whom 8 died, it was presumably serous. In 19 it was purulent, of whom 7 died. Pericarditis was diagnosed in 19 cases, death occurring in 11. Phlebitis occurred in 10 cases, the largest number involving the veins of the left lower extremity. Meningitis was

diagnosed in 7 cases, autopsies being obtained in 3 of these. In these three cases the meningitis was regarded as a manifestation of a general infection, as an acute endocarditis was present in all. Acute endocarditis was found in 9 cases at autopsy, most often at the aortic valve, which was affected in 7 instances, 5 times alone and once each with the tricuspid and mitral. The mitral was involved alone twice. Eight of the cases were due to the pneumococcus. Otitis media was present in 16 cases. Regarding treatment, those who received cold sponges or cold baths showed a slightly higher mortality (1.7 per cent.) than in those who did not receive them.

SURGERY.

UNDER THE CHARGE OF

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A Clinical Lecture on Idiopathic Dilatation of the Oesophagus.—SWAIN (*British Medical Journal*, November 16, 1901) states that dilatation of the oesophagus which exists without any obvious mechanical obstruction is a rare condition. The author reports the case of a woman, aged twenty-six years, who complained of difficulty in swallowing and a sensation of choking followed by vomiting, especially at night. There was difficulty in swallowing and considerable pain in the chest, which, however, was relieved by vomiting. Examination showed that a tube could be easily passed into the stomach. After washing out the stomach through the tube and then withdrawing it about eight inches of undigested food could be washed out of the oesophagus. The reaction of the fluid removed from the stomach was acid, while that from the oesophagus was neutral. Treatment has not proved very satisfactory—electricity, tonics, and feeding with the stomach-tube have given no relief. A comparative degree of comfort only has been obtained by the ingestion of soft foods and the systematic lavage of the dilated oesophagus. The question of gastrostomy has been considered, but has not been thought advisable as long as the patient is keeping up her nutrition.

The After-care of Cases which Have Been Operated upon for Perforation of the Stomach.—GILFORD (*British Medical Journal*, November 16, 1901) states that the most important points in the after-treatment of these cases are the food, the position occupied by the patient, and the use of aperients. Food: This should of necessity be liquid, either previously digested or of such a character as to require no gastric digestion. If the

indications are favorable, nothing but water should be given during the first twenty-four hours. If the operation has been severe, or if there is reason to suppose that the stomach will not tolerate liquids, it is best to inject a pint of warm water into the rectum to begin with. This may be repeated every four hours, and the quantity should vary according to the tolerance shown by the patient and the degree of thirst. In the great majority of cases hot water may first be given by the mouth four hours after the operation, but only a teaspoonful at a time should be allowed, and care should be taken that it is really hot. All fluids should be stopped as soon as nausea is felt, and should not be given again until after the lapse of an hour or more. When it is found that no nausea or sickness is produced, a little food may be introduced with advantage. Milk is a bad food to begin with, for the reason that it contains proteids, and therefore undergoes digestion in the stomach. Peptonized milk is equally objectionable, for the reason that peptones are decidedly irritating, and the milk so treated is not palatable. While food should not be unpalatable, it must not, on the other hand, be too rapid or stimulating. For this reason, beef-tea and the other watery extracts of meat are objectionable. There is a similar objection to the use of alcoholic drinks, such as whiskey and champagne, whose stimulating action entirely overshadows their nutrient value, whatever that may be, and they should be considered rather as drugs than as foods. The combinations of alcoholic drinks with eggs are most undesirable. The best food that fills all the requirements is grape sugar flavored with raisins. It is best made by pouring boiling water on to half its bulk of chopped raisins. This is stewed for two hours and then filtered. The filtrate may be given either with water or without, and either hot or cold, according to the wishes of the patients. The author states that a decoction of malt made in a similar manner has proven equally serviceable, but, as a rule, it is not as palatable as the "raisin tea." After raisin and malt have been given from one to three days, a mixture of cream and water should be added to the list. This consists at first of one part of cream to twelve of water, but it is gradually made stronger until it is double this strength. These three preparations may be continued until the end of the first week or longer, but generally after the cream has been given for a day or so, one should mix with it some starchy food containing not more than a trace of proteid, such as arrow-root or corn flour. At the end of a week, if the patient has done well, solid food may be commenced, and to begin on it it is best to select those foods that undergo intestinal rather than stomach digestion.

The position of the patient should be regulated by the physiological requirements and the position of the ulcer. In the case of ulcers on the posterior surface the patient should first be placed well over on to the right side, and should afterward assume the sitting-up position, or a compromise may be affected by raising the shoulders and turning the patient slightly over to the right. This position is good for all ulcers except those which are low down on the anterior wall, and it may then perhaps be best for the patient to be lying on his back.

An enema of four ounces of glycerin mixed with half an ounce of turpentine should be given twenty-four hours after the operation; the time should vary according to the amount of distention and eructation of gas. Soap and

water with turpentine may also be given, and either of these may be repeated every four hours until decided relief is obtained.

The End Results of Castration for Tuberculosis of the Testicles.—
BRUNS (*Archiv f. klin. Chir.*, Band lxiii., Heft 4) states that the importance of this subject cannot be over-estimated. As the result of a discussion before the Paris Society of Surgeons two years ago, it was determined that the majority were against castration. Briefly the arguments that have been urged against castration are: 1. It is often unnecessary to sacrifice the whole testicle, as almost invariably it is only the epididymis that is affected. A destruction of the tubercular area or the excision of the epididymis suffices in these cases. 2. Excision of the testicle is often insufficient, as other portions of the urogenital apparatus may be affected. The author's experience has been that tuberculosis of the testicle itself is usually secondary to tubercular infection elsewhere in the urogenital tract. 3. The excision of both testicles or a double vasectomy is not to be considered, because of the severe psychic disturbances which almost invariably follow, and also in tuberculosis of each testicle there is usually infection of the prostate, seminal vesicles, and other portions of the urinary tract. The author reports 111 cases in which operation was performed; in 78 cases a one-sided castration, and in 33 cases both testicles were removed. In all these cases the vas was resected high up, but the prostate and seminal vesicles were not touched. A careful study of the history of these cases showed that in the first six months after the appearance of the disease the head of the testicle had become involved. In the Berne clinic in 75 per cent. of the cases the involvement of the testicle was bilateral. It is a question whether or not early castration can prevent this large percentage of involvement on each side. In 38 per cent. of the author's cases the disease was bilateral. In those cases in which castration of one side was performed, 23 per cent. ultimately developed tuberculosis of the other testicle, which usually is apparent within two years after the castration. It is probable that in fully one-half of these cases both testicles were involved at the time of operation. Of the one-sided castration cases, 12 per cent. died of urogenital tuberculosis, 15 per cent. of tuberculosis of the lungs or other organs; but it is important to note that of these cases fully one-third at the time of operation had tuberculosis of either the lungs, bones, joints, or glands. After one-sided castration, 46 per cent. of the cases remained cured, the period of observation varying between three and thirty-four years. After bilateral castration 15 per cent. died of urogenital tuberculosis elsewhere, and 56 per cent. of the cases remained cured, the period of observation varying between three and thirty years. In not a single case was there any psychic disturbance as the result of this bilateral operation. The results of castration performed in the presence of tubercular involvement elsewhere, either in the urogenital tract or other portions of the body are not encouraging; in fact, as a general rule, operation is contraindicated in these cases. In conclusion, it may be stated that the results of castration for tuberculosis of the testicle are very much better than one dares to believe, and compare very favorably with the results obtained by the conservative method of treatment. In fact, there is no positive proof against the value of castration in these cases.

Operation for Epityphlitis in the Free Interval; Entero-anastomosis.—MARTIN (*Centralblatt für Chirurgie*, September 28, 1901) states that Jaffe recommended entero-anastomosis as the best operation in the free interval for those rare cases of epityphlitis in which it is impossible either to find the appendix or to remove it. Jaffe reports two such cases, in each of which he resected the cæcum, but both developed a fecal fistula, while in another case he performed entero-anastomosis after a long and fruitless search for the appendix, but the case died of peritonitis. As a result of this experience Jaffe has since stated that he believes entero-anastomosis to be an operation of no value in this type of cases. The author reports the case of a man who had had repeated attacks of epityphlitis during three years, the attacks recurring at shorter intervals, with finally the formation of a tumor in the ileocæcal region, severe pain, and other symptoms of intestinal obstruction. As a result the patient was incapacitated from his work, and operative interference was decided upon. On operation it was found that the ileum was bound down to the cæcum with old tight adhesions, and as a result it was impossible to find the appendix. So then the ileum was then anastomosed with the colon and the wound closed. The patient made an uninterrupted recovery except for attacks of slight pain in the ileocæcal region. Although it is impossible to draw results from one case, still this case shows that in those cases of relapsing epityphlitis, operated on in the free interval, where the appendix cannot be found except by a prolonged search, or even then not at all, it is much better to perform entero-anastomosis at once than to endanger the patient's life by a perhaps fruitless search for the appendix.

The Surgical Treatment of Thrombosis of Varicose Veins of the Leg.—KRAMER (*Centralblatt für Chirurgie*, No. 14, 1901) states that thrombosis is a very unpleasant complication of varicose veins which is generally followed by repeated attacks of inflammation, swelling, eczema or even abscesses, which can only be cured after weeks or months of careful treatment and rest in bed. The author recommends an incision, under local anæsthesia, over the thrombus, the opening of the vein in its longitudinal direction, and then carefully turning out the mass of clot. The author has performed this operation in fifty cases with most gratifying success; in no case were there sequelæ such as emboli, fistula nor bleeding from the open vein, which was impossible, because the peripheral and central lumen were closed or obliterated by clots. The wounds healed kindly by the patient being at rest, and left behind small painless scars. The veins became obliterated, and under careful asepsis this operation is a simple and safe method of treatment.

Experimental Uro-genital Tuberculosis.—BAUMGARTEN (*Archiv für klin. Chirurgie*, 1901, Band lxiii.) as the result of his experiments states that he was unable to produce tuberculosis of the testicle by injecting tuberculous material through the urethra. By this means it was possible to produce tuberculosis of the deep urethra, prostate, and bladder, but never of the vas deferens, testicles, ureters, and kidneys. On the other hand, in every case in which the testicle was inoculated the disease extended to the vas deferens and prostate, but in no case did the other testicle or vas deferens become involved. The author states that he finds the explanation for his successes

and failures in the generally known law that tubercle bacilli will never travel against the current of fluids, whether they be secretory or excretory; tuberculous infection may descend from the kidney or ascend from the testicle, but never vice versa. Clinically it has been noted that the disease may have the testicle as its primary focus.

Suture in Fracture of the Patella.—BARKER (*Archiv für klin. Chirurgie*, Band lxiii., Heft 4), after describing in detail the technique of his well-known method of subcutaneous suture, states that he has performed this operation twenty-one times. With the exception of one case, which died of delirium tremens on the seventh day after the operation, the results were perfect, the patients regaining the full use of their legs.

Discussion upon Spinal Anæsthesia.—HENDRIX (*Jour. de Chir. et Annales de la Soc. Belge de Chir.*, No. 7, 1901) states that Bier, the discoverer of this method of anæsthesia, has stated that he considers it more dangerous than chloroform, disapproves of the enthusiasm with which it has been received, and that it should never be employed without full knowledge of those modifications which have been found to be indispensable.

PEDIATRICS.

UNDER THE CHARGE OF

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Influenzal Nephritis.—D. J. MILTON MILLER (*Archives of Pediatrics*, January, 1902, p. 1) reports a marked case of acute hemorrhagic nephritis complicating influenza in an infant of thirteen months, and makes an elaborate analysis of forty cases of influenzal nephritis. From this study he draws the following conclusions:

Nephritis is a rare complication of influenza, occurring chiefly in young adults and children, and in infancy is almost unknown. Albuminuria is more common and is probably present in all severe cases. Hæmaturia alone is not infrequent, and, like nephritis, is seen oftener in early life. The sexes are affected equally. The nephritis may appear early in the disease, or at varying periods after the acute symptoms have subsided. It is usually an early complication, occurring in one-half the cases within eight, and in two-thirds of the cases within twenty-one days of the commencement of the influenza. The clinical type varies: it may be that of an ordinary acute nephritis, but in the majority of cases it is of the hemorrhagic type, this form being especially frequent in young adults and children. Edema is

absent in more than half of the cases, and is apt to be slight. The onset is usually attended with fever. The nephritis is of short duration, generally lasting under three weeks. The prognosis is good, recovery being the rule; although a small number of cases pass into the chronic subacute stage. When influenza attacks those whose kidneys are already diseased, it is apt to be serious and fatal. From the meagre pathological reports the lesions appear to be those of an infectious or toxic nephritis, often taking the form of a glomerulo-nephritis.

A Case of Acute Articular Rheumatism in an Infant Twenty-seven Days Old.—PAUL J. BARCUS (*Archives of Pediatrics*, January, 1902, p. 37) reports a case of articular rheumatism in a new-born infant, the second child of healthy parents. At birth the baby weighed seven pounds, was perfectly healthy and continued so, although artificially fed, until the twenty-seventh day of life, when the mother noticed that its left arm seemed paralyzed and was painful when moved. The physician was not called until the third day of the disease, when he found the groin temperature 99.5°, and the pulse 120. The left elbow was much swollen, red and very sensitive. The child was taking nourishment well and there was no gastrointestinal disturbance. On the following day the left wrist also was affected, and one day later the knee and ankle of the same side were involved, with increase in the temperature to 102°, and in the pulse-rate to 130. Subsequently the right knee and ankle became affected, followed in a few days by involvement of the right elbow, and, later, of the right wrist. The temperature never exceeded 102°, but after subsidence to normal on the nineteenth day the pulse remained somewhat high (150) for several weeks. Profuse acid perspiration was present early in the attack. About a month later the right knee again was swollen, red, and sensitive, but returned to normal in a week. The treatment consisted of salicylate and bicarbonate of sodium, a grain of each every three hours at first, and then less frequently. These doses were continued for some time after the first attack and were resumed during the relapse. No digestive disturbance occurred. Two months after the beginning of illness the infant was gaining slowly and was entirely free from the joint affection. The heart showed nothing abnormal on auscultation, but was beating at the rate of 125, with the apex slightly displaced.

The only suggestion of hereditary taint was an attack of what the doctor had called muscular rheumatism of the post-cervical muscles, which the mother experienced when five months pregnant. It lasted for one week, and was not accompanied by fever or joint involvement. No history of chorea or tonsillitis in either parent was elicited, and no pus focus or tendency to joint suppuration could be discovered in the infant.

The striking features of the case pointed out by the author were the great tolerance of the stomach for the medicine and the remarkable regularity of all symptoms that go to make up the classic picture of acute articular rheumatism.

[This case is peculiarly interesting, besides, as suggesting confirmatory support to the theory of the infectious origin of rheumatic fever, in favor of which might be adduced the polyarticular character of the attack and the

well-known susceptibility of the newborn to infectious processes, for which the umbilical wound offers a favorable port of entry. The absence of gastrointestinal symptoms seems to exclude the possibility of a lactic-acid intoxication through the food. The possibility of maternal influences of a similar character are also eliminated, since the child was nourished artificially, presumably for some time before becoming ill.—ED.]

Diagnosis of Suppurative Pericarditis in Children.—FREDERICK E. BATTEN (*Pediatrics*, November 1, 1901, p. 328) calls attention to the difficulties of diagnosis of this condition, which he has found to be present in nearly 3 per cent. of the deaths in the records of the Children's Hospital, Great Ormond Street, London. This paper is based on the observation of some six cases, five of which were boys and one a girl; their ages varied from ten months to three years. Their illness on admission to the hospital had lasted from four to nine weeks, and the total duration of the illness from the onset till death varied from seven to fifteen weeks. In every case a definite onset to the illness could be fixed, although it was not necessarily acute. In two cases measles had been the starting-point of the disease, while in the remaining cases it had been bronchitis or pneumonia. The importance of careful attention to the history is great, and the history obtained closely resembles that obtained in cases of empyema rather than the longer history of malaise which is usually obtained in cases of tuberculosis.

It is difficult to point out any very definite feature in the temperature which may be considered characteristic of the disease, but sudden falls are not uncommon and are attended with collapse; the general curve of the temperature is irregular, with but few subnormal points. The pulse is almost uniformly rapid and out of proportion to any distress or discomfort which the child may exhibit; in fact, a pulse of 150 or 160 may be found with apparently little distress. The respiration is usually increased, but maintains its normal ratio to the pulse.

The children are usually pale, often with fat and flabby cheeks; their bodies are often fairly nourished, but the muscles are flabby and wasted. There may be some cedema of the legs and feet, but cedema of the chest wall has never been observed by the author.

Severe attacks of syncope not infrequently occur. The impulse of the heart is generally to be felt either in the normal situation or in the epigastrium. The area of cardiac dulness is not necessarily increased. No murmur or alteration of the sounds of the heart could be detected, though they were noted as feeble in one case; but at no time was any pericardial friction audible. With regard to the lungs, it is important to note that in every case there was evidence either of apical or basal consolidation of the lung, or of effusion into the pleura, and numerous catarrhal sounds were to be heard, which, owing to the chronicity of the disease, were likely to be attributed to tuberculous infection.

In the post-mortem examinations of these cases both pleuræ were universally adherent. In nearly every case there was more adhesion between the sternum and the outer surface of the pericardium than normal; and while in three cases the area of the pericardium was noticeably increased,

and in two others in which the area occupied by the pericardium and the abscess together was considerably in excess of the normal pericardial area, it had not been detected on physical examination during life.

The amount of pus varied from a few drachms to six ounces; in some cases it was a thin opalescent fluid, and in others a thick pus embedded between masses of lymph. It is noteworthy that in four of these cases there was a localized purulent effusion into the pleura, while out of nine cases of fatal empyema in the hospital at the same time the pericardium appeared normal in all.

The use of the exploring-needle is deprecated, incision in an intercostal space or resection of a rib being considered a safer procedure, even for diagnostic purposes. While no pathognomonic sign can be suggested, the points emphasized in the paper should serve to arouse an intelligent suspicion of the condition, and operative treatment by incision and drainage offers the only possibility of recovery, though the percentage must, unfortunately, be small.

Serous Meningitis Due to the Typhoid Bacillus.—GUINON (*Société de Pédiatrie*, Séance, October 8, 1901; *Revue Mensuelle des Maladies de l'Enfance*, November, 1901, p. 529) saw in consultation a child suffering from typhoid fever, which had begun with disturbance of the stomach and torticollis. The course of the disease had been characterized by high fever, ranging for a time between 104° and 105.4° F. Early in the course of the disease the attending physician had observed a rigidity of the neck, but owing to the absence of Kernig's sign had dismissed the thought of meningitis. Several days later, when Guinon saw the patient, the symptoms of meningitis were quite distinct. Lumbar puncture gave issue to a serous fluid which was found to contain typhoid bacilli. The case terminated fatally.

A New Sign of Infantile Pneumonia.—WEILL, of Lyons (*Revue Mensuelle des Maladies de l'Enfance*, October, 1901, p. 482), calls attention to a new sign which he has observed in pneumonia, and one that is nearly always constant. This is a lack of expansion of the subclavicular region of the affected side. This sign, which has been watched for in all affections of the respiratory tract, has been encountered only in pneumonia. In pleurisy and pneumothorax lack of expansion may be noted on the affected side, but it embraces the whole side and is in direct connection with the seat of the evolution. It may be confined to the base, or, if the effusion is extensive, may involve the whole side. In pneumonia, on the contrary, the lack of expansion is confined to the subclavicular region, even and especially when the pneumonia affects the base. It is an early sign which is observable from the first day and lasts often through the whole course of the disease.

This precocity constitutes the chief value of the sign, for it often happens that the physical signs are wanting up to the fifth, sixth, or seventh day in certain forms of the disease. It is well known that at the beginning pneumonia may simulate various affections, one of the most important of which is appendicitis. An instance is given in which Prof. Weill was enabled by this sign to make a correct diagnosis of pneumonia in a case which had been

diagnosed as appendicitis. By the same means, pneumonia occurring in meningitis, typhoid fever, or influenza has been detected at once.

In practice this sign is easily elicited. It suffices to expose the chest while the patient is in the dorsal position waiting until the agitation, which accompanies the first moments of examination, passes away and the breathing becomes regular. The difference of expansion on the two sides may then be readily observed. This may be better appreciated by passing the hand alternately over the two apices, or a stilet may be used to emphasize the difference in expansion on the two sides.

Duodenal Ulcer in an Infant of Ten Months.—VANDERPOEL ADRIANCE (*New York Academy of Medicine*, Séance, January 10, 1901; *Pediatrics*, July 15, 1901, p. 63) reported the case because of the very early age of the patient, a male infant in the Nursery and Child's Hospital. The family history was negative except that the mother was a hard drinker and irresponsible. On admission to the hospital the history was that the child had been very restless and had developed some gastro-intestinal symptoms. The condition became steadily worse, and the child cried constantly and placed his hands on the abdomen. After the first two weeks of rapid emaciation, and another fortnight in which the condition remained stationary, a relapse occurred, and about two pounds were lost in the week preceding death. Shortly before the end there were a number of bloody stools and some blood was vomited. At autopsy an ulcer was found in the duodenum, measuring 1 by 2 cm., which had nearly eaten through all the coats. The liver was excessively fatty.

The youngest case heretofore recorded was in a child, aged four years, the ulcer developing after a burn.

A Case of Subcutaneous General Emphysema.—W. L. CARR and SAMUEL PIERSON (*New York Academy of Medicine*, section on Pediatrics, Meeting May 9, 1901; in *Pediatrics*, October 15, 1901, p. 314) report a rare instance of this complication in a boy, aged four and a half years, suffering from pneumonia. For five days there were noted only the clinical symptoms of pneumonia, without detectable physical signs; from this until the tenth day the physical signs became clearly marked in the right lung, and then the other lung became affected. Five days later slight emphysema of the neck was noticed, and by the next day it had become general on the arms and trunk. Both lungs cleared up about this time, but the emphysema extended all over the body, gradually disappearing within the next three weeks. The child made a good recovery. The case is interesting from the fact that this complication is extremely rare with pneumonia; and with the lobar type, of which this was an instance, a general emphysema is almost without exception the forerunner of a fatal result.

Gangrene after Scarlatina and other Infectious Diseases.—EICHHORST (*Deutsches Archiv für klinische Medecin*, Bd. lxx., Hefte 5 u. 6) adds a new case of this very rare complication of scarlatina. The patient was a girl, aged four years, who had an unusually severe attack of scarlet fever. At the end of the third week symptoms of embolism of the left leg appeared

suddenly, followed by dry gangrene, the line of demarcation finally forming about the middle of the leg. Examination of the bloodvessels after amputation showed evidence of endarteritis of the popliteal artery with a thrombus 1 cm. above its bifurcation, extending into both the anterior and posterior tibial arteries. Pure cultures of the streptococcus pyogenes were obtained from the pus of an otitis media and from a furuncle on the face.

According to the author's researches in the literature only two other cases of gangrene following scarlatina have been recorded. Both these occurred in children and involved both legs. In the course of this study 166 cases of gangrene following infectious diseases were collected, of which the greatest number followed typhus, 42; typhoid, 40; and influenza, 19. Of the rarer instances 10 were found following pneumonia, 5 after measles, 1 after varicella, and 1 after diphtheria.

Treatment of Adenoid Vegetations.—LAPEYRE (*Société de Pédiatrie*, Séance, October 8, 1901; *Revue Mensuelle des Maladies de l'Enfance*, November, 1901, p. 530) calls attention to the good effects he has secured in the treatment of adenoid vegetations by the internal administration of compound tincture of iodine. He commences with six drops three times a day, and gradually increases the dose, according to the age, until thirty to fifty drops are given. Despite these high doses he has never observed accidents. Atrophy and disappearance of the vegetations have been observed in a large number of cases.

The Use of Artificial Serum as a Means of Prognosis in Diphtheria.—In a series of observations covering the use of subcutaneous injections of artificial serum in infectious diseases, especially diphtheria, RABOT and BONNAMOUR (*Lyon Médical*, 1901, No. 34) have brought out the fact that the reaction of the patient to such injections may be taken as an almost infallible indication of the favorable or unfavorable outcome of the case. If, after the injection a prompt diuresis occurs, without vomiting or diarrhoea, the outcome of the disease will be benign, whatever the general state of the patient; if, on the contrary, after injection the quantity of urine is not increased, and vomiting or diarrhoea occur, the prognosis is fatal, even though the child may seem to be doing well at the time. In all the cases tested in this way by the authors the disease terminated in accordance with this law. Sometimes a first injection has indicated a bad prognosis, even when the child's general condition seemed excellent; but a second injection has given the same response, which did not fail soon to be confirmed. In illustration of the certainty of prognosis under these conditions, the authors detail a case of diphtheria in a child, who, even after the injection, passed scarcely 300 grammes of urine in twenty-four hours, and had frequent vomiting attacks. He was apparently in good condition, however, laughing and playing about the bed. Despite the employment of several diuretics, including theobromine and lactose, and the use of saline intestinal injections, the excretion of urine did not increase, and death occurred on the eleventh day.

Despite the certainty of prognosis thus early indicated, treatment should not be neglected. The serum test indicates the condition of the kidneys,

and every effort should be made to favor the elimination of toxins. Diuretics should be employed, lactose in particular and lavements of salt solution. The general state and fever, if present, should receive attention. Further injections of artificial serum should be avoided, since they can only aggravate the conditions by producing an excessive increase of blood tension.

By observation of the reaction produced by subcutaneous injections of artificial serum, one may gain definite knowledge of the state of the heart and of the degree of infection, and thus may be established an almost certain prognosis in embarrassing cases, so frequent in the diseases of children, in which the general state does not correspond with the local, and *vice versa*.

A Simple Procedure for Increasing the Amount of Fat in Diluted and Sterilized Cow's Milk—ROMANOFF (*Vratch*, September 1, 1901) suggests a method for accomplishing such a modification which does not seem to possess any advantage over our more accurate laboratory methods. Fresh milk is diluted with an equal part of oatmeal-water containing 4 per cent. of sugar. The mixture is poured into six bottles, sterilized in Soxhlet's apparatus for ten minutes, and then placed on ice for from two to three hours. The bottles are then carefully removed, without shaking, and the lower half of the milk in each bottle is siphoned off. The remaining milk is shaken, warmed, and given to the infant. This upper milk is said to show three or four times as much fat as the part removed by siphoning, being estimated at from 2.9 to 3.6 per cent.

[As this method makes no provision for varying modifications of the prepared percentage, which in this mixture must be constantly about 1.50 to 1.75 per cent., a percentage which many infants cannot digest in the earlier months of life, it would seem that the product offers little advantage over other mixtures of the same kind, excepting, perhaps, in previous sterilization (which may be undesirable), and in the fact that the emulsion of the fats is little disturbed (also a gain of little practical importance in most cases). The inflexibility of the sugar percentage is also another defect.—T. S. W.]

THERAPEUTICS.

UNDER THE CHARGE OF

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The Analgesic Antipyretics.—DR. G. POUCHET gives a valuable practical summary of the analgesic antipyretics, based on the relation of their physiological action to their chemical structure. He divides them into six groups, as follows: (1) *Quinoline group*, to which belong analgine, kairine,

thalline, cupreine, quinine, euquinine, and chinaphtol; (2) *Pyrol group*, in which are found antipyrine and its salts, analgesine, acetopyrine, tolypyrine, tolysal, pyramidon, anilipyrine, quinopyrine, ferropyrene; (3) *Hydrazine group*, phenylhydrazine, pyridine, antithermine; (4) *Aniline group*, aniline, diphenylamine, and other secondary amines, acetanilid, exalgine, euphorine, thermidine and neurodine; derivatives of para-amidophenol, phenacetine, triphenyl, lactophenine, citrophen, malakine, kyrofine, phenosal, cosaprine, phesine, phenocol, eupyrene and pyrantine; (5) *Aromatic acids*, benzoic, salicylic, benzacetine, aspirine, asaprol; and (6) *Alcohols and Phenols*. Of the first group the author believes that quinine is the only one that merits serious attention, the others having fallen by the wayside. In the second group antipyrine is the most important. The hydrazine group merits close attention, particularly because of the number of compounds which act prejudicially on the organism. Particularly all of the derivations of phenylhydrazine act upon the hæmoglobin of the blood, either to fix it and diminish oxidation or to cause hæmolysis. Antipyresis by such agents is not to be commended. The fourth group, the anilines, is perhaps the most important, containing the largest number of compounds. Many of this group have antiseptic actions as well; in fact, in some the antiseptic action overshadows any other for which it may be employed—thus the action of methylene blue on the malarial plasmodium is an example. Others of the group, acetanilid, phenacetine, are more employed for their analgesic action than for antipyresis. The aromatic acids, benzoic and salicylic, are closely allied, but benzoic acid being less readily broken up, oxidized, or eliminated is less of an antipyretic or analgesic, although an excellent urinary antiseptic. The salicylates, however, have marked analgesic powers, aspirine (salacetic acid), fulfilling most of the indications of this drug very acceptably. In the sixth group antiseptic action is the keynote, rather than analgesia or antipyresis. The author then discusses in detail the principles of the fascinating subject of the introduction of certain radicals into compounds and the resultant pharmacodynamic actions, a subject so ably discussed by recent advanced pharmacologists.—*Bulletin Général de Therapeutique*, 1901, vol. cxlii., p. 917.

Veratrum Viride in Pneumonia.—DR. T. G. STEPHENS believes in this drug for the treatment of pneumonia. In a series of fifty-four cases of pneumonia he reports fifty recoveries and four fatalities. The fatal results were necessarily so from the start. Norwood's tincture has given him the best results, and he employs the following formulæ: Tincture of veratrum viride (Norwood), 1; wine of ipecac or spirit of nitrous ether, 2. Ten drops every three hours, in a little water; increased one or two drops each dose. Tincture of veratrum viride (Norwood), 1; compound syrup of squill, 2. Same dose as above. Tincture of veratrum viride (Norwood), 2; tincture of aconite root, 1; syrup of tolu, 4. Fifteen drops every three hours, increased three drops each dose.—*Therapeutic Gazette*, 1901, vol. xxv., p. 731.

Pulmonary Hemorrhages and Gelatin Injections.—Drs. A. HAMMELBACHER and O. PISCHINGER, in two cases of hæmoptysis, report successful treatment by means of subcutaneous injections of 2½ per cent. gelatin solution.—*Münchener med. Wochenschrift*, 1901, vol. xlviii., p. 2000.

[In this connection it may be well to bear in mind that in the same journal Dr. F. Kühn calls attention to the dangers which may arise from the use of these gelatin solutions if they are not absolutely sterile. He there reported a fatal case of tetanus following the use of gelatin which had been used to relieve a severe hemorrhage following adenoid operations.—R. W. W.]

Some New Preparations of Valerian.—DRS. H. KIONKA and A. LIEBRECHT say that the uncertainty of the action of valerian is wholly due to the instability of the preparations. Inasmuch as the fresh leaves are not always obtainable, with which to make comparatively stable preparations, they prepared a synthetic combination with ethylamid, producing a diethylenamid valerianate ($\text{CH}_2, \text{CH}_2, \text{CH}_2, \text{CH}_2, \text{N}, \text{C}_2\text{H}_5$). In lower animals, frogs, etc., convulsions and paralysis result from its use; in cats and dogs, much mental excitement, not due to the odor, may be added; in man but few effects were noted. In hysteria, neuralgias, hæmicrania, neurosis of traumatic origin, and in the nervous symptoms of the menopause and pregnancy the drug acted well. Its field of usefulness might be extended to the milder types of melancholia.—*Deutsche med. Wochenschrift*, 1901, vol. xxvii., p. 849.

Treatment of Rheumatism.—DR. D. K. COVERLEY presents in *résumé* some of the phases of evolution in the manufacture and use of anti-rheumatic remedies. The objections to salicylic acid being many, the search for substitutes has been both active and prolific. Salol, salophen, salocoll, and aspirine have each been used and found effective in particular cases. The author makes a special plea for acetyl-salicylic acid, aspirine, which he has found agreeable and non-irritating. In alkaline media it liberates salicylic acid in a nascent state, and is an efficient analgesic, anti-rheumatic, and antipyretic. The clinical histories of six cases are reported.—*Therapeutic Gazette*, 1901, vol. xxv., p. 729.

Renal Tension and its Treatment.—DR. REGINALD HARRISON first drew attention to the fact that some cases in which the patient suffered from albuminuria and other renal symptoms that might have been produced by a stone or other removable causes had completely recovered after an exploratory operation on the kidney by puncture or incision, although no calculus or recognizable cause was discovered. This observation led by natural steps to a further generalization that tension might be an important factor in the production of some forms of renal disease. This deduction he finds to be corroborated by the testimony of many modern writers, and he draws certain analogies between the effects of intra-ocular tension in glaucoma and its effects on the retinal nerve cells and the effects of increased renal tension on the kidney parenchyma. However capable of gradual distention the kidney capsule may be, he believes that without doubt it is very intolerant of sudden increases of intrarenal tension, and experience gained by operation on the kidney has taught him that in certain conditions of congestion the capsule is so tightly stretched and its substance exposed to such pressure as quite to explain any interference with its function. Direct surgical intervention is therefore of value in such cases, and the problem arises as to

the type of cases to be chosen. These are in kidneys that are acted upon by occasional irritants derived from various infectious conditions, notably scarlet fever, diphtheria, measles, etc., and from alcohol and food intoxications. Not all of these require surgical intervention. The author considers three types of patients with acute nephritis which respond differently. In the first, a common type, there are varying degrees of rash, fever, and desquamation, with nephritis as indicated by blood in the urine, albumin, casts, and epithelium. The tendency is toward recovery, and surgical intervention is not to be countenanced for a moment. A second group, allied to the first, does not end in the same manner. In these convalescence is delayed and the signs of nephritis do not disappear. The disease does not progress toward recovery, but is stationary, or tends to become worse. Surgical intervention is applicable to these. The third type is allied to malignant scarlatinal nephritis. Here the acute, overwhelming process of intoxication with suppression of urine and uræmia, coma, convulsions, and death are correlative with great tension, as evidenced post-mortem. These cases are also to be considered as amenable to surgical treatment. The author advocates incision of the capsule for the relief of these kidneys. The patient is placed in the lumbar nephrotomy position. The kidney is exposed by an oblique incision, and its capsule opened, preferably along the convex border. Occasionally local signs of congestion may determine a different location for the incision. Simple punctures may be of service also. Drainage is essential in the after treatment, and permanent fistula is not to be dreaded.—*British Medical Journal*, 1901, No. 2129, p. 1125.

The Effects of Lead upon Lead-workers in the Staffordshire Potteries.—DR. FRANK SHUFFLEBOTHAM describes a systematic examination which he made of 528 people who are employed in lead processes among 6000 or 7000 workers in the Staffordshire potteries. As a result of his studies he comes to the following general conclusions: 1. That of the 528 persons working directly with lead processes he did not find one marked case of lead-poisoning; 2, that individual symptoms which at first sight might have been attributed to lead-poisoning were found, on closer examination, to be from other causes; 3, that the health record of the lead-workers was excellent (the complaints he mentions being for the most part only minor ailments). Of 348 men, 196 had not lost a single day's work through ill-health from any cause whatever since they commenced to work in lead, and the same may be said of 90 out of 124 single women; 26 out of 55 married women had only been absent through confinements; 4, that the general condition of the work-people was good and would compare favorably with a like number of workers in any average healthy trade; 5, that the 91 operators who had worked in lead for over twenty years were not suffering any ill effects from their employment, although they had worked for years under practically no regulations; 6, that it must always be remembered that lead-workers are subject to ailments of life just in the same ways as other people.—*Lancet*, 1901, No. 4078, p. 1109.

Hay Fever and Aristol.—DR. E. FINK, after a thorough discussion of the various theories concerning the etiology of hay fever, concludes that Helm-

holts's theory of a bacterial origin of the disease is nearest the correct one. The bacteria find their lodging-place in the various facial sinuses and are dislodged therefrom only with difficulty. The author blew aristol powder into the antrum of Highmore and obtained excellent results.—*Deutsche med. Wochenschrift*, 1901, vol. xxvii., p. 805.

Poisoning by Oleander.—DR. S. WATTEFF writes that this common ornamental plant so widely used for household decoration is occasionally used in the form of a leaf decoction as an abortifacient. Gastro-intestinal irritation, nausea, vomiting and slow pulse were symptoms observed in a case reported. Poisoning from the odor alone is here reported as having occurred.—*Deutsche med. Wochenschrift*, 1901, vol. xvii., p. 801.

[The active principle of oleander is well known as oleandrin (nereine), an active glycoside allied to digitalin in its action.—R. W. W.]

Quinine Rash.—DR. H. C. WOOD, JR., reports a case of quinine rash, and gives in addition a complete summary of modern literature bearing on this subject.—*Therapeutic Gazette*, 1901, vol. xvi., p. 8.

Epilepsy and its Treatment.—DR. W. H. BROADBENT bases his practice with regard to the treatment of epilepsy on the idea that the use of the bromides is to diminish the frequency and the severity of the fits, while the cure of the disease, the removal of the instability of the nervous system, is to be sought by other means. Occasionally the instability of the nervous system may be due to some visceral irritation, especially perhaps ovarian, and protection from this by the diminished sensitiveness to external impressions, which is produced by the bromides, may allow the nerve centres to gain their equilibrium or give time for the subsidence or the removal of the disturbing influence. He gives a number of rules which may be summarized. In long existing epilepsies, bromides are not given regularly, but only when some indication of over-excitement, such as restlessness or insomnia, are manifest. If the attacks occur punctually at definite long intervals, a short, sharp course of bromides preceding is advocated. If certain premonitions are recognized by the patient as regular precursors of the attacks, bromides may be taken to advantage. When the attacks are frequent, it may be necessary to give the bromides continuously in doses sufficient to afford relief. A fruitful subject for inquiry is to be found in the nocturnal attacks or in those that occur on rising. Here the author believes there is cardiovascular instability which reacts on the nervous instability. For these hot beef-tea or hot milk may be given at bedtime, or, in the case of morning attacks, such stimulants may be taken immediately on awakening. The author prefers food to drugs; but food failing, a dose of digitalis or other cardiac tonic may be given at night or in the morning. A careful regulation of the mode of life is considered as essential as drugs, if not more so. General discipline is paramount. Functional derangements should be overcome or corrected. He asks, after all is done, can anything be done to improve the stability of the nervous centres by such remedies as are known to be of service when the nervous tone has been impaired or when the functional activity of the brain has been disturbed by conditions of the circulation?

For such, phosphorus and the hypophosphites, or arsenic, with strychnine, and sometimes iron, are recommended. In many instances the cessation of the bromides has achieved excellent results.—*British Medical Journal*, 1902, No. 2140, p. 1.

Iodic Purpura.—DR. ALFRED STENGEL calls attention to a rare manifestation of chronic iodic intoxication. The familiar acne is very common, but purpura is a rare complication. He reports in detail the histories of patients who suffered from this complication. There was marked fetid breath, soreness and some sponginess of the gums, tremor of the extremities, with great moisture of the skin, rapidity of the heart's action, and swelling of the thyroid gland (symptoms which drew attention to the possible occurrence of Graves' disease); these symptoms accompanied the marked purpura. The eruption invaded mainly the leg below the knees. There were dark blue patches of discoloration. In other spots the color was rose-red and the eruptions were clustered in clumps.—*Therapeutic Gazette*, 1902, vol. xxvi., p. 1.

Treatment of Tabes.—DR. R. T. WILLIAMS has given a valuable critical review of the recent work bearing on this affection, from which the following notes on treatment may be excerpted: The value of anti-syphilitic treatment has been much discussed. In well-advanced cases it is usually of no service; but some instances are on record in which it has appeared to bring about some improvement. In all cases in which there has been syphilitic infection only a comparatively short time before the onset of the tabetic symptoms (five years, Collins) anti-syphilitic treatment should be employed at first. It should likewise be given when any evidences of syphilitic disease are present, or when the diagnosis is not quite clear between syphilitic pseudo-tabes and true tabes. Further, when the patient has not had any anti-syphilitic treatment for the tabes it is well to give it a trial (as Gowers recommends), in order that no complaint may be made later that any method of treatment has been omitted that might have been of service. Gowers and Taylor recommend *liquor arsenicalis*, and think that it brings about some improvement. Recently Motschutkowski has again strongly recommended the suspension method which he introduced in the treatment of tabes. He has employed it in 993 cases and has observed improvement in 207. The improvement was apparent in the gait, the muscular tone, the muscular sense, the paræsthesia, neuralgic pain, sexual power, bladder functions, and the general condition. The patient should be suspended at least one hundred times; the duration of each suspension being from one-half to five minutes, and should be carried out three to four times per week. One patient was suspended between four and five hundred times, and another over a thousand times. Chipault and Bardesco have recorded cases showing the value of stretching of the posterior tibial nerve in causing perforating ulcers to heal. Crocq employed faradism in one case and obtained good results. A small electrode was placed on the posterior tibial nerve behind the internal malleolus, and a larger one on the sole of the foot just behind the ulceration; a strong current being passed through the parts. After three weeks' treatment the ulcer became distinctly smaller, and in six weeks was

healed. The greatest advance in the palliative treatment of tabes is Fränkel's method of re-education of the movements of ataxic limbs. There can now be no doubt that the ataxia of tabes can often be greatly diminished by this system of careful training, in which the patient performs methodically co-ordinated muscular exercises, and by repeated practice learns to perform definite movements with care and precision. Even in severe cases of ataxia good results may be obtained. In carrying out the treatment, however, over-exertion should be avoided. As a rule, these exercises should not be performed while the patient is undergoing bath treatment; and Fränkel recommends massage of the muscles during the course of treatment. The exercises should be performed two or three times a day. For the arms special apparatus is necessary, but for the legs no special appliances are required. Muscular exercises of the legs are performed first while the patient is in the recumbent position; then exercises are carried out in the sitting posture and while standing, and, finally, walking exercises are practised. The movements are so arranged that care and precision are needed in carrying them out. Objects are touched by the toe or heel; and, in the walking exercises, lines are drawn on the floor and careful movements mapped out. The treatment is carried out at many special institutions on the Continent, and much time and patience are necessary.—*Medical Chronicle*, 1901, vol. xxxiv., p. 433.

Diphtheria Antitoxin Eruptions.—DR. ARTHUR STANLEY reports the observations made on a series of 500 cases of diphtheria during two years' work in a diphtheria ward at the Northwestern Hospital, London. All the cases were treated by antitoxin. The total number of deaths was 80, a death-rate of 16 per cent. The antitoxin was prepared at the laboratories of the Royal College of Physicians and Surgeons in London, and was injected in amounts usually of 4000 Behring, varying, however, according to the severity of the case from 1000 up to 30,000 units. There was no constant relation between the quantity of antitoxin given and the frequency of eruption. In this series of 500 cases, 112 patients had eruptions. Of these 58 were erythematous in character; in 15 there was combined erythema and urticaria; in 30 the eruption was an urticaria, 6 were scarlatiniform, and 3 morbiliform; in 17 patients there was a transient early erythema and an urticaria, usually at the seat of the injection. Thus the skin eruptions appeared in about one-fourth of the patients. The period of onset was usually during the second week after the giving of the antitoxin. The typical diphtheria antitoxin eruption is a marginate erythema on the psoriasis regions, tending to run in arcs of a circle, lasting about three days, and accompanied by slight malaise and a rise in temperature of about 3° F. The margins of the erythema are raised and turgid. The spread of the rash is most frequent from face and trunk to limbs, and from flexor to extensor surfaces. The eruption lasts from two to five days, but may, in cases of an urticarial or scarlatiniform nature last a few hours only. Sometimes the rash is general. Desquamation may occur. The occurrence of the rash did not appear to influence the course of the disease either favorably or unfavorably.—*British Medical Journal*, 1902, No. 2146, p. 386.

GYNECOLOGY.

UNDER THE CHARGE OF

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ASSISTED BY

WILLIAM E. STUDDIFORD, M.D.

Statistics of Uterine Cancer.—WALDSTEIN (*Centralblatt für Gynäkologie*, No. 50, 1901) from a careful analysis of the record of Schanta's clinic finds that a little over 14 per cent. of the cases of cancer of the uterus were operable. Of these, 8.8 per cent. succumbed to the operation. The writer concludes that less than 4 per cent. of all patients with uterine cancer are really cured by radical operation.

Dysmenorrhœa.—MENGE (*Centralblatt für Gynäkologie*, No. 50, 1901) distinguishes two forms of dysmenorrhœa, the so-called idiopathic, which is independent of pelvic disease, and the "secondary" which is due to disease of the genital tract. The writer believes that painful menstruation, from whatever cause, is referable to the tendency to uterine contraction which is present at the time of the monthly period. These are due not only to the premenstrual swelling of the endometrium described by Fritsch, but also to the mere presence of blood within the uterine cavity, which acts as a foreign body. These contractions in healthy women are insensible. In hysterical or neurasthenic subjects, on the contrary, the uterus, though entirely normal, is hyperæsthetic and dysmenorrhœa results. Mechanical obstruction to the escape of blood, added to this hyperæsthesia, increases this pain. In diseased conditions of the pelvic organs the dysmenorrhœa is more pronounced, especially if the two former factors are also present. Hence the deduction: Normal menstruation, as well as dysmenorrhœa, is accompanied by pains simulating labor-pains, although the latter may be entirely of nervous origin. It follows that local treatment alone is not sufficient to insure permanent relief, but the general nervous condition of each patient must be carefully considered.

The writer rejects the theory of a reflex nasal origin of dysmenorrhœa. Though he has seen some brilliant results from cocainization of the nasal mucous membrane, he is inclined to regard this as principally an application of the method of suggestion.

Fibromyoma in Cul-de-sac of Douglas.—ROSENSTEIN (*Centralblatt für Gynäkologie*, No. 50, 1901) reports a case of tumor in Douglas' pouch, which was diagnosed as an ovarian cyst with twisted pedicle. On opening the abdomen a fibromyoma was found behind the uterus with a pedicle, which was attached to the right side of the cul-de-sac. There were numerous intestinal adhesions. The writer inferred that the growth developed

from the smooth muscular fibres of the sacro-uterine ligament, and could find no similar case on record.

[Several years ago we reported a case of fibromyoma, which sprung from the under surface of the sacro-uterine ligament and grew downward into the posterior vaginal fornix, so that it was easily removed per vaginam. It seems possible that the tumor in the case above mentioned may have been an aberrant, or migratory, fibromyoma of the uterus.—ED.]

Parametritis Posterior.—BRÖSE (*Zeitschrift für Geb. und Gynäkologie*, Band xli., Heft 1) reports ten cases in which, after a long course of unsuccessful treatment, ventrofixation was performed. In five cases a pure parametritis was found, the peritoneum not being affected; in two the disease of the adnexa was mistaken for parametritis. Seven patients were cured; in one case there was a recurrence, and two were failures.

[The writer states that the uterus was anteфлекed, which leads one to ask whether tenotomy of the shortened sacro-uterine ligaments previous to ventrofixation of the uterus would not meet the indication more perfectly. This we have done in several instances with satisfactory results.—ED.]

Influence of Pregnancy and the Climacteric on the Ultimate Results of Operations for Cancer.—HEYSE (*Centralblatt für Gynäkologie*, No. 51, 1901) collected 122 cases in which the cancerous uterus was removed during pregnancy or the puerperium. Eighty-two of these were selected, in which at least five years had elapsed since the operation, but only 41 of these were suitable for comparison. Of these a radical cure was obtained in 24 per cent. Of 73 cases of hysterectomy for cancer in patients who had passed the climacteric, 50 per cent. were cured. These statistics would seem to show that pregnancy has an unfavorable influence on the prognosis, while the reverse is true of the climacteric.

Indications for Operation in Chronic Salpingitis.—LEGUEN (*Revue prat. d'Obstétrique et de Gynécologie*, No. 9, 1901) recognizes three indications for operation, viz.: 1. The general condition of the patient, especially in the presence of fever and constitutional symptoms; 2, the local condition, i. e., well-marked enlargements of the adnexa, or fistulous communications with the bladder or rectum; 3, severe and persistent pain, which is not relieved by treatment continued for several months.

Enlargement of the Inguinal Glands in Cancer of the Viscera.—VINAY (*Lyon Méd.*, No. 38, 1901) lays considerable stress upon this condition as a diagnostic sign of cancer of the pelvic and abdominal viscera. It is most frequently observed in the former class of cases. He distinguishes three forms of glandular enlargement, viz., simple inflammatory or pre-cancerous, metastatic, and inflammatory resulting from infection of ulcerating cancerous growths. This infection may follow the course of the direct lymphatics, as in cancer of the uterus, or may be carried through the superficial lymph-vessels from the umbilicus, when disease of the abdominal viscera has extended to the parietes. The glands are at first hard, movable, and painless, then they become adherent and painful in consequence of repeated

attacks of inflammation. The enlargements are usually unilateral, or are unevenly developed on the two sides. The differential diagnosis from bubo, gumma, and incarcerated hernia is sometimes difficult. This enlargement of the glands is of great importance, as it is sometimes the first evidence of malignant disease. The prognosis is unfavorable, since it denotes the extension of the primary trouble.

The Lymphatic Glands in Cancer.—COVDRAV (*Revue prat. d'Obstétrique et de Gynécologie*, No. 9, 1901) concludes an extended paper on this subject by expressing doubt as to the advisability of extirpating glands which are apparently healthy, the cancerous growth being in an incipient stage. Glands which are enlarged and non-adherent should always be removed; if adherent, their ablation is usually fraught with danger to the patient. Theoretically, exposure of the open lymphatics favors fresh infection; but practically there is as yet no method of operation which avoids this danger.

The writer suggests what he calls the "sclerogenous" method, which aims at closure of the lymphatics in the neighborhood of the tumor by making numerous injections of chloride of zinc around the growth. He reports a case of scirrhus of the breast in a woman, aged fifty-eight years, five years after treatment by this method. Nothing remained of the original growth but a small, hard nodule. Five years later the cancer had recurred, with enlargement of the axillary gland. He infers that in a certain class of cases the growth of circumscribed cancer can be arrested for a long period by the artificial production of fibrous tissue around the growth, which causes obliteration of the lymphatics. This treatment is especially applicable to patients who refuse a radical operation.

Spasms of the Clitoris in Tabes.—KÖSTER (*Münchener med. Wochenschrift*, No. 5, 1901) describes a peculiar localization of the crises in females affected with locomotor ataxia, first mentioned by Charcot. They appear early in the disease and are associated with the usual lancinating pain in the limbs. The spasms of the clitoris occur at frequent intervals and may be accompanied by ejaculations of mucus, or by spasms of the *constrictor cunni*, without pleasurable sensations.

Treatment of Bartholinitis with Salicylic Acid.—REYGASSE (*La Revue Médicale*, Heft 17, 1900) reports a series of cases in which he injected into the inflamed gland seven or eight drops of a saturated alcoholic solution of salicylic acid. The patient experienced a sharp pain, which soon subsided and was not followed by any unpleasant results. Within five or six days the swelling had entirely subsided. A second injection is rarely necessary.

Gonorrhœa and Marriage.—ZEISSL (*Wiener med. Presse; der Frauenarzt*, September 20, 1901) replies to the question "When may a man with gonorrhœa marry?" that this is allowable only when repeated clinical and bacteriological examinations give an absolutely negative result. The absence of gonococci in the secretions is not sufficient, but the patient must still be kept under observation until he is beyond suspicion. The presence of opalescent threads in the urine show that the discharge has probably ceased to

be infectious. This opinion is strengthened if only a few round epithelial cells are seen under the microscope. If diplococci are found, even though these do not grow in cultures, the innocent character of the secretion cannot yet be inferred. If no additional information can be obtained by the use of the endoscope, the writer recommends that the patient should use an irritating injection of nitrate of silver and drink beer frequently. If a free discharge from the urethra appears, and at once ceases spontaneously and no gonococci reappear, the patient may be regarded as cured.

Complications and Degenerations of Fibroid Tumors of the Uterus.—NOBLE (*American Journal of Obstetrics*, vol. xlv., No. 3) makes an interesting analysis of 218 cases operated upon for fibromyoma uteri. Complications and degenerations were encountered in 126 cases. In 71 of these cases the conditions were such as, without operative interference, would have led to the death of the patient. Of the fatal complications and degenerations 32 were of the tumor, and 39 of the appendages. In 25 cases the complications were such as to threaten the life of the patient, and in 30 cases led to more or less permanent invalidism. It is estimated that without operation 78 of the cases would have died of the complications or degenerations of the fibroid tumors. Added to this 15 patients would probably have succumbed to conditions produced by the tumors themselves, viz.: hemorrhages, chronic anemia leading to degeneration of heart and kidneys, pressure of the ureter and bowels, etc. The author concludes that had this series of cases remained without operation there would have been an estimated mortality of 42 per cent. Comparing this with the mortality from operation of from 2 to 10 per cent., early removal of the tumors seems undoubtedly to be the proper treatment. Such removal not only lessens the mortality, but eliminates the long period of invalidism that is otherwise unavoidable.

Migrated Ovarian and Parovarian Tumors.—EDEBOHLS (*Medical Record*, vol. lviii., No. 7) reports four cases of ovarian and parovarian tumors that had become detached from their original connection with the broad ligament. In Case I. the tumor presented all the characteristics of a parovarian cyst, but no trace of its former attachment could be found, both tubes and ovaries being normal. It was attached by a pedicle 10 c.m. wide to the lower free edge of the omentum, and at the time of operation was gangrenous. Case II. was operated upon four days after the onset of symptoms of strangulation of a large parovarian cyst. The tumor was found free in the abdominal cavity. The left tube and ovarian ligament had been completely torn from the uterine cornu and were found on the stump of the cyst-pedicle. The author concludes that had the patient survived without operation the tumor would have taken on new attachments and formed a true migrated tumor. That this conclusion is justified seems to be proved by Case III. The patient had an intraligamentous cyst on the right side; the left ovary was absent, and the left tube, with the exception of a twisted stump, 2 cm. long at the extreme end, was missing. Attached to the posterior aspect of the uterus and a portion of the left broad ligament was a firm membrane of irregular thickness. This was removed, and on subsequent examination proved to be part of the sac of a former ovarian cyst. Case IV. had a large monocyst of the left ovary. The

right ovary and all but 2 cm. of the right tube were absent. The lower border of the omentum was enormously thickened and was wrapped completely around a tumor 15 cm. in diameter. On enucleation this was found to be a dermoid of the right ovary containing teeth, hair, and sebum.

New Formation of the Female Urethra.—NOBLE (*American Journal of Obstetrics*, vol. xliii.) reports a case in which eleven unsuccessful operations had been performed for cure of a urethral fistula. The entire wall of the urethra was destroyed, and a fistula existed involving the neck of the bladder. The situation of the urethra was marked by a strip of mucous membrane continuous above with the vesical wall. The opening into the bladder was large enough to admit the index finger. The edges of the fistula were cicatricial, and upon each side of the urethra were extensive cicatrices, probably due to incisions made at the various operations to relieve tension on the sutures. After two unsuccessful attempts to form a new urethra, owing to the extreme tension on the sutures, the following procedure was successfully carried out: In order to increase the retentive power of the bladder the urethra was elongated so that the meatus should be at the clitoris instead of at the normal site. The internal coat of the urethra was made from the mucous membrane of the vestibule and the remains of the urethral mucous membrane. This was sufficiently loosened, after parallel longitudinal incisions had been made, so that it could be united over a Sims sigmoid catheter (one-third the usual calibre). This catheter was left *in situ* for ten days. The raw surface at the sides of the urethra and over the neck of the bladder were widened. An incision was made along the inner base of the left labium minus, and the labium detached from the subjacent structure and unfolded. This flap was drawn back into the vagina, covering over the urethra and then united by interrupted sutures to the edges of the raw surface. At the anterior end of the new urethra the lateral edges of the labia minora were united, firm union was obtained, and the bladder function was restored.

Etiology of Movable Kidney.—WATSON (*Boston Medical and Surgical Journal*, vol. cxlv., No. 12) conducted a series of experiments on the cadaver to determine the structures chiefly concerned in the fixation of the kidney. Twelve cadavers in which death occurred not longer than twenty-four hours previously, and in which the kidneys and their surroundings presented no abnormal changes, were used in the investigation.

After opening the abdomen downward traction was made by the finger tips upon the upper pole of the kidney with as little disturbance as possible to the neighboring tissues. The descent of the organ was from half an inch to an inch and a half. The outer leaf of the mesocolon was then divided and the colon and other viscera overlying the kidney were drawn inward. The descent of the kidney under traction was now increased by half an inch. Next, the fatty tissue in front of and beneath the lower end of the kidney was removed, but with no appreciable effect upon its mobility. The removal of that portion of the peritoneum which on the right side forms an actual peritoneal investment of the upper portion of the kidney, and is reflected from it onto the duodenum and stomach, permitted a further descent of a

quarter to half an inch. Thus, after removal of all the structures in relation with the anterior surface and lower end of the kidney, the descent of the organ had not exceeded the lowest point of its normal excursion by more than three-quarters of an inch in any case.

The structures connected with the posterior surface and upper pole of the kidney were now broken down, beginning above the upper pole of the organ. Further descent occurred in direct proportion to the destruction of these tissues, the movement finally becoming entirely free except for the restraint exercised by the main bloodvessels, whose action drew it toward the spinal column. These experiments seem to harmonize with the anatomical studies of Gëröta and others, and the author comes to the following conclusion: The structures vital to the restriction of the kidney's mobility within its normal excursion are those which form the attachments between the posterior and upper aspect of the tunica propria, and the fascia covering the lumbar muscles and the peritoneum covering the diaphragm respectively, aided by the less essential ones connecting the anterior surface with the peritoneum overlying it.

OBSTETRICS.

UNDER THE CHARGE OF

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Three Successful Cæsarean Sections.—KERR (*Medical Press*, November 6, 1901) reports three successful Cæsarean sections in the Glasgow Maternity Hospital. He prepares the patient by thorough aseptic precautions, scrubbing the cervix and vagina with lysol, 1 per cent. In each of the cases labor was in progress and each patient was sterilized by tying the Fallopian tubes in two places and dividing them between the ligatures. In one case sterilized silk was used to close the womb, and in the other two catgut. Elastic tubing was not used to control the bleeding, but a vulcanite ring was employed to prevent hemorrhage when the uterus was opened.

His first case was that of a multipara in her third pregnancy, her other labors having been terminated by craniotomy. The os was fully dilated and the membranes ruptured, but the pelvis was contracted and the head freely movable above the brim. This patient's convalescence was interrupted by a severe bronchitis, occasioned by the irritation of chloroform, and also of coal-gas with which the building was lighted. The patient and her child made a good recovery.

The second case was a primipara with contracted pelvis. The child was dead, and no trace of heart action could be discovered. The patient made a good recovery, but had very scanty lochial discharge and a somewhat

rapid pulse. When this patient was admitted to the hospital the heart sounds were strong and distinct, but through an error the operation was delayed three hours after admission. The child perished during this time.

In the third case the uterus grasped the child so firmly at operation that the lower end of the uterine incision was slightly torn in delivering the fœtus. This patient had one rise of temperature during her convalescence, which soon subsided.

Kerr believes that craniotomy should be positively declined when the child is living and in good condition. He believes that sterilization should not be practised, because the mortality is now not sufficiently great to prevent a repetition of the operation if necessary. He believes that it is well to select the time of operation to have the circumstances favorable. It is important not to wait until the lower uterine segment has developed, because this is readily torn and easily infected. The placenta in each case was on the posterior wall of the uterus and the Fallopian tubes were close together upon the anterior wall. In the Fallopian tube tissue excised decidual cells were not found.

Puerperal Tetanus.—In the *Indian Medical Gazette*, vol. xxxvi., No. 5, 1901, KEDARNATH DAS reports the case of a Hindoo woman delivered at full term of a living child. On the tenth day the infant was seized with trismus neonatorum, and died within twenty-four hours. The mother developed tetanus, and was admitted to a hospital. She was treated by washing out the uterus with an antiseptic solution, with the free use of chloral by rectal injections and by hypodermoclysis. The spasms continued six weeks before the patient began to grow essentially better. An abundance of liquid food was given and the bowels were kept open regularly. During her illness the patient received 8 ounces, 6 drachms, and 10 grains of chloral hydrate. Das has collected 68 cases of puerperal tetanus, showing a remarkably small percentage of recovery. He could find but five recoveries, giving a mortality of 92 per cent. There were 21 cases treated by antitoxin, given either by subcutaneous or intracerebral injections. Of these but one recovered, a mortality of 95 per cent. Among these cases of tetanus but 4 occurred in India, where the disease does not seem to be common. One of these recovered, giving a mortality of 75 per cent.

Prolonged Retention of the Fœtus During Labor by the Enormously Dilated Cervix.—RAPIN (*La Semaine Médicale*, November 13, 1901) reports the case of a primipara, aged twenty-five years, who was suffering from a tedious labor. The head came down into the pelvic cavity and upon the pelvic floor, when the attention of the midwife was attracted by a tumor situated between the symphysis and the umbilicus. The descent of the head ceased and assistance was summoned. On examination the pelvis was found somewhat contracted in its antero-posterior diameter. The head was in the pelvic cavity, the smaller fontanelle obliquely and toward the left. The fetal heart sounds were not heard, and upon the presenting portion of the head there was a tumor which bled somewhat. A tumor of elastic consistence was found between the pubes and the umbilicus. Thinking that this was a distended bladder, the patient was catheterized, but the tumor did not dis-

appear. The forceps were then applied, but the head could not be delivered with moderate traction. Digital examination could discover no apparent reason for the retention of the fetus. The patient was accordingly transferred to the hospital. Upon examination there it was discovered that the two arms of the fetus seemed engaged in the pelvis beside the head, and that if these could not be dislodged the child could not be delivered. Accordingly the fetus was cautiously delivered by version and found to be dead and somewhat macerated. It was perfectly formed and in no way a monstrosity. Immediately after delivery there was free hemorrhage, which ceased after the expulsion of the placenta, a hot intra-uterine douche of lysol and compression of the uterus. The abdominal tumor disappeared. Upon further examination it was found that the abdominal tumor had been composed of the upper uterine segment containing the placenta. The child had been contained in the enormously distended lower uterine segment and cervix.

He also reports the case of a multipara who had had six normal confinements previously. She had a flat rachitic pelvis. The head was presenting and fixed in the pelvis, the heart sounds feeble and irregular. Meconium was escaping from the vulva, and there seemed to be fetid gas in the uterus. It was evident that the cervix was enormously distended. The forceps were applied at the superior strait, and a male child asphyxiated was delivered. The child was resuscitated. The placenta was removed and the uterus thoroughly douched with a hot antiseptic solution, when upon examination it was found that the upper uterine segment had been invaginated into the lower. It was enormously distended.

Both of these patients recovered, which must be considered an excellent result in view of the very serious distention which was present.

Labor Brought on by Alcoholic Neuritis.—LE PAGE reports in the *Comptes Rendus de la Société d'Obstétrique de Paris*, 1901, vol. iii., the case of a patient in her second pregnancy whose first pregnancy had terminated normally. In the present gestation the patient suffered severely from nausea and vomiting, and had great difficulty in walking, with much pain in the lower limbs. A physician whom she consulted ordered baths and a milk diet. She finally became much worse, with frequent pulse, elevated temperature, dry tongue, delirium, and marked and excited movements. The lower extremities were paralyzed, the feet extended upon the legs, and the hands were immobilized and flexed upon the forearms. There was no inequality of the pupils and no strabismus. The abdomen was slightly distended, and there were sordes on the borders of the gums and teeth. There was nothing abnormal about the heart and lungs and no albumin in the urine. The appearance of the patient was that of a severe typhoid infection.

The serum diagnosis was negative, and labor was induced by the introduction of an elastic bag. During the treatment of the case it was learned that the patient had used large quantities of alcohol, drinking rum very freely. The delivery was spontaneous. The patient afterward made a very tedious recovery, with atrophy of the muscles, which yielded slowly to electrical treatment. The case was a typical one of polyneuritis, undoubtedly of alcoholic origin.

Autopsy in a Case of Pernicious Nausea of Pregnancy.—DE RIBES (*Comptes Rendus de la Société d'Obstétrique de Paris*, vol. iii., 1901) had the opportunity to make an autopsy upon a woman dying from pernicious nausea of pregnancy. The patient was aged thirty-one years, pregnant for the fourth time at about six months and much emaciated from constant vomiting. Examination was negative; her pulse was 100. She was admitted to the hospital and various means were tried to control the nausea. This failed, and she was finally delivered spontaneously of the foetus. After the delivery the patient steadily grew worse, presenting a typhoid appearance, with sordes upon the teeth and a red tongue. She did not vomit after the uterus was emptied, but her urine was highly albuminous. Intermittent delirium supervened, followed by coma, and death in convulsions.

Upon autopsy an active secondary nephritis was found in both kidneys. The brain, the heart, and lungs presented no lesion, but the liver was greatly altered. It presented a blanched appearance, with small areas of yellow. A large infarct was found upon the lower portion, which had replaced necrotic parenchymatous tissue. The lesions were those of an active toxæmia of hepatic origin, with multiple hemorrhages. The lesions greatly resembled those often seen in eclampsia.

Retroplacental Hemorrhage following Traumatism in the Abdominal Region.—DE RIBES (*Comptes Rendus de la Société d'Obstétrique de Paris*, 1901, vol. iii., p. 171) reports the case of a patient in her second pregnancy at the seventh month, who struck the abdomen violently against the corner of a table. The patient fell unconscious, but speedily revived. She had severe pain at the region of the blow and could not perceive foetal movements, and lost neither blood nor amniotic liquid. She was admitted to the hospital, and on examination the pulse was frequent and weak, the axillary temperature normal. The urine was not albuminous, and over the region of the blow there was an ecchymosis and brownish discoloration. The region was painful on palpation. Auscultation was negative, palpation also. On examination the cervix was permeable to the internal os, which was closed. The lower uterine segment was higher and distended, and the lower extremity of the foetal ovoid could be indistinctly felt. There was no hemorrhage from the genital organs. The patient's pains continued regularly, and labor finally came on. When the membranes ruptured a large mass of blood-clot was expelled and immediately afterward the foetal head. The child had recently died, and an autopsy upon its body was negative. The placenta was expelled, but the membranes were adherent. On exploring the uterus the membranes and some old clots were found and removed. The uterus was thoroughly douched with an antiseptic solution. On examining the placenta it was found that blood had accumulated between the placenta and the wall of the uterus throughout a considerable portion of the attachment of the placenta.

A similar case is reported by LE PAGE (*Comptes Rendus de la Société d'Obstétrique de Paris*, 1901, vol. iii., p. 113). This patient was a primipara, who struck the abdomen violently against the corner of a piece of furniture, causing violent pain, which disappeared after a short time. On the following day the patient resumed her usual occupation, and while her appetite

was not good, and she felt uncomfortable, she had no hemorrhage and did not seem ill. On the following morning she was taken with violent pains, and lost a large quantity of blood. On admission to the hospital no heart sounds could be heard, the patient's pulse was 80, and her temperature sub-normal. She had ceased to bleed. The expulsion of the fœtus continued, and a dead but not macerated child was spontaneously expelled. The placenta and a mass of blood-clots immediately followed. The patient continued to bleed for some time after labor, but finally made a good recovery.

Retroversion of a Fibroid Uterus Simulating Pregnancy.—RUDAUX reports in the *Comptes Rendus de la Société de Paris*, October, 1901, the case of a woman, aged thirty-four years, married six months, whose menstruation had been irregular since its first establishment. A few days before entering the hospital she had pain in the lumbar region, with an abundant discharge of blood, for which a midwife gave her several hypodermic injections of ergotin. On the following day, during a bowel movement, the patient expelled a considerable quantity of clot. A physician who saw her diagnosed an abortion at two months. After this the patient's condition became alarming; she had difficult micturition and severe and persistent abdominal pain, for which she was brought to a hospital. On examination the uterus was found retroverted, the posterior cul-de-sac filled by a mass the size of a large orange. A diagnosis was made of retroversion of the uterus, gravid between two and three months. The patient had a foul discharge and fever, and curetting of the uterus, with its replacement, was decided upon. When this was done it was found that the patient was not pregnant, but that upon the posterior wall of the uterus there was a sessile soft fibroid which had become infected. As the patient's condition continued to grow worse, hysterectomy was practised, followed by a fatal issue.

This case calls attention to a familiar error, the diagnosing of abortion because a patient whose menstruation has been irregular is taken with pain and hemorrhage. A positive diagnosis of abortion cannot be made, unless some portion of the embryo or its appendages is clearly identified.

Torsion of the Pedicle in Hydrosalpinx Complicating Pregnancy.—In the *Comptes Rendus de la Société de Paris*, October, 1901, PINARD and PAQUY report the case of a woman, aged twenty-six years, in her second pregnancy, who suffered from severe pain in the right side, which recurred at irregular intervals. The patient vomited bile and had a chill. The examination and history pointed to a cystic tumor with twisted pedicle. Upon abdominal section the tumor was found to consist of an enlarged ovary, with hydrosalpinx, whose pedicle had become twisted. The patient made a good recovery from the operation.

Atony of the Uterus Causing External Hemorrhage during Gestation.—OLIVER (*Edinburgh Medical Journal*, December, 1901) draws attention to the frequency of hemorrhage from the uterus at about the sixth week of gestation. The vessels over a localized area and the mucous lining of the uterus increase in size, forming sinuses. The wall of the womb itself grows thin and distensible. But when the maternal vessels receive the pressure of

the opposing villi, blood-pressure is maintained and hemorrhage from the sinuses prevented. If from any cause the coaptation of the villi and the uterine wall becomes impaired, then the maternal vessels may rupture and hemorrhage result. Atony of the uterus is a not infrequent condition in many ill-nourished and debilitated patients in whom hemorrhage during early pregnancy is not infrequently observed.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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The Pupil in General Disease.—JACK, Boston (*Boston Medical and Surgical Journal*, September 26, 1901), gives a summary of the pupillary phenomena in general disease.

The afferent path of the reflex arc for light reaction is the optic nerve and tract branching by some path to the pupil-contracting centre in the floor of the third ventricle, whence the third nerve to the iris is the efferent path. The associated contraction to convergence and accommodation is brought about by the same centre near which are situated those for convergence and accommodation. The active dilating centre is the cilio-spinal in the lower cervical cord. The normal moderate dilatation is dependent upon the constant psychical and sensitive stimuli transmitted through the sympathetic to the iris—the sympathetic reflex. The eyes should be tested one at a time for the light reflex, the other being covered. The patient should look at a distance so as to avoid bringing in the associated contraction for convergence and accommodation. So long as perception of light remains the reflex will take place, but the reverse is not true—i. e., the vision may be good and no contraction of the pupil occur, as in paralysis of the sphincter, for example, by a mydriatic. The pupil of the second eye will also react to light thrown upon its fellow because of the nervous connection between the two sides. A blind eye will not react to light thrown on its own retina, nor will the second eye, but both will react perfectly if light be thrown on the other sound eye. There is a rare exception to the rule that pupils of blind eyes will not react—that is, when a lesion is high up in the optic tract, as in uræmia. Local diseases of the eye and drugs internally or locally may cause anomalies in the reactions and shape of the pupil, and these must be excluded in studying the effect of general disease. Dilatation may be paralytic (third nerve) or irritative (sympathetic); so likewise contraction may be paralytic (sympathetic) or irritative (third nerve). Paralytic mydriasis

is seen in progressive paralysis (myosis at first) and in various disease processes at the base of the brain affecting the third nerve or its nucleus. Irritative mydriasis occurs in spinal meningitis of the cervical portion of the cord, in the spinal irritation of anæmic or chlorotic patients, after severe illness, as a premonitory sign of tabes, from intestinal worms, psychical excitement—acute mania, melancholia—in general paralysis, oftener then in one eye only with myosis in the other. Unilateral mydriasis occurring at short intervals, first in one eye and then in the other, is, according to von Graefe, a premonitory sign of mental derangement. Unequal pupils are said to be always pathological, but there are rare exceptions. They are said to occur in hysteria. Irritation myosis occurs in the early stages of inflammatory affections of the brain and meninges; later there is dilatation in the stage of depression. Paralytic myosis occurs in spinal lesions above the dorsal vertebrae—tabes, injuries, inflammations, etc. In acute mania, if myosis comes on, general paralysis may be prognosticated.

In the Argyll-Robertson pupil—reaction to accommodation, but not to light—the reflex arc is broken somewhere probably between the point where the optic fibres leave the tract and the nucleus of the third nerve. This is a frequent and early symptom in tabes; the pupils are usually, but not always, very small. The reaction to convergence is lost later. The same phenomenon is present in general paralysis, but less frequently, and usually later. In this disease pupillary symptoms are frequent and early, beginning usually as an inequality, the same being present in other forms of insanity. The Argyll-Robertson pupil always means the existence or approach of serious nervous disease.

Wernicke's symptom—light reflex preserved when light is thrown on the blind half of an hemianopic eye—is present if the lesion be above the reflex arc in the brain. Exceptionally the pupil may react when the lesion is in the arc, the special fibres for the reflex being perhaps more resistant than the ordinary fibres.

The paradoxical pupil symptom is the slight oscillation—hippus—which rapidly goes on to dilatation instead of contraction—considered an early sign of coming paralysis, also reported in hysteria and disseminated sclerosis. In the algid stage of cholera, if the light reflex is present the prognosis is good; if absent, bad. In epilepsy there is usually contraction first and dilatation later, the light reflex being lost—an important point in distinguishing the attack from hysteria. The writer, however, asserts that he has observed dilated and irresponsive pupils in hysteria. Both bilateral and unilateral mydriasis has been noticed in diabetes.

For diagnosis of the situation and nature of intracranial lesions the pupillary symptoms, if alone present, are of but little significance. If other branches or nerves are involved the importance may become considerable, especially considering the early period at which some pupillary changes occur.

Depression of the Lens in Cataract.—MR. POWER, London (*British Medical Journal*, October 26, 1901), makes a plea for the occasional performance of this operation. After referring to the fair measure of success obtained by native itinerants in India, who practice the operation with a

total disregard of all antiseptic precautions and even common cleanliness, he thinks that a field for it exists, notwithstanding the brilliant results obtained from extraction by modern methods. The classes of cases in which he advises it are those in which conditions are present which render it doubtful whether any operation should be undertaken—for example: (1) Those who are greatly enfeebled by age and other infirmities; (2) where physical obstacles to extraction are present, *e. g.*, small palpebral fissures, small eye and deeply set in the orbit; (3) chronic conjunctivitis and dacryocystitis; (4) considerable degree of deafness; (5) in the insane; (6) chronic bronchitis; (7) fluid vitreous, with tremulous iris; (8) where extraction has been unsuccessfully performed in one eye; (9) in the hemorrhagic diathesis.

Paralysis of the Facial and Paralysis of the Lateral Associated Movements of the Eyeballs of the Same Side, with the Electrical Reactions of the Affected Muscles.—PECHIN and ALLARD, Paris (*Gaz. Heb. de Med. et de Chir.*, August 29, 1901) insist that paralysis of the associated lateral movements of the eyeball alone or accompanied by other palsies, as a rule, presents great difficulties in diagnosing the situation of the lesion, due in part to the fact that our knowledge of the co-ordinating centres of the ocular globes is still imperfect. When the paralytic phenomena appearing to be due to an organic lesion show departures from the usual clinical picture, and, besides, the patient is an hysterical subject, the difficulty of determining the syndrome becomes very great. They report the following case:

Female, aged forty-six years, married, has suffered from hysteria and also true mental disturbance. About a year and a half before coming under the authors' observation she experienced sudden disturbance of vision, with deviation of the angle of the mouth. She continued to have frequent nervous crises, with visual and auditory hallucinations. Examination showed complete facial palsy on the left side. The skin of the forehead and cheek perfectly smooth; the left eye more widely open than the right; the mouth is slightly deviated to the right and drawn downward; the frontalis and orbicularis palpebrarum completely paralyzed on the left side; the left eye cannot be perfectly closed—during sleep a portion of the globe is exposed—but in spite of such partial lagophthalmia there has been no neuroparalytic keratitis; no epiphora; slight deviation of the tongue and uvula to the left (the paralyzed side).

Electrical examination gave the following results: No modification in the limbs and trunk; in the face considerable augmentation of faradic and galvanic irritability of the nervous trunk of the facial, of its three branches, and of all the muscles of the face. Diminution of the cutaneous sensibility to faradization.

Frequent headache, buzzing in the ears, especially in the right; hemianæsthesia of the left side, extending to the face; pharynx anæsthetic; ovarian spot on the right side.

Gait peculiar, spasmodic. She scrapes the ground with her feet, the latter hardly raised. Knee-jerk normal; reflex of the fascia lata preserved; Babinsky's sign absent.

Ocular conditions: Paralysis of the lateral associated movement upon attempting to look to the left. The eyeballs do not pass the middle line

Recently a modification has taken place in the right eye—slight adduction, with nystagmiform movements. The left eye always stops in the so-called primary position. There is no abduction. Convergence is preserved. When the patient closes the right eye and fixes a line drawn upon a sheet of white paper she quickly has diplopia followed by polyopia. There is besides colored vision. A black line appears bordered with a red and yellow margin; she sees intermittently two superposed rainbows. This monocular diplopia with disturbance of the chromatic sense was always present at repeated examinations.

Cornea completely anæsthetic to touch, with diminished sensibility of the conjunctiva, both palpebral and bulbar, of the left eye.

The visual field seems contracted, but the examination is very difficult because of the patient having a nervous crisis at every attempt at examination. This crisis is characterized by pain in the forehead, followed by contraction of the upper extremities; the head is thrown backward. It terminates by yawning and hiccoughs. It can be stopped by striking the sides of the neck with the hand. The crisis appears both when the attention is fixed and by electrifying the frontal region.

Fundus of the eye normal; no optic neuritis; no amaurosis; no amblyopia.

As to the seat of the lesion causing the ocular and facial paralysis, after excluding an hysterical, a peripheral, nuclear, and cortical origin, the writers conclude that the palsy is due to a lesion seated in the corpora quadrigemina or their neighborhood—the co-ordinating centres of the associated movements; these centres are extra-nuclear, supra-nuclear. The lesion is one of pressure, of doubtful nature, perhaps tubercular, involving the posterior longitudinal fibres. A lesion situated there would explain both the facial and ocular palsies.

Ocular Complications of Smallpox.—M. DUFOUR (Lausanne) finds that some ocular lesion occurs in from 10 to 15 per cent. of all cases of smallpox. In the majority of these it is a conjunctivitis, but in about 30 per cent. of the ocular cases the cornea is involved. Next in frequency to the cornea the lacrymal apparatus is liable to suffer. He reports a case in which on the twelfth day of the eruption the cornea became involved, an abscess formed, and it seemed as though useful vision would be lost. Subconjunctival injections of a solution of corrosive sublimate 1 : 2000 were practised at intervals of two or three days, with instillations of atropine. The corneal disease was promptly checked, and the eye recovered with a partial leucoma, numerous synechiæ, and vision equal to counting fingers at 3 metres, which might be improved by a subsequent iridectomy.

J. COURMONT and ET. ROLLET (Lyons) have encountered forty-five cases of involvement of the conjunctiva or cornea in 691 cases of smallpox. They have resorted to instillations of a solution of methylene blue, 1 : 500, repeated several times a day, to abort these ocular lesions. They regard the value of this treatment in preventing a permanent damage to the eye from variola as equal to that of the Credé method of using solutions of silver nitrate for the prevention of blindness from ophthalmia neonatorum. They regard the corneal affection as a specific process not dependent on the ordinary pyogenic organisms.—*Annales d'Oculistiques*, May, 1901).

Optic Atrophy Due to Mumps.—H. DOR (Lyons) reports two cases of optic atrophy following mumps, in which no other probable cause could be discovered. In one the visual disturbance was not noted for several months after the attack of mumps. In the other vision was markedly reduced in one month, and two months later the case had gone on to advanced atrophy. —*Transactions of the Thirteenth International Medical Congress.*

Iritis and Keratitis Due to Mumps.—A. PECHIN (Paris) reports the case of a young recruit who suffered a severe and prolonged attack of mumps which began the middle of February. When the patient left the hospital on April 6th there was still considerable swelling of the parotida. The latter part of April he noticed impairment of vision and redness of the eyeball, but without pain, photophobia, or excessive lacrymation. When seen the last of May there was well-marked iritis in both eyes, with some corneal infiltration in the right. Under general treatment, with atropine and hot compresses to the eyes, there was slow improvement, with restoration of full vision in the left, and V. 1/2 in the right.—*Recueil d'Ophthalmologie*, June, 1901).

Ocular Mumps.—Under this title, SENDRAL, Physician-Major in the French Army, gives a general review of the eye lesions that attend this disease. The most common is inflammation of the conjunctiva, with a strong tendency to chemosis, but with very little discharge; it is often accompanied by oedema. In a certain proportion of cases the cornea participates in the inflammation.

The lacrymal glands are liable to involvement along with the salivary glands, presenting an inflammation of much the same character.

The optic nerve may be the seat of congestion or pronounced inflammation, with temporary impairment of vision, and in rare cases this condition may be followed by optic atrophy. (See Dor's case above.) He finds that these ocular complications are much more likely to arise in adults than in children.—*Recueil d'Ophthalmologie*, February, 1901).

[The ocular complications of mumps are probably more common than is generally realized, the involvement of the conjunctiva along with other mucous membranes being the only one usually mentioned in descriptions of this disease. The fact that mumps frequently occur without the nature of the attack being recognized, and even without the patient coming under the observation of a physician, is a reason for special care not to overlook this origin for obscure disease of the eyes.—ED.]

Metastatic Ophthalmia in Pneumonia.—P. PETIT (Rouen) reports a case of bilateral conjunctivitis, and serpiginous ulcer of the right cornea, and panophthalmitis of the left eye. The patient, a woman seventy-nine years of age, had been sick for two weeks before entering the hospital. She came with both lungs somewhat involved, although the principal lesion was in the base of the right. Ten days later there was a conjunctivitis, with moderate discharge, and the cornea of the right eye became involved. One week after that the left eye exhibited oedema of the lids, exophthalmos, chemosis, haziness of the cornea, and slight hypopyon, tenderness of the globe and increased

tension. Vision was reduced to light perception. Subsequently the right cornea perforated, but in the left eye the inflammatory symptoms subsided. The patient died six weeks after admission to the hospital.

The case appeared to be one of general pneumococcus infection, this micro-organism being found in the various lesions, both pulmonary and ocular.—*Annales d'Oculistique*, September, 1901.

Postpartum Metastatic Panophthalmitis.—W. L. PYLE (Philadelphia) reports a case of this now rare disease.

The patient had a prolonged labor and instrumental delivery, followed by severe pain in the abdomen, high fever, and delirium. On the tenth day the right eye became inflamed and continued in that condition. When the patient was first seen, two and a half months later, although the cornea was clear and the iris not greatly altered in appearance, the eye was already atrophic. On microscopical examination it was impossible to determine whether the processes had originated in the choroid or retina and no micro-organisms were detected by the Gram-Weigert method.—*Proceedings of the Philadelphia County Medical Society*, April, 1901.

[This condition, happily now rare, was far more common before the application of antiseptic principles in midwifery. J. Schöbl, in his recent article on "Diseases of the Retina," speaks of having, "concerning this eye affection, experiences as numerous as they are sad." He served in the Obstetric Clinic and in the Eye Clinic at Prague, and says: "I remember several days during which I saw daily more than twenty individuals suffering from puerperal fever, of whom four or five had metastatic eye disease."]

In spite of the great change for the better, cases of the kind still occur, and there is danger in the presence of the serious primary disease that the eye complications may be overlooked or neglected—ED.]

Visible Blood Current in the Retinal Veins in Leukæmia.—K. GRUNERT (Tübingen) reports the ophthalmoscopic observation of a distinct visible movement of the blood-current in the retinal veins in a case of severe leukæmia. The patient, a woman, aged thirty-eight years, showed red corpuscles reduced to about 2,000,000 and the proportion of white corpuscles increased to 1:2.8. Vision was good— $\frac{5}{6}$ in each eye. But severe neuro-retinitis was present, each nerve entrance being covered by a swelling double its diameter, and extending six or seven dioptries into the vitreous. By close observation of the erect image there could be seen in the thicker venous trunks a distinct blood current, a fine drizzle (Rieseln), as if sand were rapidly moved in a red glass tube. The blood movement was naturally confined to the centripetal current obstructed at the papilla. In the smaller veins and in the arteries no such current was noticed. There was no appearance of pulsation.

The visible blood current has been observed with the ophthalmoscope in the eyes of fish and some of the other lower animals. The blood corpuscles in such animals are much larger than in man, and the amplification of the ophthalmoscopic image is also greater. Grunert explains the appearance in his case by the greatly increased proportion of the larger blood cells, the leucocytes, and by the fact that these larger blood cells would predominate

in the sluggish current near the walls of the vessel, while the erythrocytes would be carried by the swifter stream in the axis of the vessel.—*Centralblatt für praktische Augenheilk.*, August, 1901.

[Grunert has been unable to find in the literature any allusion to a similar observation regarding the blood current in the human eye; but such an one was published by USHER (*Ophthalmic Review*, December, 1896) among observations on the retinal blood stream at the time of death. As he describes it, "When the blood flow became visible the blood column in the vein was unbroken. The normal homogeneous appearance of the blood in the vessels became at first finely granular, and later on more coarsely granular; still later beading occurred, the red blood columns being broken up by clear gaps. There was a granular appearance in the arteries also."

The explanation offered by Usher was, "in the slowing of the circulation in the retinal vessels to such an extent as to allow the red blood-corpuscles to become visible, the larger granules being perhaps groups of red corpuscles, the smaller ones single corpuscles." The beading alluded to by Usher was similar to that which has been observed after obstruction of the central retinal artery, attended with slow movement of these comparatively large visible masses of blood along the vein toward the optic nerve entrance. It is quite different from the granular appearance, which preceded it in Usher's observation and which Grunert now describes. The full general history of Grunert's case is not given.—ED.]

Purulent Inflammation of the Palpebral Portion of the Lacrymal Gland, Rosenmuller's Gland.—LOB (*La Clinique*, Bruxelles, No. 45, 1901) calls attention to the above affection which has been but rarely described. He reports five cases. The accessory lacrymal gland is contained between the two leaflets, an anterior and a posterior, of the aponeurotic expansion of the elevator of the upper lid. These two leaflets unite above with the infero-inner wall of the orbital portion of the lacrymal gland, permitting only a very narrow path of communication with the palpebral portion; below the accessory gland is in immediate contact with the conjunctiva. The latter being little resistant, any swelling of the gland or collection of liquid therein encroaches upon the palpebral fissure, obliterating the cul-de-sac. This explains why the pus tends toward the conjunctiva.

The symptoms of the affection are: an inflammatory swelling, more or less hard and painful, the size of a small bean, situated entirely in the external upper portion of the upper lid, encroaching upon the palpebral fissure; there is partial chemosis limited to the external palpebral bulbar region; a yellowish point soon appears followed by a purulent fistula; finally as negative signs, the absence of serious involvement of the eyeball as well as of all influence upon the general health. At the most there may be present a slight saburral condition of the digestive tract; there is moderate local pain, some disturbance of motion upon looking outward, with momentary diplopia. The pre-auricular gland is frequently swollen and a little painful.

The inflammation sometimes involves the orbital portion of the gland as well, exaggerating the symptoms. As regards the tears, no modification is observed in disease of this portion of the gland, unlike when the orbital portion is affected; in the latter case the tears are diminished on the affected

side, lending support to the view that the orbital portion supplies the lacrymal secretion of psychic origin.

The disease lasts from one to two weeks. It is an affection of childhood and adolescence. The treatment is medical, not demanding surgical intervention; it will be very rarely necessary to open the abscess. It is preferable to let the little collection of pus evacuate itself. Warm antiseptic lotions and cataplasms are all that is necessary.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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OF PHILADELPHIA.

Dislocation of the Arytenoid Cartilage.—DR. HENRY L. WAGNER, of San Francisco, reports (*The Laryngoscope*, August, 1901) a case of traumatic dislocation of the left arytenoid cartilage in an elderly man, aged seventy-two years, who was struck on the neck by a drunken soldier. On regaining consciousness he was unable to speak, breathing was difficult, and he had intense pain in the region of the neck where he had been struck. Laryngoscopic examination the next day revealed an amount of swelling which rendered it difficult to trace the contour of portions of the larynx. Slight crepitation could be felt on the left side, but no abnormal motion could be perceived. A few days later, when the swollen condition had largely subsided, the left arytenoid cartilage was seen somewhat dislocated and thrown to the front of its normal position.

Mycosis of the Tonsils, Palate, and Base of the Tongue.—DR. E. HARRISON GRIFFIN, of New York, reports (*New York Medical Journal*, December 14, 1901) a case in a female school-teacher, which was eventually controlled by the combined effects of frequent tobacco smoking, avulsion of the vegetation, followed by cauterization with chromic acid, the use of a gargle of chloride of iron 3 drachms, glycerin 2 ounces, and water enough to make 3 ounces, 1 drachm of which was used as a gargle every three hours, and then swallowed.

Dr. Griffin states that he has treated more than fifty cases of this affection, all of them, as far as he remembers, in females. He suggests that the habit of smoking in the male may be the cause of preventing the development of the affection.

Paralysis of the Tongue.—DR. J. WESLEY SHAW, of Springfield, reports (*The Dental Cosmos*, November, 1901) a case of tongue paralysis of dental origin in which he located the seat of the disturbance in the upper left bicuspid tooth, which had a large amalgam filling disto-approximally.

Removal of the filling and systematic destruction of the gangrenous pulp and proper refilling of the tooth cured the case in a few days. During this treatment an induced current of electricity was passed from the nape of the neck to the floor of the tongue, principally by way of diverting the patient, and is therefore not accredited as essential in the cure.

Lipoma of the Pharynx.—A case of this infrequent pharyngeal neoplasm in a man, aged fifty-five years, was mentioned by MR. ROBERT H. WOODS, of Dublin (*Journal of Laryngology, Rhinology, and Otology*, October, 1901), in a discussion on tumors of the pharynx, before the British Laryngological, Rhinological, and Otological Association; and Mr. Lennox Browne referred to a case of Barnard Holt's whose patient died of suffocation while he was smoking.

The discussion, which is well worth consulting, was opened by Mr. Woods, who, after mentioning some typical instances of various morbid growths of the pharynx, detailed his methods of operative procedures, including some ingenious devices for securing loops in position for operation by incandescent section.

Fracture of a Gottstein Curette during an Operation for Adenoids.—DR. CHRISTIAN R. HOLMES and DR. H. STOWE GARLICK, of Cincinnati, report (*The Laryngoscope*, May, 1901) a case in the practice of each in which the cutting portion of a Gottstein curette broke as pressure was made in the initial step of the operative procedure. In Dr. Holmes' case, operated upon under general anæsthesia, the fragment was extracted by pressing his finger upon one end of the broken blade, so that it became embedded in his flesh, enabling him to withdraw it into the mouth, whence it was removed with forceps. In Dr. Garlick's case, operated upon without anæsthesia, the fragment was swallowed and passed three days later.

These instances, though rare, would indicate that the strength of the instrument, which might be diminished by repeated sharpenings, or even be originally defective, should be well tested before each operation.

Rhinoscleroma.—DR. CHARLES W. ALLEN, of New York, presented (*Journal of the American Medical Association*, December 14, 1901) at the fifty-second annual meeting of the American Medical Association a very interesting and admirably illustrated article on this subject.

Loss of the Cartilaginous Septum of the Nose during Convalescence from Influenza.—DR. L. DE MILLY, of New Orleans, reports (*Revue Hebdomadaire de Laryngologie, d'Otologie et de Rhinologie*, October 12, 1901) a case of an infant, aged five years, brought to him by its parents in consequence of a deformity of the nose. Examination showed that the cartilaginous septum had become effaced without any destruction of the mucous membrane, which was intact upon both sides.

Morbid Growths of the Nose.—DR. GEORGE DESVAUX, of Angers, reports (*Revue Hebdomadaire de Laryngologie, d'Otologie et de Rhinologie*, October 5, 1901) a case of supposed malignant tumor of the nose which was removed by several

operations with the incandescent electric snare, and which proved to be an hypertrophy of the mucous membrane of the lower turbinated body, containing numbers of capillary vessels surrounded with sarcomatous cells.

Morbid Growths of the Upper Air-passages ; Diffuse Papilloma in the Larynx and Trachea of a Child, Aged Four and One-half Years, and Requiring, Successively, Tracheotomy, Two Thyrotomies, and Fissure of the Entire Length of the Trachea.—DR. LEON BÉCO, of Liège, reports (*Revue Hebdomadaire de Laryngologie, d'Otologie et de Rhinologie*, October 19, 1901) a case in an infant from whom he had removed enlarged tonsils and voluminous adenoid tumors from the vault of the pharynx. It was subsequently found that in addition to these troubles there were papillomas of the larynx in the arytenoid region. These increased in size and rendered a tracheotomy necessary. Six months later thyrotomy was performed and vegetations were found filling the larynx. They were removed with cutting forceps, bistoury, scissors, and the electric cautery. Recurrence ensued. Five months later tracheotomy, followed by thyrotomy at the same sitting, was performed, revealing two enormous papillomas in the trachea as the cause of some precedent suffocative paroxysms. The laryngeal cavity contained considerable papillomas, although less extensively than at the first operation. Five days later the trachea was incised down to the sternal notch and growths removed from several portions of its extent, since which time the patient had remained free from trouble for eleven months.

Vocal Nodules.—DR. CHARLES H. KNIGHT, of New York, contributed a paper on this subject at the last meeting of the American Laryngological Association (*The Laryngoscope*, November, 1901) in which, among other things, he describes a case which developed under his personal observation. The case improved under systematic vocal exercises and daily instillations of adrenalin chloride, 1 : 5000.

Tuberculous Laryngitis.—At the recent British Congress on Tuberculosis, DR. ST. CLARE THOMSON, of London, read a paper (*Journal of Laryngology, Rhinology, and Otology*, October, 1901) on the "Principles of Treatment of Tuberculous Laryngitis," in which he states that the statistics of the Pathological Department of the Brompton Consumption Hospital show that the larynx is affected in more than 50 per cent. of the cases which succumb to pulmonary tuberculosis. (A remarkable showing to the writer of this notice in the light of his own forty years' experience.)

Dr. Thomson deprecates attempts at topical interference, and the principal conclusions arrived at are that pathology and clinical experience show that the focus of infection is near or in the crico-arytenoid joint in the majority of cases ; that many topical measures only distress the patient and hasten the progress of the disease ; that any persistent suspicious laryngeal catarrh should be treated seriously on even a presumptive diagnosis ; and that symptomatic treatment should be directed to an irritative, catarrhal, or obstructive condition of the air-passages. Silence should be enjoined, the disuse of the voice being proportionate to the degree in which the focus of infiltration approaches or interferes with the arytenoid joint.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Recent Cell Studies of J. Arnold.—The more recent studies of J. ARNOLD (*Archiv f. mikrosk. Anat.*, 1900, Band lv., p. 479-488, and *Anat. Anz.*, Band xvii., p. 517-519) turn chiefly upon the so-called *Granulalehre*, a theory of certain cytoplasmic granules. With a special technique Arnold has investigated certain general problems, such as the origin (pre-existence or importation) of the granules, their relation to phagocytosis, their value as an index or property of the cell's life. The technique is in general simple enough and the materials few; tongue, web, mesentery, subcutis, and dorsal lymph sac of the frog; warm stage, elder pith for inclusions; stains—especially methylene blue and *Neutralroth*, and a variety of inert substances—oils, iron, and the like—for injection.

A good idea of his work may be obtained from an earlier article, "Ueber Granulafärbung lebender und überlebender Leukocyten," *Virchow's Archiv*, Band clvii., S. 424. The tissues are examined alive and at various time-intervals. The effects are shown of using the stains in powder or solution, of injecting substances *intravital* or applying them to excised tissues.

The granules apparently pre-exist as granules or as hyaline droplets. The demonstration of the granules is possible both before and after the phagocytosis of indifferent bodies, such as fat granules. The granules Arnold takes to be essential structural elements of the cells—true plasmasomes—and their staining he looks upon as a vital property. Take, for illustration, a recent experiment: Upon a frog's tongue, held in the Thoma apparatus, dust methylene blue in finely granular state; drop on 1 per cent. NaCl, cover, and watch under a microscope. In from fifteen to twenty minutes certain cells in the middle of the so-called nerve papilla assume a blue-gray color, due to fine granules of the stain as yet laid only on cell surfaces. Later, an intense fine granulation appears in the subepithelial tissue, which is gradually made out to stand in connection with fine varicose fibres. Fibres are given off which end in a small swelling below the surface. The fibres remain stained several hours.

Certain curious and unpredictable results are obtained. For instance, in the above process cells occur at the periphery of the papilla which assume the stain early. These, it may be supposed, are peculiar or perhaps moribund cells. But Arnold is as yet uncertain whether diffuse staining of either nucleus or cytoplasm means in all cases cell-death. Again, the number of granules demonstrable by methylene blue is, as a rule, smaller than by *Neutralroth*, although the number of granules demonstrable in mast-cells is larger by methylene blue.

In the mesentery the stains occur especially near the lymph paths, of which the cells have granules in their processes.

It is hard to appraise these studies which Arnold has made. Little appears to have been done by others either in confirmation or in extension of this work.

An earlier study by Arnold ("Ueber die Structur und Architectur der Zellen," *Archiv f. mikrosk. Anat.*, Band. lii., S. 134) may be interpreted as an attempt to get the solid tissues into a state wherein blood-technique may be applied to them. By maceration in Lugol's solution Arnold was enabled to isolate the most varied cells (including nerve and smooth muscle cells) and to examine them in smears. The later work takes its departure logically from the desire to obtain for the solid tissues a second advantage usually conceded to blood work—the observation, namely, of the cells alive and in process of assuming stains; hence proceeds the stress now laid upon the "vital" nature of the granule staining. It is very easy to see, therefore, that many of Arnold's articles are hard to understand by themselves, and must be thought of as ancillary to the main problem—the cytology of the solid tissues of vertebrates.—E. E. S.

Structure and Histogenesis of Neuroglia Tissue under Pathological Conditions.—BONOME (*Virchow's Archiv*, 1901, vol. clxiii., p. 441) notes the dearth of recent work on pathological glia, and reports his own series of some twelve cases (including, besides gliomata, some glioses). Bonome attacks the problem after the fashion of the histologists. He wishes to do for pathological glia what has been done for normal glia, and so uses almost exclusively Weigert's differential methods. The plates recall those of Weigert's 1895 monograph.

The paper is divided into a number of sections, comprising the cytology of glioma, the distinctions between glioma and gliosis, the significance of epithelial elements in glioma, and the interrelations of cell and fibril in glia new-formation.

Gliomata are true tumors characterized by cellular richness (suggesting endothelial sarcomata), by exceeding vascularity, by tendency to necrobiosis with cavity formation, and by the absence of included nerve fibres or ganglion cells. Histologically, the cells of gliomata possess more or less embryonic characters, and resemble in turn sarcoma cells (proliferative activity marked) and ectodermic elements (proliferative activity less marked). Fibrillæ are rarely so numerous as in either normal glia or simple gliosis. In simple gliosis the fibrillæ are so numerous as to remind one of a fibrin network. The cells in gliosis are less numerous and less atypical than in glioma, for in glioma the cells seem never to have reached the embryonic stage in which epithelial and spongioblast forms have become differentiated; the cells are larger, the nuclei are more vaguely outlined, the picture is in every way more "peculiar" than in gliosis. The paucity of fibrillæ demonstrable in gliomata Bonome compares with a similar lack in three to five months' embryos treated by the same method.

Of interest on the genetic side is the finding of epithelial elements in gliomata (either lining cystic cavities or in groups or cords in a field of glia cells). Such are to be considered, according to Bonome, not as developmental errors or insignificant inclosures, but rather as instances of genuine preservation of the embryonic type, and, in particular, of the wandering

tendency which characterizes those earliest cells. To explain such heterotopia of glia cells alone one may suppose no neuroblast elements were present in the original foci, or perhaps that the differential capacity of such cells is lost and that the wandering derivations are fated at least to lie as inactive elements within the tumor—neither true glia cells nor true nerve cells. Certain cases are adduced, notably one of Stroebe's, to give color to the belief that the occurrence of epithelium in gliomata is not secondary and that, whether or no the cavities occur as lateral branchings of the central canal or in truly independent foci, the deposit of glia is secondary, that of epithelium primary.

Bonome's conclusions upon fibrils are as follows: The fibrils are chemically different from the cell-protoplasm, nor are any transitions observable in staining reaction according to the vicinity or remoteness of the fibril from the cell. The system of glia fibrils, it is a triumph of modern technique to have shown, is an intercellular (not cytoplasmic) substance. But the fibrils are probably derivatives of the cytoplasm, not sproutings from it, but chemical deposits incident to a certain stage in the cytoplasm's development. In general the earlier one catches either normal or pathological glia the fewer fibrils; glia cells of the embryonic type do not produce fibrils (how easy then to confuse tumors of such type with sarcoma!). The oldest part of a glioma will be doubtless that which contains most fibrils.

From the necessary combination of such an array of factors is easily explained the frequent absence or paucity of glia and fibrils in the neighborhood of foreign bodies, tubercles, and the like. Return to embryonic condition, over and above some stimulus, is required to yield new glia. As for fibrils, the proper stage in the cytoplasmic development may not have been reached. If new fibrils are developed out of proportion to cells present one must consider that the cytoplasm may contribute merely the differentiating element in their evolution, or perhaps the cells, exhausted in the production of fibrils, may be earlier destroyed.

In certain cases glia acts precisely like connective tissue (though always chemically to be distinguished). A plate is given to show penetration of pia by new-formed glia. Glia fibrils are distinguished from fibrin-threads not alone by failure to branch, but also by being subject swiftly to cadaveric change (granulation). [E. E. S.]

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ERRATA. DR. ALFRED C. CROFTAN's article in JOURNAL for April, 1902.

Page 666, fourth line of Experimental Series 10, for "102 oz." read "1 oz."

" 671, fourth line from bottom, for "by a process" read "a by-process."

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THE LOCALIZATION OF THE MENTAL FACULTIES IN THE
LEFT PREFRONTAL LOBE.

BY CHARLES PHELPS, M.D.,
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IN order to ascertain with the greatest possible certainty whether the law which seemed to have been established by my own observations, connecting symptoms of mental disorder or default exclusively with left prefrontal lesion, was absolute, still larger groups of cases with limited lesions were gathered without discrimination from such sources as were available, and subjected to analysis.

These were divided into three classes: 1, atrophies; 2, pistol-shot wounds; 3, abscesses and tumors.

First: Cerebral atrophies form a smaller class than either of the others, and are very often unsuited to the study of cerebral localization by reason of associated congenital idiocy. Idiots, lunatics, and epileptics suffer from general cerebral disturbances which make it usually impossible to define the pathological relations of lesions of special areas. If progressive mental decadence begins at some period subsequent to birth, and hemiatrophy is found to exist, it might be a legitimate inference that they are directly related. It is better, however, to limit the field in which it is sought to establish the relation between a normal or an abnormal mental condition and a cerebral atrophy to less complicated cases.

I have found only twelve cases reported in comparatively recent years, including two in the series of traumatisms recorded by me heretofore. There are probably others which I have failed to discover, or which are noted in Continental journals, which I have not consulted. The number is so small that they may be reproduced here in more or less detail.

CASE I.¹ *General atrophy*.—Patient, aged fourteen years. "Mind a miserable wreck." The brain was shrivelled, but particularly in front and on the upper and lateral part of the left hemisphere; the surface was irregular, as though elevated; and rose to various heights according to the degree of atrophy.

CASE II.² *Left hemiatrophy*.—Idiot girl, aged seven years; hemiplegia from infancy; indicated early all the signs of imperfect mental development; speech very defective; communicated mainly by gestures and inarticulate sounds; imbecility progressive; "became more and more like an unclean beast;" remarkable atrophy of the right side of the body.

Necropsy. "Right hemisphere of cerebrum apparently quite healthy; color of gray matter was the usual light red, and convolutions were natural." Left hemisphere much smaller; whole hemisphere soft and fluctuating like a bladder, on account of the ventricular fluid; gray matter very pale and yellow; convolutions much thinned; arachnoid membrane much thickened on left side, and considerable subarachnoid fluid. Atrophy of the right cerebellar lobe and of the right side of the spinal cord. Skull much thickened on the left side.

CASE III.³ *Left hemiatrophy*.—Patient, aged seven years; quite well till within seven weeks; illness began with convulsions; great mental decadence; could understand at times, but had no power of speech; did not know his own bed; ate his own feces; walked actively, but dragged his limbs a little; right side of body atrophied and flabby; left hospital in nine weeks, returned five months later, and died from a burn four months afterward.

Necropsy. Left hemisphere smaller in all its dimensions; right hemisphere quite normal.

CASE IV.⁴ *Left hemiatrophy*.—Patient, aged forty-five years; mental condition not stated.

CASE V.⁵ *Atrophy of both frontal lobes*.—Patient, aged seventy-three years; under observation for five years; during first three years showed some signs of mental alienation; speech entirely ceased; asked for nothing either by eye or gesture; food put in his hand, even, was not carried to his mouth; did not recognize his companions or the bed in which he slept, or know when he was cold; "finally lost all sentiment of spontaneity of will;" no hemiplegia.

Necropsy. Atrophy of both frontal lobes, involving first, second, third, and ascending frontal convolutions; also ascending parietal convolutions and paracentral lobules.

CASE VI.⁶ *Left hemiatrophy*.—Patient, aged fifty-five years; "there was a deficiency of intelligence."

Necropsy. Left hemisphere weighed three and one-quarter ounces less than the right; convolutions softened to depth of one-fourth inch.

CASE VII.⁷ *Left hemiatrophy*.—Patient at age of two years was feeble-minded, and at about that time became paralyzed; at ten years of age he became epileptic.

¹ Barlow. *Lancet*, 1849, vol. i. p. 92.

² J. L. C. Schroeder and Van Der Kolk. *A Case of Atrophy*, etc. London, 1861.

³ Jones. *London Medical Times and Gazette*, 1874, vol. i. p. 371.

⁴ Boyer. *Bull. Soc. Anat.*, 1877, p. 609.

⁵ Baraduc. *Bull. Soc. Anat.*, 1876, p. 271.

⁶ Boyd. *Med. Chir. Trans.*, vol. xxix. p. 18.

⁷ Spiller. *Journal of Nervous and Mental Disease*, 1898, p. 1.

Necropsy. The entire left lobe was notably diminished in size; the prefrontal lobe was well developed. The motor fibres of the left hemisphere were totally destroyed, yet he was able to walk without a crutch. The left pyramidal fibres were entirely destroyed.

CASE VIII.¹ *Left hemiatrophy.*—Patient, a child, aged eleven months, died from diarrhoea after five days' observation at the New York Foundling Asylum. Was exceedingly restless and prone to beat its head against the side of the crib.

Necropsy. A pad of fibrous tissue well supplied with bloodvessels, closely adherent to the dura mater, and separable from the pia mater only by tearing, covered the left half of the brain; marked atrophy of nearly the whole left hemisphere; tip of frontal, the occipital, and lower portion of temporal lobe normal; throughout the shrunken portion the convolutions were small, hard, and of a yellow wax-like color. The middle cerebral artery was apparently intact and pervious. The fibrous pad was considered to be secondary to the atrophy.

CASES IX. and X.—Atrophy of both frontal lobes and of the left frontal lobe, respectively, are detailed in the previous series of traumatisms.

CASE XI.² *Right hemiatrophy.*—Patient was a woman, aged thirty years, mentally and physically well till the close of her fourth year, when she became epileptic. At school she was able to learn fairly well. Subsequently she manifested an ill-will to children. The irritability and violence which led to her being placed in an asylum arose in part from her epilepsy and in part from her being a subject of ridicule to young children from her uncouth appearance. "After admission to the asylum she employed her time in reading the Bible and complaining about her illness." She had no aphasia and no hemiplegia. She died from phthisis pulmonalis.

Necropsy. The convolutions of the left hemisphere were normal in size and appearance. The right hemisphere was greatly atrophied, both as regarded size generally and as to the frontal, parietal, and occipital convolutions. The right superior middle and inferior frontal convolutions were present, but were of only one-third the size of those of the opposite hemisphere. The ascending frontal convolutions could not be defined with any accuracy. The corresponding portions of the centrum ovale were also atrophied. "The small side of the pons corresponded with the left or atrophied side of the cerebellum, while the small side of the medulla was the right, corresponding with the atrophied side of the cerebrum."

CASE XII.³ *Right hemiatrophy.*—The patient was for three years previous to his death under the observation of Dr. Pearce Bailey. He had left hemiplegia, with rigid contractures of the arm and leg, and scarcely any other symptoms; only a little subjective numbness; no objective anaesthesia; no loss of special sense; and no loss of control of sphincters.

In contrast with the pronounced physical symptoms, pointing to an extensive lesion of the right half of the brain, was the absence of disturbances referable to the psychic faculties. Speech was perfectly

¹ Bovaird. Med. Record, 1899, vol. ii. p. 762.

² Howden. Journ. Ment. Sci., 1875, p. 238.

³ Bailey. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1899, vol. cxvii. p. 321.

normal; reading was not interfered with; memory was apparently unaffected.

"I never noticed in my frequent conversations with him during the three years, nor was there ever reported to me by the house staff or attendants, anything to indicate that the man's character or mental capacity had been affected in any way by the cerebral affection. He was courteous, intelligent, and patient; he was cheerful and attentive, and his power of attention was very good; he read the papers attentively, liked to talk politics, and was interested in the affairs of the hospital. He was singularly free from fits of depression, emotional excitement, irritability, apathy, or of any other of the manifestations of abnormal mental states. He was cleanly, and required such care only as his physical infirmities rendered necessary."

Necropsy. "Dura mater moderately thickened over anterior part of the right hemisphere; beneath it in this region was a cyst in the meshes of the pia mater, six inches in diameter. The left hemisphere was normal in size and in configuration of the convolutions. The right hemisphere was greatly shrunken. That portion of it posterior to the fissure of Rolando was atrophied in the individual convolutions, but the convolutions retained their natural topography. The region anterior to this fissure, comprising all the frontal lobe except that part lying to the inner side of the olfactory tract, was occupied by a yellowish-white semi-translucent oedematous tissue overlying a soft mass as a membrane. Its surface was very uneven, presenting elevations and depressions in such wise as to suggest diminutive convolutions. On section it was found to be dirty white in color, fibrous, and somewhat more resistant than brain tissue, and showing no brain matter.

"Thus the macroscopical appearance of the right hemisphere presented posterior to the fissure centralis a slight atrophy of the brain substance, while anteriorly to it nearly the whole frontal region was represented by a shapeless mass. There was no atrophy of the cerebellar hemispheres, but there was of the right basal ganglia and of the right half of the pons and medulla."

Microscopical examination. The membrane covering the mass was thickened pia mater; the mass itself was connective tissue, cells, and fibres, and a very friable amorphous matter which contained blood pigment. There were no traces of ganglion cells new-growth as such.

There are three cases of right hemiatrophy quoted by Van Der Kolk, which, like the two just described at length, afford striking evidence of the independence of the intellectual faculties of anatomical substrata in the right cerebral hemisphere.

These were: Andral's case; traumatism confined to right hemisphere; "pia mater formed a cyst in which no traces of cerebral matter remained;" floor formed by optic thalamus, corpus striatum and parts on a level; "yet he had a good education, had a good memory, good address, and exhibited as much intelligence as most men."

Thiaudiere's case: Same mental condition as in that of Andral; nothing remained of the right hemisphere but atrophied corpus striatum and optic thalamus.

Gall's case: Clergyman preached and catechised three days before his death; half of the right hemisphere completely softened and changed into a yellow granular substance.

Three of the preceding cases are not convincing; two as instances of atrophy involving both frontal lobes, and one as the history of an idiotic subject. The twelve others afford substantial evidence that the control of the mental faculties resides exclusively in the left hemisphere.

Second: Pistol-shot wounds are especially well calculated to exemplify differences in symptoms, as the right or left lobe is the seat of lesion, since they are injuries in which the lesion is often strictly localized. The whole number of cases reported from English and American sources during the years 1879 to 1895, inclusive, is probably not more than 150, of which it has been possible to collate for the present purpose 110; 58 of this number involved the frontal lobes; 26 of these were limited to the right lobe, and 24 to the left lobe; eight included both lobes. The cases which terminated in recovery, and on that account probably deemed most worthy of record, were in large proportion; and in them the situation of the lesion was made certain by the use of the probe, by operation for the removal of the ball, or by the appearance of brain matter at the surface. Not one of the 26 cases in which the cerebral wound was confined to the right side presented at any time any symptom of mental or emotional disturbance aside from the stupor or delirium which is characteristic of general contusion.

In 32 cases the ball traversed the left frontal lobe, in 8 of which there was no means of determining the mental condition, the primary loss of consciousness having been permanent or the general symptoms entirely unnoted. In 13 of those remaining, manifestations of mental derangement were distinctly evident. In several of the others the mental condition was not specifically mentioned, and the histories were otherwise deficient. There are still a limited number of cases in which with a wound of the left lobe the mental faculties were said to have been unimpaired; some of these cases are stated to have exhibited some form or degree of aphasia, and in nearly or quite all of them the lesion was of the posterior part of the lobe.

This series of cases, so far as the histories are complete and intelligible, fully indicates that intellectual and emotional derangement are symptomatic, not only of lesion of the left frontal lobe, but also of its anterior and central portions.

Third: Cerebral abscesses and tumors, like pistol-shot lacerations, are often limited to a single lobe, and therefore like them are well suited to a study of the relation which symptoms bear to the site of injury. A collection of cases of this character involving the frontal lobes which I have made is wholly confirmatory of the conclusions which have been deduced from the examination of cases of traumatic origin. It

comprises all the cases, both those reported in full and those condensed from the transactions of societies in the leading English and American journals from 1891 to 1900, inclusive. It also includes the earlier cases instanced by Ferrier to demonstrate the absence of motor function in the frontal region. Out of 607 limited lesions of the nature specified, 137 were of the frontal lobes, 21 of which were not further specialized; and in 18 others the mental conditions were not mentioned; leaving 98 cases for analysis; 51 of these involved lesion of the left, and 34 of the right lobe, and 13 of both lobes. The lesions in all cases were verified by post-mortem examination.

The cases in which lesion was limited to the left lobe were 51 in number; of these, 46 presented symptoms of mental default or disorder, and 5, it was said, did not. The affirmative cases are too numerous for description in detail, but some generalizations will serve to indicate the character of their symptoms. In many cases which were briefly stated the mental condition is noted simply as one of "mental failure," "dementia," or "decadence," or of "psychical changes." In others, stupor or stupidity, apathy, hebetude, silliness, loss of power of comprehension, loss of interest in affairs, general or special changes in character, delusions, irritability, incoherence, loss of memory, refusal to speak, various disturbances of speech, and still other individualizations of mental disorder, singly or in combination, are variously specified. As might naturally be expected, the observation made for direct traumatism, that subcortical disintegrations or extensive cortical lacerations were usually followed by mental default rather than by mental aberration, has an application to the lesions now under consideration. Tumors or abscesses centrally developed which attained large size were very characteristically accompanied by stupor, hebetude, or apathy too profound to admit the manifestation of more positive psychical changes. If the lesion were of smaller extent at the time the history of the case began, a greater number of mental symptoms was likely to be developed in its progress. The loss of the power of attention, which Ferrier regarded as the essential result of his removal of the prefrontal lobes in certain of the lower animals, and which some writers have insisted upon as characteristic of prefrontal lesion in the human subject, was not a distinctive symptom of the acute traumatism which have been described, and it does not seem to have been recognized as such in these cases of more chronic development. It was undoubtedly often noticeable in the progress of the cases which I have personally observed, but only as an incident of general conditions of hebetude, apathy, indifference, or progressive dementia, not as a primary defect. Another symptom which has been regarded as a specific indication of frontal lesion is a loss of control of the rectal and vesical sphincters. This diagnostic inference is true only in certain cases. It then implies

a lack of mental self-control resulting from deficient will, or a mental impairment by which the exercise of reason and judgment is prevented or suspended. Lack of sphincteric control from such mental deficiency was illustrated in the case of tumor, Case I., recorded heretofore.¹ In that instance the attendants noted in the beginning the patient's evident first impulse to get up and go to the closet, and then his momentary hesitancy before he urinated in the bed. It is, however, quite as frequent as an indication of laceration of any other part of the brain as of this, and is diagnostic of the nature rather than the seat of the lesion.

In an enumeration of symptoms indicative of mental disorder or default dependent upon left frontal lesion, delirium and loss of consciousness in whatever degree must be excluded. These conditions attend general cerebral lesions, such as hyperæmia and œdema, or the cortical inflammation which is a factor in arachnitis. They occur in cases involving distinctive left frontal injury, but the always possible or probable existence of a concomitant general lesion prevents their acceptance as localizing symptoms. They are not only not characteristic of limited lesion, but are more pronounced in its absence, and directly proportionate to the extent of the general circulatory disturbance. If the cerebral contusion is slight it will not occasion delirium or a recognizable loss of consciousness; if it is well marked some degree of delirium, or some loss of consciousness, is inevitable.

If the cases which have been cited have been correctly interpreted, other forms of mental disturbance or mental default do not occur without an additional and specific lesion of the left frontal lobe. Delirium is a mental disorder, and as such, it would seem, should be referable to the same frontal area. It would not be unreasonable to suppose that it depends upon that part of the general lesion which implicates centres through which the mind is controlled, just as the irregular movements or imaginary sights and sounds may depend upon implication of the motor or of the visual and auditory centres.

Consciousness has been variously defined, but all definitions heretofore have involved an assertion of the activity of the mental faculties. It is, in fact, ordinarily manifested by the exercise of these faculties, and the persistency of mental activity while consciousness remains, even when all other cerebral functions have been suspended, is so nearly constant that its absolute constancy has been assumed, and consciousness held to be another name for the mind itself. There are reasons for believing that consciousness may be differently construed; that it is no more than a mere subjective recognition or sense of existence—a passive condition of being which simply permits the exercise not only

¹ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1902, p. 570.

of the mental faculties, but of all the functions of the cerebral system. In a state of entire unconsciousness these functions while, it may be, preserving their integrity are in abeyance; if the loss of consciousness be incomplete, one or all of them may be roused to limited action. The exercise of the intellectual faculties, like the exhibition of feelings or emotions, the production of voluntary motion, or the appreciation of tactile or special sensation, is an accident, not an essential attribute, of this state of simple existence. The dictum of Hughlings Jackson, that "will, reason, memory, emotions, are artificially distinguished aspects of one thing,"¹ remains true; but that "one thing" is a state of mental energy rather than a "state of consciousness." This conception of consciousness is sustained by three considerations:

1. Mental activity may exist when consciousness is abolished. This happens in dreaming, in the unconscious condition of general anesthesia, and in the hypnotic or somnambulant state.

2. Consciousness may exist when the mind is wholly inactive. This proposition is based in part upon introspection, or, to speak more exactly, upon retrospection, since phenomena cannot be studied at the exact instant of their occurrence. (Sully.) The same power of directing the attention inward to the phenomena of mind can be directed equally to a study of consciousness devoid of mental phenomena. It is the experience of many persons that there are occasions between sleeping and waking when the oblivion of slumber has given way to an indefinable sense of existence, void of feeling of pleasure or of pain, or of perception of time or place, which precedes the resumption of thought. This form of evidence, rejected by Comte, is recognized in modern psychological investigation, and will appeal to the judgment of different investigators according to their degree of confidence in their own and in other people's accuracy of subjective analysis of feeling as well as of mental processes. There are also certain observations of pathic conditions which confirm the reality of such a conscious state of mental vacuity. The inception of returning consciousness after the heavy sleep which sometimes marks the passage of the crisis in typhoid or other fever has been described by patients as being without thought or feeling, or, again, as being characterized by feeling without thought or emotion. In some cases of cerebral traumatism consciousness is in part regained, as shown by the feces and by responses to cerebral stimulation, when no signs of intelligence can be obtained. Localized cutaneous irritations will produce corresponding localized movements in parts controlled by the inferior cerebral sensorimotor centres, and pupillary contraction will follow stimulation of the visual area. The case of the child detailed heretofore is strongly illustrative of the condition of

¹ "Evolution and Dissolution of the Nervous System," *British Medical Journal*, 1884, p. 704.

² *THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, April, 1902, p. 575.

consciousness without the slightest evidence of mental life, which in this instance was prolonged for a number of days.

If, then, consciousness and mind may exist independently of each other, they certainly are not convertible terms.

3. The traumatic structural alterations by which consciousness is impaired or destroyed, and by which mental action is disordered, are different in character and extent. Mental disorder or default results from a limited lesion of a specialized area, which, in fatal cases at least, is usually destructive; consciousness is destroyed in greater or lesser degree by a general lesion which inhibits the cerebral nutritive processes, and is effective according to its extent, without reference to the region involved. This fact—that consciousness is not lost except when a considerable part of the brain has been functionally destroyed by inanition of its cells, without reference to region, while each differentiated cerebral function may be destroyed by a lesion of a very limited but recognized area—is pathological proof that the conclusion reached on other grounds, that consciousness is independent of mental activity, is correct.

In 5 out of the 51 cases of left frontal tumor or abscess, as in 2 others in which both lobes were implicated, it is stated that the mind was unaffected. These were:

CASE I.¹—Traumatic. "Patient cured; preserved his reason; could walk and talk."

CASE II.²—Tumor. Epilepsy for ten years; patient "intelligent."

CASE III.³—Tumor of cortex. "The mental condition was normal."

CASE IV.⁴—Traumatic abscess. Slight wound over left orbit; on the sixth day patient was "dull and heavy," and on the seventh day was trephined; ten months later, "boy was in perfect health; no mental impairment."

CASE V.⁵—Traumatic. Ramrod driven quite through the skull from just above left orbit, emerging over left motor area of brain; no brain matter followed; probe passed into cranial cavity; temperature normal; right arm clumsy; neither temperature nor paralysis increased; on second and third days bits of brain were extruded not to exceed altogether the bulk of a filbert.

CASE VI.⁶—Traumatic. Gunshot wound transversely through middle of both lobes; "no mental symptoms; died from encephalitis some months later."

CASE VII.⁷—Lesion of both lobes. Patient "retained his senses and intelligence until fatal encephalitis set in."

Not one of the cases affords evidence to justify a conclusion that the mental condition remained normal. In 6 of them no detail of history

¹ Morrin. Quoted by Ferrier from Pitres. *Localization of Cerebral Disease*.

² Batten and Collins. *Brain*, 1898, p. 291.

³ Beevor. *Brain*, 1898, p. 291.

⁴ Southam. *Brit. Med. Journ.*, 1891, vol. 1. p. 1015.

⁵ Barritt. *Lancet*, 1891, vol. 1. p. 18.

⁶ Trousseau. *Gaz. Hebdomadaire*, 1864.

⁷ Fayer. Quoted by Ferrier. *Localization of Cerebral Disease*.

is given—nothing but the mere assertion of the writer that the patient “preserved his reason,” “retained his intelligence,” or “had no mental symptoms,” or “no mental impairment.” Two of the patients whose intellect remained above reproach died of chronic “encephalitis,” and another was an epileptic fourteen years. The lesion in the seventh case, the fifth of the series, if a wound of the brain at all, was evidently, from the history given, no more than a scratch upon its surface; 4 of the cases are of traumatic origin, and are not properly referable to any one of the groups which have been collated for examination. They have been cited only in order that every known case of alleged infraction of the law that left prefrontal lesion occasions mental disturbance should be given consideration.

The 11 cases of conjoined lesion of both frontal lobes, presenting psychopathic symptoms, have in that regard nothing to distinguish them from similar cases in which the left lobe alone is involved.

The number of tumors and abscesses confined to the right frontal lobe is somewhat smaller than the number of those of the opposite lobe.

There were in all 34 cases, 24 of which, it is distinctly stated, suffered from no form of mental impairment; 10 cases are described as attended by symptoms of mental disorder or default. They were:

CASE I.¹—Abscess. Death in two days after development of symptoms, from rupture into right lateral ventricle and asphyxia; aphasia was the only symptom mentioned.

Aside from the existence of a possible functional anomaly, the occurrence of aphasia demonstrated the extension of disease to the left lobe.

CASE II.²—Tumor. “Occupied whole extent of right frontal lobe; left motor area appeared quite healthy; decussation of pyramids normal. A convulsion was the first symptom noted; this was followed later by complete right hemiplegia; the patient was mentally very dull.”

The occurrence of right hemiplegia indicates that the left hemisphere suffered from the general increase of intracerebral pressure, which, at the same time, obscured the mental faculties.

CASE III.³—Cyst. Situated in right frontal lobe, below the level of the corpus callosum. Motor disturbances of the left side and left face; patient became drowsy and apathetic.

Apathy is recognizable as a psychopathic symptom only when unattended by drowsiness. It seems in this instance to have been caused by an increase of intracerebral pressure following operation.

CASE IV.⁴—Abscess. Destroyed nearly the whole substance of the lower and anterior two-thirds of the right frontal lobe, and contained about three ounces of pus. A second and smaller abscess situated beneath ascending frontal and ascending parietal convolutions. Purulent bronchiectasis of lower two-thirds of right lung. Pain in right frontal region; convulsive seizures of left side of face; mental hebetude

¹ Mangan. Brit. Med. Journ., 1896, vol. ii. p. 1087.

² Sharkey. Brain, 1898, p. 291.

³ Sharkey. Ibid.

⁴ Eskridge. New York Medical Journal, 1895, vol. ii. p. 168.

on the seventeenth day; left hemiplegia; semi-consciousness; coma; operation; trephination over right motor area; one to one and one-half ounces of pus evacuated from smaller abscess; larger abscess not discovered.

Hebetude, semi-consciousness, and coma evidently resulted from gradually increasing intracranial pressure.

CASE V.¹—Syphilitic tumor situated in posterior part of the first and second right frontal convolutions. Epileptiform convulsion, followed by left hemiplegia; "mental condition very much impaired." Patient recovered from all symptoms under antisyphilitic treatment, and again on their recurrence. Later, died in a convulsion.

Toxæmic origin of mental symptoms demonstrated by their disappearance under specific treatment.

CASE VI.²—Syphilitic tumor situated in right frontal subcortex. Patient maniacal and incoherent, with delusions.

Mental condition the probable result of syphilitic toxæmia.

CASE VII.³—Syphilitic gummata in centre of right frontal lobe and old syphilitic meningitis. Duration of disease five years; after three years gait became ataxic; extreme dementia; muscular tremor of tongue, lips, face, and fingers; knee-jerk exaggerated; severe convulsions occurring three months before death; diagnosis of paresis made.

Symptoms were properly attributed by the writer (Clark) to pressure from products of chronic meningitis.

CASE VIII.⁴—Sarcoma of right frontal lobe following trauma. Mental decadence and loss of memory; epilepsy for twelve years.

As site of the original injury is unknown the anatomical basis of the early mental symptoms cannot be determined.

CASE IX.⁵—Glioma and cyst of right frontal lobe, involving middle of first convolution and subjacent white substance; intracranial pressure greatly increased; convolutions flattened and sulci effaced. "Marked change in mental condition; silly expression; at times irritable, obstinate, and passionate; at other times depressed and stupid; memory markedly impaired; delusions of suspicion; severe general epileptiform convulsions."

The excessive intracerebral pressure was evident in the flattening of the convolutions and effacement of the sulci. It seems probable, however, from the nature of the symptoms as well as from the great amount of pressure, that other structural changes, not mentioned in the history, had taken place.

CASE X.⁶—Traumatic meningitis and abscess. Abscess occupied greater part of right frontal lobe; "right hemisphere enlarged at expense of right temporal and left frontal lobe." Apathetic on the third day; stupor later; operation; trephination in both frontal regions disclosing arachnitis, with much serous effusion and pial hemorrhage; recovery four months later; some loss of memory and occasional attacks of stupidity or indifference to surroundings, with bulging of both trephine openings; patient intelligent, but reasoning power slow. On reopening the wound on the right side eight ounces of pus were evacuated.

¹ Bramwell. Brain, 1899, p. 1.

² Beadles. Lancet, 1896, vol. i. p. 102.

³ Clark. Journal of Nervous and Mental Disease, 1895, p. 278.

⁴ Clarke. Brain, 1896, p. 291.

⁵ Bramwell. Brain, 1899, p. 1.

⁶ McCosh. Medical News, 1896, p. 42.

Both the primary and the secondary mental impairment, like the symptoms in the previous case, are explained by the greatly increased intracranial pressure.

The reasons assigned for believing that in each of the foregoing cases such evidences of mental derangement or default as existed were to be accounted for on other grounds than the right frontal lesion is scarcely open to question. In some of them the clinical history proved the extension of disease to the left side; in others there was an admitted toxæmia, which is known to derange or suppress the functions of the cerebral nerve centres; and in many the increase of intracerebral pressure was sufficient to produce whatever symptoms of abnormal mental condition were manifest.

The influence of increased intracranial or intracerebral pressure has been generally recognized as the constant source of confusion in estimating the pathological value of cerebral lesion. Ferrier¹ has remarked the difficulty of distinguishing between "causation and co-existence" and "the continual source of doubt which exists as to whether effects are the direct consequences of the lesion or merely the expression of general functional derangements," and Charcot² has pointed out "the necessity of accepting with extreme caution cases of tumor compressing the cerebral convolutions, since the effects of pressure may be felt at a distance from the seat of the lesion." This is true not only for tumors and abscesses, but for limited traumatism attended by marked general contusions. It is in either case the secondary vascular derangements which produce pathic conditions so like those of the essential lesion as to render them indistinguishable from each other. It is, therefore, the comparatively limited and single traumatism, or the idiopathic lesions which are merely destructive or are insufficient in size to occasion more than localized circulatory disturbance, which must form the basis for cerebral localization. The acceptance of this proposition involves the exclusion not only of cases of right frontal lesion opposed to the contention of an exclusive control of the mental faculties on the left side, but of many left frontal lesions which sustain it. Of the 57 cases of left frontal lesions in the series under consideration a considerable number were undoubtedly in this category, but by far the greater number were so limited in size as to make it possible to legitimately connect them with the symptoms of mental derangement exhibited.

A collection of cases was made by Starr,³ some years ago, with direct reference to their bearing upon the question of a centre or centres of control in this region for the mental faculties; but no intimation was

¹ Localization of Cerebral Disease, London, 1878, p. 2.

² Rev. Mens., 1877, vol. i. p. 6.

³ M. A. Starr. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1884, p. 366.

given by him of any closer specialization of such a centre in either lobe, to the exclusion of the other. Ten of the histories which he cites make no mention of the mental condition; 4, which involved the left frontal lobe, note symptoms of mental derangement, and in 5 of the 9 limited to the right frontal the mental condition was stated to be normal; 2 of the right frontal lesions, an abscess and a sarcoma, were of long duration, and toward the end of life the patient became in the one case morose, and in the other incoherent—conditions common to all exhausting diseases at their close, and neither of them infrequent, as general nutritive changes in the brain increase. The 2 cases remaining, both reported by Mills, were cases of syphilitic tumor; the first invaded the gyrus fornicatus and corpus callosum, and thrust the hemispheres apart anteriorly; the second extended into the parietal lobe; and in both mental decadence was unquestionable. It may be said of these cases that they are to be excluded from consideration in the study of localization on two grounds, previously stated: (1) Their size and their implication of contiguous parts, causing disturbance of general cerebral circulation and function; (2) the concurrent syphilitic toxæmia, which might equally with the local lesion have been the cause of the cerebral symptoms present.

The mass of clinical evidence which has been here presented entirely justifies the expressed belief of Hughlings Jackson that centres of control exist in the forepart of the brain which form the substrata of the higher mental operations. Further, it equally justifies the writer's contention that these higher centres of control are resident in the left, to the exclusion of the right prefrontal lobe. The mental default, decadence, or aberration; the apathy, imbecility, delusions, changes in character, and lapses of memory—some one or more of which in some degree marked every instance of left prefrontal lesion—and their absence as specific symptoms in practically every instance of right prefrontal lesion, are facts for which no other explanation seems adequate. If the facts as reported in some exceptional instance hereafter should seem to be irreconcilable with this localization it should be borne in mind that on the one hand symptoms as well as lesions are often negligently observed, and that on the other their significance often fails of recognition. The positive evidence afforded by such cases as have been cited in which both *ante-mortem* and *post-mortem* conditions have been determined is not to be invalidated on the mere report of a few scattered cases made by possibly untrained observers, or supported only by histories deficient in the record of essential facts.

There may be other cases, like those of Mills previously cited, which, on account of the known competency of the observer, are to be more seriously regarded. Even such cases, should they be found to exist, and, unlike the Mills cases, inexplicable by other conditions, are not to

be accepted as necessarily fatal to the doctrine of a specially localized control of the intellectual faculties. It is always possible that vital points may have been omitted from the clinical record or from the notes of post-mortem examination in the history of a case reported by any observer, however competent or eminent, and it is not at all improbable in case the observations were made many years before the questions were raised which gave these omissions their vital importance.

If in the future it should be proven beyond cavil that instances of special mental default or derangement have followed upon lesion limited to the right prefrontal region, or that lesion similarly involving the left side has been unattended by mental disorder, such facts would still be consistent with the localization in the left lobe of a centre of control for mental operations. It will be remembered that cases of hemiplegia have been reported in which the lesion was of the same side, and that in each an explanation was found in the anomalous absence of decussating fibres in the medulla oblongata. Such an anomalous condition is by no means infrequent. Trousseau's case of left hemiplegia with aphasia, upon the strength of which he denied the truth of Broca's discovery, may have been of the same character; though, in the absence of necropsy, the existence of independent lesions in the left frontal and right parietal lobes is an equal possibility. Many years ago twenty-six instances of absence of the corpus callosum had been collated. Functional anomalies are also of more or less frequent occurrence. Control of the speech faculty resident in the third right frontal convolution of right-handed persons has been several times reported.

Barton's,¹ Bromwell's,² and Collier's³ cases are all comparatively recent. Luy's case, in which an aphasic patient had begun to reacquire the faculty of speech, and in which the right frontal lobe was found disproportionately developed, is of the same nature. It is conceivable that in any case an apparently contradictory pathological relation may be reconciled to an already established general law by the discovery of structural or functional anomalies. Certainly, no case in which mental disorder might be found to occur in connection with exclusive right prefrontal lesion should be given weight until after an exhaustive search for other pathological alterations has been supplemented by equally careful examination of anatomical structure.

The question finally arises as to the import of a case in which symptoms may be supposed to have been carefully observed and post-mortem examination, both of lesions and of anatomical structure, to have been minutely made, and in which mental default or derangement had been found to have had no other physical basis than right prefrontal lesion. In such case would it necessarily follow that the localization of the

¹ British Medical Journal, July 25, 1877.

² Brain, 1898, p. 343.

³ Lancet, March 28, 1899.

mental faculties in the left prefrontal lobe is erroneous? The answer lies, in part, in a reference to certain facts and considerations connected with the practically unquestioned localization of the faculty of articulate speech.

1. The faculty of articulate speech is itself an intellectual faculty. This proposition is based upon the fact that aphasia is a mental and not a motor default. In aphemia or agraphia the muscles involved are neither paralyzed nor paretic, and their power of co-ordination is retained except for movements of articulation alone. It is the loss of a single memory—of one out of many memories. Objects are remembered in all their relations, but the muscular movements necessary for their symbolic expression are forgotten.

2. The intellectual faculties collectively will hold no closer relation to their physical basis than does any one of them individually. "Reason, judgment, and memory are the mental endowments chiefly concerned in the manifestations of intelligence" (Dalton). The measure of proof required for the localization of reason and judgment should not be different from that for memory, nor for memory, in general, different from that required for the one of its specializations which is essential to the function of articulate speech.

3. The localization of a centre of control for articulate speech in the third left frontal convolution is no longer questioned, yet in essential instances lesion of that part in right-handed persons has failed to cause aphasia, as in the cases of Barton, Bramwell, and Collier, to which reference has been made. Again, in left-handed persons this centre is normally located on the right side. It is also true that in destructive lesion of the speech centre on the left side speech may not be altogether lost, or, as in Luy's case, may be in some degree resumed. In all these cases functional power normally resident in the left side of the brain has been assumed by the right side, either as a congenital functional anomaly or as a compensating phenomenon.

Ferrier has pointed out that a compensating interchange of function by different centres is anatomically impossible, and, further, that motor activity exists after destruction of cortical motor centres only in so far as it is primarily or secondarily automatic and organized in the lowest centres; but he recognized the fact that the sound hemisphere after destruction of its fellow "is capable of influencing in greater or lesser degree movements of the limbs and other parts on both sides of the body." Hughlings Jackson believes that "every centre represents all parts, but each centre represents some one very specially," and this should apply to the higher as well as to the middle

¹ Functions of the Brain, p. 367.

² Evolution and Dissolution of the Nervous System.

centres. It is, perhaps, a not altogether unreasonable assumption that in case of the mind centres, which have no corresponding lower or more automatic centres, the intellectual activities specially represented in one frontal lobe should be more largely represented in the opposite side than are voluntary movements. Whatever importance may be attached to such surmises, the essential fact remains that the control of articulate speech may in certain instances reside in the right instead of the left frontal lobe.

If, then, the localization of all the intellectual faculties is to be governed by the same laws; and one of these faculties—that of articulate speech—concededly localized in the left frontal lobe, may exceptionally be controlled by a centre in the right lobe; it will follow that exceptional instances of mental default occasioned by destructive lesion of the right lobe will not militate against the evidence of control of the intellectual faculties by a centre or centres in the left prefrontal lobe afforded by the almost if not quite invariable production of intellectual disorder or default by lesion of that region, and of that region alone.

It is somewhat remarkable that the same considerations which have led to a more or less general belief that centres controlling psychical manifestations reside in the anterior regions of the brain should not have led to their further localization on its left side. Bouillaud was astonished that his predecessors had not made the "easy discovery," as he termed it, that the centre of speech was located anteriorly in the brain; yet he himself equally failed to discover that his own observations demonstrated that it was also limited to the left side. So many recent cases have been recorded which show a direct dependence of well-marked mental disorder upon left frontal lesion, and so many other cases in which, with extensive right frontal lesion, the mental faculties had been unimpaired, that it would seem at least a suspicion of the truth should have been aroused. It must be admitted, however, that a complete demonstration of the existence of centres controlling mental functions and their localization in a limited cerebral area is essentially difficult. The insufficiency and uncertainty of functional analysis, which Broca declared to be the greatest obstacle to the settlement of disputed questions in cerebral localization, have for many faculties ceased to be factors in the problem; but they still remain the chief difficulty encountered in establishing the relation between the mental faculties and a physical basis. Paralysis or convulsive movements, whenever the result of pathic processes or experimentally produced, and losses of special sense, can be estimated at their exact value; the loss of muscular power or of sight or hearing are comparable with an unvarying standard of normal function, and their appreciation is unaffected by the subjectivity of the observer. In some cases of mental disorder the characteristic conditions are so emphasized that their

significance cannot be mistaken; many such have been detailed. In other instances, far more numerous, the mental condition is dominated by a general cerebral lesion, and all localizing symptoms are lost in delirium or coma. In other cases, more numerous still, in which less evident though positive derangements of the complicated mechanism of the mind exist, not only the want of general standards of comparison, but ignorance of the mental idiosyncrasies and limitations of the patient, and often the very brief opportunity afforded for the study of psychical disturbances before the intercurrent of delirium or access of final coma, render conclusions uncertain. The temperament and methods of the observer himself, also, will be an unknown quantity to be reckoned with. In an estimate of mental integrity or impairment, where nice distinctions are to be made, perhaps no two observers will hold to quite the same opinions. The varying extent and character of their clinical training, their different habits of observation, variations in their general acuteness of perception, justness of inference, and accuracy of statement, their comparative familiarity with the study of psychical disturbances—all lead to more or less divergent views of the same case.

In the great majority of instances, therefore—those in which frontal lesion is trivial or even of moderate extent, and in which psychical symptoms are not clearly distinctive—it is only exceptionally that the record of a case is such as to afford intrinsic evidence that the mere statement that mental disorder did or did not occur justifies implicit confidence.

The pathological demonstration of mind-controlling centres must rest upon the evidence afforded by an examination of that smaller group of cases in which unmistakable mental derangement or decadence co-exists with well-defined structural lesion essentially confined to a limited cerebral area. This group, while small in comparison with the whole number of cases which, in the series analyzed, were found on post-mortem examination to have involved the frontal lesion, still comprises a very considerable number—a number much larger than that upon which the localization of a speech centre is founded. Further, the number of cases which have been found to be apparently exceptional to the law which it is sought to establish is both relatively and absolutely smaller than in case of that accepted for the speech centre.

No clinical observation has yet been recorded which suggests the specialization of control of any individual faculty of the mind, except that of articulate speech, in an individual convolution, and there is nothing to indicate a probability of any such localization in the future. These faculties are inseparably associated and interdependent, and in the absence of isolated action independent control would seem to be impossible.

The results of experiments which have been instituted by physiologists to determine the function of the frontal lobes have been contradictory or at least variously interpreted. The motor centres in the posterior frontal region, without doubt, have been well established; but no unanimity of opinion obtains among physiologists as to what centres, if any, reside in the prefrontal region.

The physiologists who have investigated this subject may be classified for the present purpose, without regard to other differences of opinion, as those who do, and those who do not, believe that their experiments upon the lower animals have shown mental decadence to have resulted from destruction of the prefrontal lobes. The first class includes Hitzig, Horsley, Schaffer, Ferrier, Bianchi, and Wundt; the second, Munk, Luciani, Meynert, Goltz, and Moolman.

Within these classes opinions widely differ. Ferrier does not admit that the mental deterioration indicates the existence of special centres of intelligence, and regards it as essentially a defect of the faculty of attention dependent upon frontal centres which control movements of the head and eyes. Bianchi attacks the views of Ferrier, and regards the prefrontal lobe as "the centre for the highest psychical functions." Munk and Luciani believe this lobe to be the controlling centre for the dorsal muscles. Goltz agrees with Ferrier in denying the specialization of centres of intelligence; and Hitzig, like Bianchi, affirms their existence. No two observers are exactly in accord. Such diverse interpretations of what must be essentially the same post-operative phenomena are explicable in great part by the difficulty experienced in estimating changes in the exercise of the imperfectly developed mental faculties of the lower animals; and in part, perhaps, in distinguishing from each other movements controlled by higher or lower centres. The weight of authority undoubtedly supports the contention that destruction of the prefrontal lobes is followed by mental decadence.

The mutual dependence and complementary value of pathological observation and of physiological experiment have been generally recognized. The precise delimitation by the physiologist of cerebral areas of control for muscular movement and for the special senses was first suggested by the observation of pathic conditions, and was verified later for the human subject by a recurrence to the methods of the pathologist. In the matter of localizing the control of the mental faculties, physiology has borne a comparatively unimportant part. The discovery of a speech centre, since animals do not talk, was necessarily independent of experiment. The limitations of this means of investigation for localizing the other mental faculties were indicated when referring to some of the causes of a divergence of physiological opinion. The results of physiological experiment, though not always recognized as the same or interpreted in the same way, may be regarded, however,

as corroborative of the fact substantiated by clinico-pathological observation, that mental decadence follows destruction of the prefrontal region. No experiments have been undertaken designed to show whether this deterioration would be equally manifest after the destruction or ablation of either frontal lobe while the integrity of the other was preserved. Such an experiment was incidentally made by Bianchi in one instance in which the lobes were successively removed; but, as has similarly happened when one lobe in the human subject has been destroyed by accident or disease, the obvious inference was disregarded. In one of four cases detailed by him, that of a young female cynocephalus,¹ the right frontal lobe was excised some months before the excision of the left. The primary operation was done August 2d, and the right lobe excised through a plane 2 or 3 millimetres in front of the excitable area for the arm, face, and jaws. One week later recovery had taken place, and her condition was in all respects normal, "save that she was subject to fear and had left external hæmianopsia."

September 11th. She was "still alert, agile, and observant," and no mental change occurred afterward except that she became more irritable.

January 31st. Six months later the left lobe was excised through a plane immediately in front of the face centre, as determined by electrical reactions.

February 2d. "When called she raises her hand slowly, opens her eyes, looks about, and relapses into the same condition. When touched she does not try to escape or look around."

9th. She is "duller and heavier. Intellect: Attitudes stupid, loss of physiognomical expression; apparently aimless walking; she ran incoherently from one end of the room to the other; pieces of plaster offered to her are taken and eaten as if they were sugar. In all her actions there seems to be a defect of perception, which seems to be reduced to a mere elementary level, and lacks some of the factors necessary to the formation of a more complete judgment. Her psychical life seems to be reduced to the existence of actual sensations."

May 4th. Animal killed by chloroform.

Bianchi has elsewhere declared: "No perceptible differences were noted in the behavior or psychical manifestations of animals mutilated on one side only."

It would be of interest to know if in any case it was the left side which was alone removed. If he found no psychical deterioration to follow left unilateral mutilation it would probably indicate that functional compensation was more immediate and more perfect in the right lobe than in the case of the human subject. It would be impossible to

¹ Brain, vol. xviii. p. 511.

admit that any negative experimental result could outweigh the positive evidence afforded by the study of human pathology. Conditions are morphologically and psychologically different. Muscular contraction is a physiological function, definitely circumscribed, common to man and to all animals, and known to be governed by the same recognized laws. The development of the mental faculties in the inferior animals, on the contrary, is very limited, and knowledge of the conditions which control their exercise is more limited still.

It is unnecessary for the present purpose to enter upon a discussion of theories which seem to have been disproved by pathological facts. The speculative views of Ferrier, however, which form a basis of belief for all those who deny the existence of special centres of control for the mental faculties carry too great a weight of authority to be ignored. It may be recalled, in brief, that he regarded the sensory and motor centres of the whole cerebrum as the "substrata of sensory perception and ideation, and of acts of volition as well as feelings associated with their activity." He concluded, as the result of experiment, that the prefrontal lobe is the motor centre of the eyes and head on the opposite side, and, in consequence of the close relation between the movements of these parts and attention, that it is also the centre for that faculty. He declared that it would be "absurd to speak of a special seat of intellect or intelligence in the brain;" and that "the control of ideation and the power of attention form the basis of all those intellectual achievements not included in mere receptiveness, ideational or emotional mobility, and the facility of executing delicate and complex motor acts."¹

It is to be objected to the theory of Ferrier not only that it is not founded on pathological observation, but that it affords no explanation of the origin or control of abstract conceptions, moral sentiments, or congenital peculiarities of character. It may be difficult to disassociate mental operations from external impressions; but certainly obstinacy, selfishness, and generosity, or other qualities which are innate, and reasoning processes which involve purely spiritual conceptions, cannot be evolved from the perception of external objects or associated with muscular movements. In like manner, comparison, judgment, or the appreciation of moral qualities is as far from a mere conscious sensation as it is from an automatic movement.

Ferrier's views have been modified by some writers, and by others have been rejected altogether. Bianchi² not only holds that "the prefrontal lobe is the centre for the highest psychical functions," but that "there exists no centre the function of which is to inhibit, and that there exists no faculty of attention." Boris Sidis,³ in a discussion of

¹ Functions of the Brain, 1886, pp. 460-467.

² Psychology of Suggestion, New York, 1899, pp. 124-128.

³ Brain, vol. xviii, p. 497.

hypnotic amnesia, rejects entirely the theory of retention and reproduction of images by modifications of nerve elements as the essentials of memory.

Ferrier expressed the opinion that it was not necessary to assume higher centres in this region in order to explain the facts either of normal or abnormal mentation. In the progress of events clinical observers have learned that this necessity exists; and the present review of a multitude of clinical cases shows, further, that it is necessary to assume their localization in the left prefrontal lobe, to the exclusion of the right.

"One clear case," to quote again from Ferrier,¹ "in which destruction of a region had caused no cessation or disorder of a function would be sufficient to overcome our conclusion," which in this case is, as above stated, that the exclusive control of mind is resident in the left prefrontal lobe. Not only has no such clear case been found, but none in which limited lesion of the right prefrontal lobe or any other cerebral area has produced symptoms of mental disorder.

It is immaterial, so far as it concerns a control of the mental faculties in the prefrontal region, whether the function of the left prefrontal lobe is one of co-ordination or one of inhibition, or whether it is a direct control of mental, moral, and emotional phenomena comparable to the control of motility in the motor areas, as asserted by Bianchi, or whether it is a control of sensorimotor processes, which are the anatomical substrata of mentation, as insisted by Hughlings Jackson. It is simply a question of topography, entirely independent of psychological considerations. It has no concern either with Tyndall's "unthinkable passage from the physics of the brain to the corresponding facts of consciousness," with Lewes' assertion that "the neural process and the feeling are one and the same process viewed under different aspects," or with Jackson's parallelism of mental and subjacent physical states. The fact that mentation has an anatomical substratum having been universally accepted as an almost axiomatic truth, there remained for the determination of the neural physiologist and the pathologist the more or less precise delimitation or the general diffusion in the cerebrum of this physical basis.

Three propositions are justified by the cases which have been presented:

1. The more absolutely the lesion is limited to the left prefrontal lobe the more positive and distinctive are the symptoms of mental default.
2. The integrity of the mental faculties remains unimpaired in right

¹ Localization of Cerebral Disease, 1878, p. 3.

² See Hughlings Jackson on the "Localization of Movements in the Brain." London. Reprint from The Lancet, 1878.

frontal lesion, though it involves the destruction of the entire lobe, or even extends to the entire hemisphere.

3. The exceptional instances in which seemingly opposite conditions exist are always reconcilable, on more careful examination, with the assertion of an exclusive control of the mental faculties residing in the prefrontal region of the left side.

If, then, the same nature and degree of proof which is deemed sufficient for the localization of other cerebral functions may be accepted in case of the mental faculties, their centre of control has been established.

THE UNILATERAL OCCURRENCE OF KERNIG'S SIGN AS A SYMPTOM OF FOCAL BRAIN DISEASE.

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I DESIRE to report two cases in which Kernig's sign was present only on one side and appeared to bear some reference to a cerebral lesion on the other side of the brain. This sign was described by Kernig in 1883 before the Medical Society of St. Petersburg, and the next year published in German. His attention was first directed to the phenomenon in a patient recovering from epidemic cerebro-spinal meningitis. This patient could walk perfectly well, could lie in bed with legs extended, but whenever she sat in a chair she found it impossible to extend the legs on the thigh beyond a right angle. Subsequently he studied fifteen cases of meningitis, nine of which were confirmed by autopsy, with reference to this sign, and found it present in all. It could be elicited whether the patient sat up, lay on the back or on the side. He describes it as a flexion contracture in the legs (and occasionally in the arms) when the thigh is flexed to a right angle upon the trunk. Under these circumstances any attempt to extend the leg on the thigh meets with severe resistance as a result of contraction of the hamstring muscles, and it is impossible to extend the leg beyond an angle of 135° , or even, in extreme cases, beyond a right angle. When the thigh is extended the hamstring tendons are relaxed and soft; when, however, the thigh is at a right angle to the trunk and an attempt is made to extend the leg they become tense and prominent. The contraction is not ordinarily associated with pain, nor with any increased rigidity in any other part of the body, and Kernig particularly noted that the retraction of the head did not become greater when the patient sat up. He states that the sign usually persists long into convalescence; it may vary from time to time in the course of the disease; it is not produced by mechanical irritation of the sciatic nerves, and may, as he noted in his original

communication, occur in certain other conditions, although in all of those that he observed there was reason to believe that irritation of the membranes existed.

The literature on the subject is very inconsiderable, although the presence or absence of the sign is now usually mentioned in connection with a suspected case of meningitis. The first important article on this subject was that of Friis, who found it present in 74 of 86 cases, and was not able to exclude it positively in all of the other 12. Henoch also obtained it frequently in children suffering from meningitis, and Blumm mentions its existence in 7 of 9 cases. In one case the contraction of the muscle came on suddenly and was painful. Bull obtained it in all positions in 2 cases: one a tumor of the cerebellum and the other a thrombosis of the left lateral sinus. Netter, in an extensive series of investigations which practically called general attention to the existence of the sign, found it present in nearly all cases of meningitis. The attention of the American profession was directed to this subject by a paper by Herrick, read before the American Medical Association. He found it present in 17 of 19 cases of meningitis and only twice in 100 other cases, one of which was a case of subdural hemorrhage, and one a case of gonorrhœal rheumatism, as a result of which the patient had lain with the knees flexed for a period of four weeks. He failed to observe it in cerebral hemorrhage, brain tumor, and other intracranial conditions. In other cases the absence of the sign was of great value in excluding meningitis. He called renewed attention to the fact that the sign can be elicited if the patient either sits or lies down. Its presence has also been reported by Widal and Merklen in a case in which the lesion was found to be a clot pressing upon the anterior surface of the pons and medulla, and by Thyne in a case in which there was hemorrhage into the left lateral lobe of the cerebellum, and an effusion of blood over the right occipital lobe and into the fourth ventricle. The patient also had retraction of the head, but meningitis was not present. The sign is more often absent in tuberculous than in any other form of meningitis. Henoch was the first to mention this fact. Netter, Herrick, and Dieulafoy have all confirmed it, and Packard has reported 3 cases of tuberculous meningitis in children aged fifteen, sixteen, and four months, in which the sign was persistently absent. With the exception of one of Herrick's cases, in which the sign was elicited in the unaffected leg of a woman suffering with gonorrhœal gonitis, mention has not been made of its unilateral occurrence.

During the past year I have had under my care in the wards of the Philadelphia Hospital, two cases in which the sign was unilateral and appeared to be a symptom of focal encephalitis. The cases are intrinsically interesting, but possess no common points except this peculiar phenomenon.

CASE I.—The patient, W. H. P., a white man, aged twenty years, laborer by occupation, was admitted to the Philadelphia Hospital on September 16, 1901. He was partially delirious, but could answer simple questions. He complained of severe headache and pains all over the body. The temperature on admission was 104° ; there was slight abdominal tenderness and an occasional dry cough. His family history was indefinite; his mother had died of Bright's disease; one sister was dead; his remaining relatives were living and well. His previous history was obtained from his father, who stated that he had had whooping-cough, chicken-pox, measles, and malaria in childhood.

In 1888 he had a moderately severe attack of typhoid fever, and at Christmas, 1899, an attack of what was diagnosed as spotted fever, which lasted for about three weeks. This came on suddenly, and it was necessary to bring him home in a patrol wagon. There was a doubtful history of an attack of appendicitis about a year ago, for which no operation was performed. For four or five weeks before his present illness he had been working on an ice-wagon. The history of his present condition was as follows: For about two weeks he had been complaining of pain in the side, backache, and headache. On September 14th he came home from work complaining of intense headache, and soon became delirious; he was not able to sleep, and on the 15th became violent. He had a serous diarrhoea, with watery, yellowish stools, and seemed to be very feverish. Upon admission the following notes were made: A well-developed, well-nourished male; numerous tattoo marks on both arms; pupils equal, react to light and accommodation; tongue heavily coated, protruded in a straight line without tremor. Pulse was strong, regular, of good volume and good strength; chest was well formed, expanded equally and well. The abdomen was negative, with the exception of slight tenderness in the right iliac region. No nervous symptoms were noted on this day. On the following day, however, when I first saw the patient, he was acutely delirious. The head was slightly retracted, although the muscles of the neck were not hard. The pulse was of medium volume. The left arm was strongly flexed and distinctly rigid. The right arm was normal. The lungs were normal; the cardiac dulness commenced at the third interspace and extended laterally from the mid-sternum to the left parasternal line; there was a weak, diffuse apex-beat in the fourth interspace. Auscultation revealed gallop-rhythm, and a slightly accentuated pulmonic second sound. The liver extended from the sixth rib by the costal border; the spleen was not palpable, but was slightly enlarged to percussion. The stomach, examined by auscultatory percussion, did not extend below the costal border; there was no eruption on the skin; the inguinal and post-cervical glands were small but palpable. There was slight hyperæsthesia over the abdomen. The supra-orbital nerves and the suprabulbar fossæ were not tender, and the patient did not manifest any discomfort upon strong percussion of the skull. The reflexes were not exaggerated. Kernig's sign was readily elicited in the left leg. It was possible to extend the right leg in an almost straight line with the thigh when the latter was flexed at right angles upon the abdomen. Posteriorly nothing abnormal was noted aside from a slight increase in vocal resonance at the base of the left lung, and numerous subcrepitant râles at both bases. A provisional diagnosis was made of tubercular meningitis, associated with hypostatic conges-

tion of the base of the left lung, and with acute nephritis. On September 18th the patient was distinctly worse; he had been delirious all night, and slept only for short intervals; he complained of pain in the precordial region; there were still signs of hypostatic congestion of the lower lobe of the left lung posteriorly. When roused there was distinct lagging of the left upper eyelid. The mouth was drawn toward the right side, and the right side of the face was wrinkled, while the left side was smooth. The reflexes were not changed. The following day the head was turned to the left side; the left pupil was smaller than the right and seemed sluggish. The tongue was dry and rough and protruded to the left side. There was marked flexor spasm in the left arm, which also seemed to be paretic, at any rate the patient did not use it to remove irritants. The right arm was moved freely and appeared to be normal. The knee-jerks were slightly increased, especially on the left side, and Kernig's sign was distinctly present in the left leg, which in addition was slightly paretic. The patient now had incontinence of urine. On September 20th the condition was about the same; the head was turned strongly to the left and could not be forcibly turned in the other direction. The left arm was firmly flexed at the elbow, and resisted straightening. The right leg was not moved voluntarily; in the left leg occasional irregular movements were noted. Kernig's and Babinski's signs were both present on the left side but not on the right. The apex-beat was in the fourth interspace, in the nipple line. A systolic murmur was heard on this day for the first time, most distinctly at the apex, and there was moderate accentuation of the second pulmonic sound at the base. At midnight of this day the patient had a hemorrhagic eruption over the entire body, consisting of very slightly raised, small round spots that did not disappear upon pressure. There were also hemorrhages under the conjunctivæ, and in the mucous membranes of the lips and mouth. The patient could not be roused, and it is possible that he was deaf on both sides. The head was retracted and rotated to the left; the right pupil was widely dilated and reacted promptly to light; the left was contracted and reacted very slowly; there was chronic drooping of the left eyelid. Respiration was of the typical Cheyne-Stokes variety. The pulse showed a very remarkable alteration, it was full, rapid, and receded quickly—a typical water-hammer pulse. Upon auscultation of the heart a loud systolic murmur could be heard at the apex and at the pulmonic cartilage; there was also accentuation of the second pulmonic sound. The left arm was flexed over the chest and could not be extended by force; the right foot was not moved even when the sole was vigorously irritated. There was marked ankle clonus on both sides. The knee-jerks were increased, more on the left side. Kernig's sign and the Babinski phenomenon were both present only on the left side. The patient was entirely unconscious and only groaned occasionally. On September 21st he grew gradually weaker, the pulse becoming smaller; respirations, however, were slightly more regular; the reflexes remained increased until death, which occurred at 6.40 A.M.

During the course of the case the urine was examined on the 17th and found to contain albumin and numerous granular and blood casts. The specific gravity was 10.14. On the 17th the leucocytes were 17,600; on the 19th, 27,000. The Diazo reaction was tested on the 18th and was not present. Blood was taken for the Widal reaction

on the 18th, and the report returned from the City Laboratory on the 20th that it was not present. On the 20th, Dr. Pfahler made a spinal puncture and also withdrew some blood from the median basilic vein of the right arm, under strict aseptic precautions. Agar tubes were inoculated with this material, and on all of them cultures of a staphylococcus that became golden-yellow appeared. These cultures were pure. On the 21st the autopsy was made by Professor Coplin, who has kindly permitted me to abstract his notes. Rigor mortis was unusually pronounced; the pupils unequally dilated, the left more than the right. The whole body was covered by a petechial eruption which involved all the visible mucous membranes. Upon opening the body hemorrhages were found in all the serous membranes. The right side of the heart was slightly dilated; the tricuspid, pulmonic, and mitral valves were normal. The anterior and left aortic leaflets were partially destroyed, and the edges showed recent inflammatory vegetations. Upon the remaining leaflet only the ventricular surface was involved, and it was slightly rough and contained a few small vegetations. The left lung contained an infarct upon the lower edge of the upper lobe; the lower lobe showed intense hypostatic congestion. The spleen was enlarged and soft; the kidneys showed cloudy swelling; the meninges were congested, and there was a softened area in the motor region on the right side. The right tibia was slightly deformed, and upon incision was found to be the seat of an old osteomyelitis, the marrow having been largely converted into compact bone, and the compact substance being unusually dense.

The autopsy, therefore, confirmed the final diagnosis as far as the heart, kidneys, and lung were concerned. We had supposed during life that a meningitis existed, and as a matter of fact the membranes were intensely congested and cedematous, and over a small area of the ascending frontal gyrus at the level of the second frontal convolution they were slightly altered. The main lesion in the brain, as subsequent examination showed, consisted of an area of hemorrhagic softening in the middle of the ascending parietal convolution, and extending inward through the centrum ovale toward the internal capsule, which, however, it did not involve. Surrounding this there was an area a few millimetres wide, of partial discoloration of the brain, apparently due to oedema. At the autopsy Dr. Coplin permitted me to make cultures from the brain and gall-bladder, and from both an organism was obtained which was similar in all respects to the one obtained by Dr. Pfahler in his cultures from the blood and cerebro-spinal fluid. This organism has twice been injected into rabbits, once intravenously, without producing fatal effects. Dr. Coplin also made cultures from the heart blood and spleen, and obtained a coccus culturally and morphologically identical with the staphylococcus pyogenes aureus. Sections through the brain showed that the lesion consisted of an area containing considerable hemorrhagic extravasation that was surrounded by a layer of polynuclear cells, in the midst of which there were numerous masses of cocci. The surrounding brain tissue showed swelling of the ganglion cells and thickening of the glia. The case, therefore, is a rather interesting example of acute malignant endocarditis, due to staphylococcus infection, and associated with a focal encephalitis. In view of the existence of a chronic osteomyelitis of the right tibia it is perhaps permissible to suspect, in the absence of

any other focal lesion, that possibly the organism remained latent in this situation, and then, for some reason, invaded the body, producing the endocarditis and subsequently the various pyæmic manifestations. Unfortunately cultures were not made from the bone lesion.

CASE II.—J. P., white, aged thirty-seven years, was admitted to the hospital on November 16, 1901, complaining of pain in the back and partial loss of power in the left side of the body. The patient is an intelligent man who had been healthy until a year ago, when he occasionally had attacks which were diagnosed as rheumatism, and a fistula in ano. He has used alcohol to excess; he denies venereal history, and is the father of four healthy children. Three weeks before admission to the hospital the patient, who at that time was in good health, went to bed with a feeling of general malaise and pain in the back and limbs. On the following morning he discovered that he was unable to use the left side of the body; speech was thick; he still had pain in the back and also in the left arm and leg. Physical examination on December 7th showed slight inequality of the pupils, the right being the larger, although both reacted to light and accommodation. The tongue was protruded in a straight line; the supra-orbital reflex was equal on both sides; there seemed to be slight impairment of motion on the left side of the mouth. Speech was defective, the utterance was thick, and there appeared to be special difficulty in pronouncing the letter "R." This, the patient says, has developed since his present sickness. The platysma moved freely on both sides and there was slight rigidity of the muscles of the neck on the left side posteriorly. There was permanent spasticity of the muscles of the right arm, and of all the muscles controlling the left wrist-joint; although the flexors were more involved, and the arm was therefore held completely flexed with the wrist bent at right angles. There was no rigidity of the fingers of the left hand, excepting some spasticity of the long extensors of the thumb. The muscles appeared to be slightly wasted; the grip was weak; the movements of the arm were weak, and the patient was unable to extend it beyond a right angle, although it could readily be extended by the application of force. The biceps jerk was diminished; the triceps jerk was slightly exaggerated, but the muscles did not show mechanical irritability. There were no sensory disturbances in the arm, and all the muscles of the arm and shoulder responded readily to faradic electricity. Aside from the hampering effect of the spasticity, motion was normal, and there was no evidence of inco-ordination. The stereognostic sense was normal. Whenever an effort was made to move the left arm the patient moved the right arm with it in a somewhat similar manner, and this associated movement could not be controlled voluntarily. The right arm was entirely flaccid; movements were normal and were not accompanied by movements of the left hand. The little and ring fingers were persistently flexed on the palm, and there was wasting of the thenar and hyperthenar and interossei muscles. This was evidently due to an injury to the ulnar and median nerves received fifteen years ago, the scar of which was still distinct. The thumb, index and middle fingers were capable of free movement, and the grip was fairly strong. There was slight numbness of sensation of the ulnar side of the hand. The arm reflexes were normal; sensation was entirely normal. The left leg was distinctly spastic; it was held habitually in a position of extreme extension. The knee-jerk was greatly exaggerated; there

was persistent patellar clonus, and a rapidly exhausted ankle clonus. Babinski's phenomenon was present to an extreme degree; the plantar reflex was normal. The movements of the leg were executed slowly and clumsily, but without any evidence of inco-ordination. The toes did not resist passive movement in any direction. Sensation was everywhere normal; localization was good, and the muscle sense was not destroyed. When the thigh was flexed upon the abdomen the leg could not be extended beyond an angle of 90° ; if the patient sat on the edge of the bed it was impossible to extend the left leg beyond a perpendicular position. Whenever this sign was tested the hamstring muscles, which were soft and relaxed when the leg was extended, became hard and rigid. The spasm was not associated with any pain. Kernig's sign had been tried daily since it was first elicited, and the angle through which the leg could be moved diminished from an estimated 115° on November 22d, to between 90° and 95° on December 6th. The right leg was flaccid; the knee-jerk was exaggerated; patellar clonus could not be elicited, and there was no ankle clonus. The Babinski reflex was distinct and characteristic; there was no impairment of co-ordination and no disturbance of sensation. The muscles of both legs responded readily to faradic electricity. The muscles of the ulnar side of the right hand failed to respond. The abdominal reflex was present and apparently normal. The patient states that his memory is not as good as it formerly was; he has occasional emotional attacks apparently due to a realization of the serious nature of his disease, but is otherwise cheerful and exceptionally intelligent. He has complained of diplopia ever since his admission, and an examination by Dr. de Schweinitz on December 4th showed that the vision in each eye was reduced to 5/15, and there was a paresis of 8 prism degrees of the left external rectus. The eye-grounds were normal, and there were no changes in the form fields.

On December 20th, the condition of the patient had improved considerably. The diplopia was rarely present excepting at night; the rigidity of the left arm persisted; both knee-jerks were moderately exaggerated, and there was slight spasticity of the left leg. Kernig's sign had completely disappeared, and in spite of the spasticity it was possible to extend the left leg completely. The patient was able to walk with assistance; speech showed distinct improvement, but he was still very emotional.

The nature of this case is somewhat obscure. The lesion is evidently located in the upper portion of the medulla on the right side, and extends far enough forward to involve the nucleus of the left abducens. It cannot be complete because the paralysis is not complete on the left side, and it must be limited because there is no disturbance of sensation. Tumor was at first suspected, and the patient stated that at the commencement of his attack he had headache and vomiting; this, however, soon disappeared, and the absence of choked disks rendered the diagnosis of tumor unlikely. There is no reason to suspect embolism of one of the branches of the basilar artery, but thrombosis of this artery or syphilitic disease of the artery causing partial degeneration in the surrounding tissue cannot be excluded. The sudden onset of the attack leads me to suspect the possibility of a non-suppurative focal encephalitis of the type described particularly by Nonne.

The patient has received potassium iodide in increasing doses since admission. There is, however, no other reason to suspect a syphilitic

lesion, for the patient has no recognizable signs of syphilis. The most interesting feature is the presence of Kernig's sign while the symptoms were severe, and its disappearance as improvement progressed.

The nature of the mechanism by which Kernig's sign is produced is still obscure. Kernig himself did not attempt any explanation, but was content to prove that it was not due merely to increased intracerebral pressure, nor to pressure upon the sciatic nerves. Friis suggested that it was due to irritation of the *corda equina* by the infected cerebro-spinal fluid. Of course, the occurrence of the sign in purely cerebral lesions renders this view untenable. Henoch spoke of it as a reflex manifestation. I do not know exactly what he means by this, but there can be no doubt that the position of the thigh which stretches the flexor muscles evidently produces at a certain point a contraction in them. To call this a reflex does not explain the sign. Exactly the same view has been maintained by Bull, who called attention to the fact that it is difficult fully to extend the leg when the thigh is at right angles to the trunk, and regards the sign merely as an exaggeration of this normal condition. Netter and Herrick, who next to Kernig and Friis have made the most extensive investigations upon this subject, merely discuss the theories that have been suggested by others. Widal and Merklen look upon it as a manifestation of irritation of the spinal meninges, but do not attempt to explain how this irritation produces the very peculiar phenomenon. Chauffard, the most recent author to discuss its nature, explains it as an exaggeration of normal phenomena, due to a hypertonicity of the muscles. It is closely analogous—according to him—to the retraction of the muscles of the neck and back, and he therefore defines it as a contraction mono-regional or multi-regional affecting physiologically predominant groups of muscles, and occurring in attitudes which normally bring these muscles into play.

In the case that I report the common feature was the spastic paresis of one side of the body due to unilateral cerebral lesion. In both cases this spasticity did not produce any retraction of the hamstring muscles when the leg was extended, but gave rise to all the characteristic phenomena of Kernig's sign when the thigh was flexed upon the trunk. In the majority of other cases in which this sign occurred there is reason to suppose a certain degree of irritation of the pyramidal tract associated with depression of its conductile power, that is to say, a condition similar to the one that existed in both of these cases, a spastic paresis. And it seems not unreasonable to suppose that more extensive and careful observations will show that in the majority of cases Kernig's sign may be regarded as one of the symptoms of a partial lesion of the pyramidal tract, that is to say, a lesion which does not destroy it but which prevents its complete functional activity. This does not serve to explain its mechanism, unless we assume with Bull and Henoch that

it is merely an exaggeration of normal conditions; but to assert this positively is of course at present unwarranted. Its mechanism is still as uncertain as is the mechanism of Babinski's sign, which in some respects is analogous to it, but apparently is present when the lesion of the pyramidal tract is much more severe than is compatible with the existence of Kernig's sign. Kernig is the only author who states that the arms may be affected by similar condition. In the two cases which I report there was a flexor spasm in the arms on the affected side, but in both cases this could be overcome by force, was persistent, and bore no definite resemblance to the manifestations in the leg.

It is of course obvious that it is not a lesion of the meninges but of the subjacent nervous substance that causes the occurrence of Kernig's sign. Practically all cases of leptomeningitis are associated with a more or less severe degree of encephalitis. As far as I know, however, the occurrence of the sign in the course of a typical case of encephalitis has not been recorded. In Nonne's series of cases no mention whatever is made of it, although he distinctly states that in one or two cases symptoms of meningitis were not present. Whether this includes the sign or not is of course impossible to determine. It is impossible to say whether the sign is frequently present in encephalitic lesions involving the pyramidal tract or not; that it may occur in such cases, Case I. sufficiently proves, and Case II. supports the affirmative view, although, of course, the diagnosis has not been confirmed by autopsy. At any rate, I think the following conclusions are justified: First, Kernig's sign may occur as a symptom of focal encephalitis, and in this condition may be present upon only the opposite side of the body. Sometimes it is associated with spastic paresis of the leg upon that side. Second, in these cases there may be a persistent tonic spasm of the flexor muscles of the arm, which, however, does not resemble Kernig's sign in its mechanism. Third, the most reasonable explanation of Kernig's sign that we have at present is to ascribe it to an irritative lesion of the pyramidal tract that diminishes but does not destroy its functional activity.

At my request, my resident physician, Dr. Shields, has made careful studies of 100 cases at the Philadelphia Hospital, with reference to the presence of Kernig's sign. The results of these studies are appended to this article.

In addition, Dr. Clark has, at my request, reported three cases of meningitis, all confirmed by autopsy, in which the sign was absent during the entire period of observation.

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REPORT OF ONE HUNDRED CASES, ALL NON-MENINGITIC, EXAMINED FOR KERNIG'S SIGN.

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THIS report is to show the result of the examination of 100 non-meningitic cases, both febrile and afebrile, for the presence of Kernig's sign. In every case both legs were examined for the sign in both erect and recumbent postures, excepting when the condition of the patient would not admit his sitting up. In 5 cases Kernig's sign was present; 3 showed the sign unilaterally, and 2 bilaterally, 1 case of ur  mia and 1 case of typhoid fever, and it is interesting to note that in both of these cases the sign could not be obtained after recovery. Kernig's sign persisted in the 3 remaining cases—2 cases of right-sided hemiplegia, and 1 of typhoid fever—and in all it was obtained on only one side. The case of typhoid fever is still quite ill, and it is possible that the sign will have disappeared by the time the patient has regained her normal condition. In both cases of typhoid fever which showed the sign, delirium was marked and persistent. This predominance of mental symptoms may have been an indication of febrile or toxic cerebral irritability, which might have in some manner been accountable for the presence of the sign.

| Number of cases of each. | | | | Diagnosis. | Right leg. | Left leg. | Angle of extension. |
|--------------------------|----|---|---|--------------------|------------|-----------|---------------------|
| (Case No. 4) | 1 | . | . | Cirrhosis of liver | — | — | — |
| (Case No. 5) | 1 | . | . | Hemiplegia (right) | + | — | 115   |
| | 1 | . | . | Hemiplegia (right) | + | — | 120 |
| | 3 | . | . | Influenza | — | — | — |
| | 2 | . | . | Malaria | — | — | — |
| | 8 | . | . | Pneumonia | — | — | — |
| | 4 | . | . | Rheumatism | — | — | — |
| | 48 | . | . | Senile | — | — | — |
| | 25 | . | . | Tuberculosis | — | — | — |
| (Case No. 1) | 1 | . | . | Typhoid (B. E. P.) | — | + | 105 |
| (Case No. 2) | 1 | . | . | Typhoid (J. W.) | + | + | 110 |
| | 3 | . | . | Typhoid | — | — | — |
| (Case No. 3) | 1 | . | . | Ur  mia (J. C.) | + | + | 110 |
| | 1 | . | . | Ur  mia | — | — | — |
| 100 | | | | | | | |

The table shows the diagnoses of the cases and the number of each examined. The condition in each leg with regard to the presence or absence of Kernig's sign is noted, and in those cases in which the angle of extension was 120° or less this fact has been stated. Brief reports of the 5 positive cases are given.

CASE I.—C. E. P., girl, aged thirteen years, was admitted to the hospital with a history of having been ill for about five weeks. She suffered from malaise, headache, pain in the chest, abdomen and legs, and for about a week she had had epistaxis, and nausea and vomiting for about three days. There were constipation, cough, and expectoration of a mucopurulent material. Two other persons were ill with typhoid fever in the same house. The patient was pale, the pupils were large and reacted to light and distance. The tongue was dry and coated; there was marked hyperæsthesia on the cutaneous surface, especially the arms and legs; high temperature, and a roseolar eruption on the chest and abdomen. The spleen was enlarged; Kernig's sign was present on the left side, the angle of extension not exceeding 105° . The leucocyte count was 5600. The Widal reaction was positive. On the third day after admission a loud systolic murmur was heard at the apex, and the pulmonic second sound was accentuated. Two weeks later she developed otitis media, and about the same time suppuration of the axillary glands. The patient gradually improved, and at present is convalescent, but Kernig's sign still persists in the left leg, the greatest angle of extension being 105° . The spasm is not painful.

CASE II.—J. W., white man, aged thirty-six years, an iron-worker by occupation. Patient when first seen was treated for alcoholism. After his acute alcoholic symptoms with consequent gastritis had subsided, he continued to have elevated temperature. His blood was examined for the Widal reaction and a positive report returned. He had only three typical typhoid spots distributed over the abdomen. He had constant pain in the head and limbs. Constipation was present all through the attack. No enlargement of the spleen could be determined. The tongue was thickly coated with a yellowish-brown fur. Delirium was present in a marked degree for a considerable time. The subsequent course of symptoms, signs, and temperature proved it to be an undoubted case of typhoid fever. The patient showed Kernig's sign typically on both the right and left sides during the height of the disease while lying in bed. Later in the course of the disease, when the patient's condition warranted his being placed in the erect posture, the sign was obtained when he sat on the edge of the bed. When lying in bed the greatest angle of extension was 105° on each side, whereas, when he sat up ten days later the legs could be extended to an angle of 115° with the thigh. This man recovered completely from his enteric attack, and at the end of convalescence Kernig's sign was not present on either side.

CASE III.—J. C., aged sixty-one years, a bricklayer. On November 3, 1901, the patient was admitted suffering from uræmia. He was unconscious, he could not be aroused, the pulse was rather small and fairly rapid. The eyes were rotated upward and to the left, the pupils being somewhat contracted. No reaction to light could be ascertained. The skin was dry and hot. The body temperature was somewhat elevated;

the tongue was dry, brown, and hard; the urine, obtained by catheter (6 drachms), showed a large amount of albumin, with numerous hyaline and granular casts. He was treated with ordinary uræmic therapeutics and rapidly regained consciousness. In two weeks' time he had improved so much that he required no special treatment or attention. When first examined he showed Kernig's sign on the right and left sides, the greatest angle of extension being about 110° . On the eighth day of observation this sign could not be elicited on either side. This patient was only examined for the sign when he was lying in bed, the thigh being flexed upon the abdomen at an angle of 90° .

CASES IV. and V.—The two hemiplegics referred to were both cases in which the mentality was below par. They were both rather typical cases of right-sided paralysis, which condition had lasted in one case for over twenty years, and in the other for about fifteen years. The mode of onset as well as the subsequent course of the affection could not be ascertained. They were examined, both when sitting up and in a recumbent posture, and Kernig's sign was found present on the right side in each case. In one case the greatest angle of extension was 115° , and in the other 120° . In both cases when the thigh was not flexed the leg could be extended to an angle of 180° .

In concluding this report of 100 cases it is essential to state that in no positive case was there any joint involvement either osseous or tendinous; also that in all these cases the legs could be fully extended, and when the thigh was flexed the tendons became hard and tense. I have called no case positive in which the angle was more than 120° . The only case on this list in which the presence of Kernig's sign might have proved misleading by resembling meningitis was Case I., in which there were delirium, elevated temperature, rapid pulse, a red eruption over the chest and abdomen, with hyperæsthesia of the arms and legs. A leucocytic count of 5600, with positive Widal reaction, and two persons ill with typhoid fever in her home at the same time, made the diagnosis clear.

THREE CASES OF MENINGITIS IN WHICH KERNIG'S SIGN WAS PERSISTENTLY ABSENT.

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(From the service of Drs. Hughes and Sallinger, of the Philadelphia Hospital.)

CASE I.—D. C., Italian laborer, aged twenty-two years, had a severe chill associated with pain in the abdomen and headache. In a short time he became unconscious, and when admitted to the hospital one day after the onset, his eyes were open and staring, the pupils were dilated, the right slightly larger than the left. Both reacted to light and accommodation. There was some rigidity of the muscles of the neck which increased to marked retraction of the head as his symptoms progressed. Photophobia and hyperæsthesia were prominent

symptoms. There were herpes labialis, and sordes on the teeth, but no eruption on the skin. The knee-jerks were normal; Kernig's sign could not be elicited in either leg in the recumbent or erect postures. Lumbar puncture was performed on two occasions, and a turbid fluid containing pus cells withdrawn. Microscopical examination of this fluid for bacteria was negative. There was a leucocytosis of 15,000, and a slight albuminuria with a few hyaline casts. The patient died on the fifth day without regaining consciousness. At the autopsy there was venous congestion of the meninges, and there was an exudate upon the under surface of the cerebellum and on the thoracic portion of the cord. The dura mater was adherent to the skull.

CASE II.—A. H., a salesman, aged twenty-three years, was suddenly attacked with vertigo associated with faintness and nausea. This lasted about five minutes. Similar attacks recurred at intervals, and he also complained of nose-bleed. Three weeks later when he was admitted to the hospital it was noted that his vision was impaired. He complained of dizziness; the gait was staggering and hesitating; the eye-grounds were reported to be characteristic of inherited syphilis, showing extensive exudative disseminated retinochoroiditis. Potassium iodide, however, produced no effect. Speech was hesitating; there was occasional vomiting; the knee-jerks were increased, and patellar clonus was present on the right side, and ankle clonus on both sides. Babinski's sign and Kernig's sign were not present. There was no retraction of the head, no loss of consciousness, but speech became more and more impaired, and the patient died in the fourth week of the disease. The autopsy showed the presence of a subacute tuberculous leptomeningitis. The membranes at the base of the brain were very adherent, and were separated with considerable difficulty. A few old calcified tubercles were found in the lung, and the right lobe of the liver showed some old healed tubercles.

CASE III.—T. B., an Englishman, aged forty-eight years, a tailor by occupation. Two days before admission he had become violent and had destroyed the furniture of the house. On admission he was unconscious, delirious, and there was continual twitching of the hands and feet. The thoracic and abdominal organs were normal. Four days after the onset of the disease he developed marked retraction of the head, but the reflexes were normal. Babinski's sign was not present and Kernig's could not be elicited. Lumbar puncture was performed on two occasions, and the tubercle bacillus was found in the clear fluid withdrawn. There was slight albuminuria and a few casts. The patient died on the seventh day, and at the autopsy miliary tubercles were found in the pia mater, and tuberculosis of the left lung, the liver, the spleen, and the kidneys.

Briefly summarized, there is one case of acute cerebro-spinal leptomeningitis whose bacterial nature was not determined, and two cases of tuberculous meningitis. In all of these cases Kernig's sign was repeatedly tested and was never present, no difficulty being experienced in any instance in extending the leg beyond an angle of 150°. This extension did not cause undue prominence of the hamstring tendons, nor apparently hurt the patient. The cases confirm the opinion that Kernig's sign is especially unreliable in tuberculous meningitis.

THE TREATMENT OF THROMBOSIS OF THE LATERAL SINUS FOLLOWING MIDDLE EAR SUPPURATION.¹

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Up to about ten years ago the infection of intracranial venous channels following an inflammatory process within the middle ear and mastoid was looked upon as an almost necessarily fatal complication. At this time operative otology was in its infancy. The aural surgeon was exceedingly loath to institute any radical method of treatment for the relief of middle-ear suppuration. Even incision of the drum membrane for the evacuation of pus within the tympanum was looked upon as a somewhat grave procedure, and was resorted to only in cases where, after a long period, nature did not evacuate the abscess by means of spontaneous rupture of the membrana tympani. When it became evident that the inflammatory process had extended into the osseous structures adjacent to the ear—that is, into the mastoid cells—the surgeon was even more loath to employ radical measures to relieve the disease. An operation having for its purpose the thorough cleaning out of the mastoid cells and the securing of free drainage of the tympanum through a posterior opening was only resorted to at the very last moment, and was then looked upon as a grave procedure.

Schwartz² may be looked upon as the pioneer in mastoid work; he was the first to lay down the indications for operative interference upon the mastoid and to formulate a rational operative technique. While the indications for the necessity of operative interference have undergone no essential modifications during the last decade, the technique of operation has naturally been greatly improved.

The original Schwartz operation consisted in thoroughly opening the mastoid antrum and establishing through-and-through drainage, in most cases, between the posterior opening and the external auditory meatus, although this was not insisted upon absolutely. As time went on operators gradually became more and more radical. Instead of limiting their operative procedures to the antrum alone, the adjacent mastoid cells were explored for evidences of disease. It remained for Gruening, of New York, however, to first thoroughly formulate the technique of an operation which had for its purpose the removal of

¹ Read before the Southern Section, American Laryngological, Rhinological, and Otological Society, February 3, 1902.

² Archiv für Ohrenheilk., vol. vii. p. 157.

practically all of the cellular structures of the mastoid and the eradication of all foci of diseased bone. Gruening laid particular stress upon the removal of the mastoid tip, thus evacuating the inflammatory products from a few large cells so frequently located in this region.

If one cares to consult the statistics of the fatality of middle-ear supuration complicated by mastoid involvement, it is surprising how the number of fatal cases has diminished within the last decade. Coincident with this change it will be found that the number of cases operated upon has steadily increased. In other words, it has been conclusively proven that early and prompt operation constitutes the only safe method of dealing with the severer forms of middle-ear inflammation—that is, those complicated by involvement of the bony structures adjacent to the tympanum. As soon as the surgeon became bold enough to perform a complete and thorough mastoid operation it was found that many of the accidents so much feared when the operation was less completely performed practically did not occur. At the time when the mastoid operation consisted simply in perforating the cortex over the region of the antrum by means of a drill, one of the most grave casualties which could occur was the accidental wounding of the lateral sinus. This was always looked upon as an injury of great severity, and one which was not infrequently followed by fatal results. When the more complete operation of removal of the entire mastoid cortex came into vogue it was found that the lateral sinus was not infrequently exposed for a considerable portion of its length, and that the favorable outcome of the case was in no way affected by even extensive exposure of this large venous channel. It was also found that even the accidental wounding of the lateral sinus was not followed by any grave symptoms. It became evident that the previous fatal cases ascribed to this accident were due to the fact that it was necessary to stop the operation before evacuating the purulent focus within the bone. With a small opening it was practically impossible to control the hemorrhage from the sinus, and the operation had to be stopped immediately. With the mastoid cortex entirely removed the hemorrhage could easily be controlled by pressure, the operation could be proceeded with, the purulent collection evacuated, and the patient entirely relieved. Gradually the operator became more daring, and when the lateral sinus was exposed during the course of the operation it was carefully inspected and examined for any evidences of infection having extended into this region. This was especially true in those cases where the temperature of the patient seemed to indicate severe systemic infection. When the sinus was found to contain a thrombus partially or completely filling its lumen, the next step was to turn out this clot, to thoroughly curette the sinus wall to remove any possible source of systemic infection, and to then control the hemorrhage by means of a firm, aseptic packing.

This procedure has been followed by excellent results. It was found, however, that in spite of such radical interference in the immediate neighborhood of the mastoid region a large proportion of cases of invasion of the lateral sinus terminated fatally or only recovered after suffering from a severe general pyæmia. A study of these cases led Ballance¹ to institute a procedure of the greatest importance in aural surgery. Realizing that the direct avenue of infection from the breaking down of thrombus in the lateral sinus was through the internal jugular vein, Ballance proposed and successfully carried out the procedure of ligating the internal jugular in the neck, so as to completely shut out any infection through this channel. Following out this plan of treatment, some surgeons were contented to simply ligate the internal jugular; others divided the vessel between two ligatures, and then attempted to wash out the thrombus by irrigation from the cut end of the jugular up through the sinus wound. A further advance in operative technique led to a ligation of all the tributaries of the internal jugular, and finally to the excision of the internal jugular from a point low down in the neck to a point just below the entrance of the vein into the base of the skull, all tributary branches being tied off. This latter procedure seems to be the one of election at the present day.

It is a matter of great importance to the otologist to know in exactly what cases a simple freeing of the lateral sinus from the infective thrombus will be sufficient to prevent systemic infection, and, on the other hand, in what cases it will be necessary to perform the more radical operation of excision of the internal jugular vein. The statistics of cases operated upon within the last decade have been thoroughly collated by Koerner² and Hessler.³

Koerner (page 72) has reported 20 cases operated upon, in 8 of which the internal jugular was not ligated. Of these 8 cases, 4 recovered and 4 died. In 12 cases the jugular was ligated, and of these 9 recovered.

Hessler⁴ reports 54 cases of sinus phlebitis treated by opening the sinus. Of these, 31 cases have recovered and 23 died. In 23 cases the jugular vein was either ligated or opened throughout its entire extent. Of these, 16 recovered and 7 died. In 4 cases where the treatment consisted simply in ligation of the internal jugular, 2 recovered and 2 died.

The statistics of both these authors would seem to indicate the advisability of either resection of the internal jugular vein or, in cases where this was impossible, of dividing it low down in the neck, between two ligatures, opening the vein throughout its entire extent and packing the cavity with iodoform gauze, in order to prevent purulent infection.

¹ *Lancet*, May 17, 1890.

² *Die Otischen Erkrankungen des Hirns der Hirnhäute und der Blutleiter*, 1894.

³ *Die Oogene Pyæmie*, 1896.

⁴ *Loc. cit.*, p. 461.

I am convinced, however, that statistics of this kind are apt to be misleading. During the last few years I have operated upon 22 cases with sinus thrombosis. Of these 22 cases 2 have died, one of septic pneumonia, without any evidence at any time that the internal jugular vein was involved; the other of an acute nephritis, apparently induced by the administration of ether. In 4 of these cases only was it found necessary to ligate the internal jugular vein, and in all of these 4 cases recovery took place. In addition to these cases I have reported a fifth case of septic thrombosis of the internal jugular following a middle ear operation for a chronic suppurative otitis media. In this case the internal jugular was also excised, and the patient made a complete recovery.¹

From these statistics it will be seen that from a relatively large number of cases of thrombosis of the sinus operated upon without interference with the internal jugular vein, the proportion of recoveries is exceedingly large and much in excess of those reported by either Koerner or Heesler; while in those cases in which the internal jugular has been excised in no single instance has death followed. In the one case in which death might possibly have been due to septic infection—that is, in which the patient died of pneumonia—it is quite possible that the pulmonary infection was tubercular in character, although bacteriological examination failed to confirm this view. The temperature chart was certainly not one characteristic of septic infection.

The question of practical moment to the surgeon and to the otologist is, then, How shall we differentiate between these cases? My own experience has led me to believe that prompt operative interference of the most radical kind is the only plan which promises safety to the patient. Cases of middle ear suppuration daily come under observation, especially in dispensary practice, with an exceedingly obscure and imperfect history. Many times it is impossible to keep the patient under observation for even twenty-four hours before operation. The indications for immediate operation upon the mastoid being imperative, there is no opportunity to observe the general condition of the patient or to make note of those variations in temperature which are so characteristic of infection of the lateral sinus and of a dissemination of this infection through the general circulation. My experience has been that the surgeon must decide during the primary operation what course he will pursue. It has been my invariable practice during the last five or six years whenever I have performed a mastoid operation to remove thoroughly all evidences of carious bone, and to follow the softened bone to the dura in every direction, if this is necessary for the complete eradication of the infected areas. When the lateral sinus is exposed,

¹ Archives of Otolary, vol. xxvii. p. 297.

unless this vessel appears perfectly healthy on palpation, and unless the external surface is white and glistening and the vessel seems to be of normal size, it is my invariable practice to incise the sinus wall for exploratory purposes. I was formerly in the habit of exploring the sinus by the introduction of a large aspirating needle. I soon found, however, that in some instances where infection of the sinus had taken place, and where a clot was present, the exploring needle withdrew fluid blood. I have, therefore, within the last few years abandoned this procedure entirely; and in every case where I am in doubt as to whether or not the sinus is normal I have made a free incision through the sinus wall. It goes without saying that previous to this exploratory incision every purulent focus must have been removed from the mastoid process—that is, the mastoid antrum must have been thoroughly cleaned out and all pneumatic spaces must have been broken down and curetted, so as to avoid any possible infection of the sinus as the result of the exploratory opening. After this has been done it has been my practice to pack off the antrum by means of iodoform gauze and to thoroughly cleanse the entire field of operation before opening the sinus. All instruments are also carefully re-sterilized before this exploratory incision is made. Before making an exploratory incision into the sinus wall it must be borne in mind that the sinus must be exposed by removing the bony covering of the sigmoid groove for a distance of at least three-quarters of an inch. Pressure is then applied to the sinus above, and the sinus wall is incised close to the knee by means of a sharp, straight bistoury. This incision should lie at about the middle of the area of sinus exposure. If there is a clot in the sinus at this point there will either be no hemorrhage at all or a very slight hemorrhage from below. A curette is then passed into the lumen of the sinus and the clot removed, so that free bleeding is established from below. Pressure is now applied at the point of incision in the sinus and the pressure above is removed. If free hemorrhage does not occur from the direction of the torcular a curette is introduced in this direction, and the lumen of the vessel thoroughly curetted until free hemorrhage is established, it being imperative that free hemorrhage be established from the upper end of the sinus, excepting in those cases where the lumen of the vessel is occluded by a firm fibrinous clot which has evidently been in position for a long time and has become thoroughly organized. Where such a condition is found, and where the lumen of the vessel seems to be obliterated and the entire vessel has been converted into a mass of connective tissue, it is then unwise to attempt to break down this firm plug in the upper portion of the sinus. Naturally, if the clot has become firmly organized no further infection can result from it. Where free hemorrhage is established both from the upper and lower angles of the sinus wound, or where the upper portion

of the vessel seems to be firmly occluded by an organized clot and there is moderate bleeding from the lower angle of the wound, I do not deem it advisable to institute a more radical procedure at the time of the primary operation. Hemorrhage should be controlled by firmly packing the sinus wound by means of iodoform gauze, and shutting off the sinus area from the middle ear and adjacent mastoid cells by means of a firm packing of iodoform gauze. Such a dressing will remain in position for from four to six days, and there is no indication for its removal unless there is a sudden rise of temperature to 103° to 104° F.

If all suspicious cases are treated in this manner at the time of primary operation, and if, whenever there is any doubt as to a possible involvement of the sinus, this vessel is opened for the purpose of exploration simply, I can say, as the result of a rather long experience, that no possible unfavorable issue is to be expected. On the other hand, if, after a period of from one to three days, the temperature shows any indication of systemic infection, I have always found it advisable to proceed immediately to prevent absorption of the poison by means of excision of the internal jugular vein.

In two cases already reported¹ and in two to be mentioned later this necessity arose; and in another case² where primary infection of the internal jugular occurred as the result of a middle ear operation this same plan was followed. In all of these cases the temperature fell immediately after the operation upon the middle ear or mastoid, but in the course of a few hours showed those marked remissions characteristic of pyæmic infection.

In these cases my procedure has been as follows: The internal jugular vein was exposed throughout its entire length by means of an incision extending from the sternal attachment of the sternomastoid muscle to the tip of the mastoid process. Owing to the ease with which the internal jugular is exposed low down in the neck, the vessel was in every instance dissected from its sheath first in this region, surrounded by two ligatures, and divided between them. The distal end of the vein was then clamped with an artery clamp, and being raised by an assistant was rapidly dissected upward until the common trunk of the temporal and facial was reached. This trunk was surrounded by two ligatures and divided between them. Occasionally the thyroid veins were large enough to demand attention, and they were treated in exactly the same manner as the common trunk of the temporal and facial. There is also found at times a branch of communication between the external and internal jugular. Where this vessel is large enough to occasion free hemorrhage after its division it should be surrounded by two ligatures and divided between them. In this way the entire trunk of the jugular

¹ Loc. cit., vol. xxix, p. 472.

² Loc. cit., p. 471.

is dissected out from its position from a point just above the clavicle to a point just below the base of the skull, the vein being followed as high up as is possible. At the uppermost point of dissection two ligatures are again thrown around the trunk of the vein and the vessel is divided between them. In this way any systemic infection is practically prevented. The lateral sinus having been freed of all infectious material from above, and the jugular trunk having been removed in its entirety from below, there only remains a small portion of the sinus occupying the jugular fossa, and commonly known as the jugular bulb, which can be a source of infection. Simple cleansing by means of irrigation through the sinus wound will effectually free the jugular bulb from any infectious material.

In cases where the infection of the vein has existed for some time, or where the process has been very rapid, the tissues may be found to be so matted together about the vein as to preclude the possibility of a complete dissection of the internal jugular.

I reported such a case in the *Archives of Otolaryngology*, vol. xxix. p. 472, in which the disintegration of the walls of the vessel had been so extensive that the vein could only be secured by pulling it forcibly upward from beneath the clavicle. The tributary vessels were so much involved that it was only possible to tie them by means of including a considerable amount of the adjacent muscular tissue and fascia between the ligatures. In this instance it was, however, decided to pass two ligatures about the vein, close to the base of the skull, and to divide the vessel between these two ligatures. In this way the infective focus was cut off from the general circulation, the entire wound was packed with iodoform gauze, and the vein left *in situ*. Complete recovery followed operation.

In addition to the three cases of thrombosis of the internal jugular already mentioned, two others have come under my observation during the last year which it is perhaps worth while to report somewhat in detail:

CASE I.—Male, aged thirty-one years, who came to the hospital with the characteristic symptoms of an acute mastoiditis. Under ether the mastoid was opened in the typical manner by one of my colleagues. Owing to a malposition of the lateral sinus, the sinus was entered either by the gouge, or, as I believe more likely, the internal table had already become carious, and on removal of a spicula of bone which was adherent to the sinus the necrotic wall of the vessel was ruptured; at any rate, quite free hemorrhage followed. I then took charge of the case, and succeeded in controlling the hemorrhage and opening the mastoid antrum. I exposed the lateral sinus for a distance of about three-quarters of an inch, and incised the wall in the manner already described. The sinus was not perfectly normal, and there was a small parietal clot. The wound was packed in the manner detailed above and the patient returned to bed. The temperature steadily rose until, on the day following the operation, it was 104.5° F. This eleva-

tion continued for two days. On the fourth day after operation the temperature reached 104.8° F., and the patient was drowsy. Although this persistent temperature elevation was not what would be expected as the result of thrombosis of the lateral sinus, I deemed it advisable, on account of the hemorrhage from the sinus at the time of the operation and from the fact that it was possible that all infective material had not been removed from the cavity of the vessel, to prevent any possibility of systemic infection by ligation of the internal jugular. The vein was therefore exposed in the neck, in the manner already described, and was thoroughly dissected out from a point just above the clavicle to a point just below the base of the skull, all tributaries being divided between two ligatures. During the course of the operation the internal jugular was wounded, and the patient lost considerable blood. The vein contained fluid blood throughout its entire extent, although it seemed to be slightly narrowed at a point just below the base of the skull. Owing to the loss of blood at the time of operation, the patient did not rally for about thirty-six hours. At the end of this time, however, the temperature had dropped slowly, and meantime the condition of the patient had improved decidedly. Although the wound in the neck had been sutured, infection occurred, and the wound healed by granulation. During the period of convalescence the patient developed a mild rheumatic inflammation of both shoulder-joints. At no time, however, did this seem to be septic in character. The patient made a complete recovery, and when seen nine months after the operation was entirely well.

This case, I think, demonstrates rather clearly the necessity of prompt operative interference, having for its purpose the cutting off of all circulation through the affected side in case the temperature after primary operation upon the sinus remains high.

Another case operated upon a few weeks ago bears out this point.

CASE II.—The patient, a male, aged eight years, was admitted to the hospital with the history of having had a purulent discharge from the right ear for about six years. For a week there had been pain in the ear, and for three or four days the patient had been feverish and somewhat chilly. On examination it was found that there was a subperiosteal abscess behind the ear. This was evacuated by operation and the cavity of the abscess curetted. On exposing the mastoid cortex this was found to be perforated in several places. The cortex of the mastoid was removed, and the interior was found to be filled with pus and granulation tissue. The bony wall of the sinus was destroyed, and the lateral sinus was bathed in pus. The sinus wall was protected by a pad of iodoform gauze; the antrum was thoroughly curetted and all necrotic tissue entirely removed. During this procedure the dura was exposed in the region of the tegmen tympani over a small area. The antrum was now packed with iodoform gauze, and the sinus was further exposed from the knee to a point just above the bulb. The wall of the sinus was incised, and this incision was followed by no bleeding, the sinus being filled by a clot which completely occluded its lumen. An attempt was made to remove this clot by means of a curette introduced into the sinus and passed downward toward the bulb. The clot was found to be fairly firm, and absolutely no hemorrhage followed the use of the curette.

The curette was next introduced into the sinus in the direction of the torcular, and free hemorrhage was established. The sinus was plugged in this region, and, owing to the fact that the clot was soft and that the lower end of the sinus could not be made perfectly patent, it was deemed advisable to proceed at once to excision of the jugular vein. This was accomplished in the manner already described. When the ligature was placed about the vein close to the base of the skull the pressure of the ligature severed the vessel completely. No hemorrhage followed. The external wound in the neck was sutured for three-quarters of its length, the wound being left open at the upper angle, where a packing of iodoform gauze was carried inward into the jugular fossa. The patient was returned to bed, and the first dressing was changed on the fourth day. The temperature had remained normal up to this time. After this the dressing was changed either every day or every alternate day until the twelfth day, when a slight elevation of temperature was noticed. The temperature was not high, but would vary between normal in the morning to 101° to 101.5° F. in the afternoon. This continued for about three days, in spite of daily dressings. At this time I dressed the case myself, and, upon the introduction of a director into the lower angle of the sinus wound, found a considerable collection of pus in the jugular bulb. The probe introduced into the wound in the neck at the point where the jugular had been divided also evacuated about 10 minims of pus. Both the lower sinus wound and the wound in the neck were thoroughly irrigated with a solution of bichloride of mercury of a strength of 1 : 5000, and both wounds were thoroughly packed with iodoform gauze. After this there was no further rise in the temperature, and the patient made an uneventful recovery. The wound in the neck did not unite by first intention, but healed by granulation. A bacteriological examination of the clot removed from the sinus showed a few streptococci. Careful inspection of the vein, however, at its upper portion showed absolutely nothing abnormal. In spite of this fact the temperature fell immediately after operation, and continued to remain normal until incomplete drainage caused a slight temporary temperature elevation.

This case seems to show the advisability of prompt operative interference in cases where the history is uncertain and where a clot is found to occlude the lower portion of the sinus. I do not say that this means that this case might not have recovered had not the jugular been interfered with. In view of the fact, however, that even after a complete cutting off of the purulent focus from the general circulation the comparatively small amount of pus retained in the bulb was sufficient to give rise to marked temperature changes, I feel convinced that had the jugular at that time been in position, thus affording an avenue of entrance for the purulent germs to the general circulation, the termination of the case would certainly have been fatal.

It seems to me wise, therefore, in all doubtful cases, to remove the internal jugular vein in order to eliminate this element of doubt in the case. I would not be misunderstood in this statement. I do not advise that in every instance of sinus thrombosis the internal jugular

be interfered with. I believe that where the patient has been under observation for a few days, and where we have a fairly complete temperature record of from twenty-four to forty-eight hours, showing no marked evidences of systemic infection, the surgeon may rely upon simply clearing out the clot in the sinus, if this be found. I believe that this same procedure should be followed in cases which first come under observation at the time of operation and in which the surgeon is able to clear the sinus fairly well of the clot by means of the curette. On the other hand, those cases which are first seen at the time of operation, and in which the sinus cannot be thoroughly cleared, and in which also the surgeon is confident that a certain amount of infected material is left in this venous channel, demand, I believe, immediate excision of the internal jugular vein. The operation is in no way a serious one, and, as it consumes but little time, does not endanger the patient by prolonging materially the period of anæsthesia, while, at the same time, it prevents any possibility of further systemic infection. I also believe that in those cases where after primary operation in which the sinus has been cleared as freely as possible, and where, in spite of this, at the end of forty-eight or seventy-two hours evidences of systemic infection are still present, the surgeon should not delay further operative interference—that is, he should immediately proceed to the excision of the internal jugular vein.

In deciding upon the advisability in the cases last mentioned I believe that the temperature chart is the only sure and certain guide. Much has been said by various authors about tenderness in the neck over the region of the jugular. This may be present, or it may be absent. Even in the absence of any such tenderness, with a remittent temperature which is gradually becoming higher and higher, and with a certain knowledge that the lateral sinus is already infected, I believe that the surgeon errs on the side of conservatism who longer delays operative interference.

HEMIHYPERTONIA POSTAPOPLECTICA, WITH SOME REMARKS ON A CONTRALATERAL ACCESSORY MOTOR SPEECH CENTRE.¹

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V. BECHTEREW² reported three cases of a condition to which he gave the name hemitonia apoplectica. Spiller,³ shortly after the appearance

¹ Presented at the November meeting of the Philadelphia Neurological Society.

² Deutsche Zeitschrift für Nervenheilk., 1900.

³ Philadelphia Med. Journ., 1899.

of this paper, read a paper on the subject, and suggested the name "hemihypertonia postapoplectica." The case here reported is the only case, other than that presented in Spiller's paper, reported in this country. One other case has been reported in the German literature.

The condition as described by v. Bechterew is one of intermittent tonic spasm distributed over one-half of the body, affecting at times different groups of muscles, without loss of power in the muscles affected, and follows an apoplectic attack. He reports in his paper three cases. In all three cases the initial lesion occurred in early life—before the tenth year. This was also true of Spiller's case. In two of the three cases the apoplectic attack followed some infection. The clinical course of the three cases and the other cases reported varies considerably. The apoplectic attack lasted a few minutes in one of v. Bechterew's cases, and in another continued over a period of five days. In Pfeiffer's¹ and in my case there was no unconscious period at all. The development of the spasm and the degree of paralysis differs in different cases. The spasm may develop immediately after the apoplectic attack or initial symptom group, or may be deferred months or even a period of two years, as in the case reported in this paper. The loss of motor power may vary from a distinct hemiplegic weakness to cases in which there is no appreciable loss of power. After a varying interval of time following the cerebral disturbance certain groups of muscles on one side of the body (the arm, leg, and face are usually affected) go into a condition of hypertonicity. A slight irritation, such as a pin-prick or rough handling, is sufficient to throw these muscles into a tetanic spasm. After a few seconds or minutes the spasm passes from the flexor groups to the extensor groups of muscles, and then disappears. In some cases there may be repeated alternations of these flexor and extensor spasms, and as each repeated spasm is associated with a corresponding flexor or extensor movement the result might be mistaken for athetosis. The spasmodic condition of the muscles is somewhat relieved by distracting the patient's attention or by light rubbing of the part. In one of the cases the spasm disappeared during sleep; in two other cases a tenseness of the muscles persisted during sleep. The deep reflexes and the muscle irritability are usually increased.

The main features in the case about to be described are: 1. The presence of an attack of dizziness, associated with numbness in the left arm, and aphasia lasting twenty minutes. 2. An attack of vertigo in 1898, following which she had severe occipital headaches for a period of about a year. 3. The development, almost a year after the second attack of vertigo (fall of 1900), of intermittent, alternating flexor and extensor spasms affecting the left lower leg and arm, associated with

¹ Neurologisches Centralblatt, 1901.

a very pronounced increase of the muscle irritability and the knee-jerks. The detailed history is as follows:

M. D., aged forty-seven years, single, female, dressmaker by occupation, came to the clinic complaining of nervousness resulting from spasm of her foot and hand. Her father had died of a paralytic stroke. There was no other nervous affection in her family. She had had the usual diseases of childhood, followed by typhoid at ten years of age. She had a second attack of typhoid at the thirtieth year. She was in fairly good health from that time until the fall of 1898.

FIG. 1.



FIG. 2.



Present History. In the fall of 1898, while at work, an attack of dizziness occurred, which lasted twenty minutes. During that time her tongue was so thick that she was unable to talk. There was numbness in the left arm and face. The left arm was weak, but she could still move it. The left face drooped, and a physician who saw her in the attack said she had a slight stroke. All the symptoms passed off with the dizziness at the end of twenty minutes. There was no loss of consciousness. In the fall of the following year another attack of dizziness occurred. Objects in front of her swayed from one side to the other. She sank slowly and easily to the floor, to prevent herself from falling. There was no unconsciousness with this attack, nor were there any of the symptoms noted in the previous attack. For a period of about a year following this attack she was troubled with severe occipital head-

aches. There has been no vomiting or other symptoms of intracranial pressure.

In the fall of 1900 the spasm of the foot began to develop. She first noticed that at times, when walking, the middle toe of the left foot would become contracted and prevent her from walking until she rested for a time and gave the part a chance to recover its normal condition. Since that time this condition has gradually grown worse, until now all the toes, the lower leg muscle groups, and to a slight extent the upper leg groups, are affected. Any rough handling or irritation or an attempt to elicit the plantar reflex will bring on a tonic flexor spasm of the smaller toes (see Fig. 1) and an extensor spasm of the great toe, which after several minutes may change to an extensor spasm of all the toes or disappear without any extensor spasm. During the spasm of the toes the calf muscles are hard and rigid. There is a considerable degree of hypertrophy of the muscle groups affected. The hand is held in a position of flexion, with the thumb drawn in toward the palm. (See Fig. 2.) There is always an increased tonus in the hand and arm muscles, and at times the condition of flexor tetanic spasm is very similar to that seen in tetany. The spasm of the leg or arm muscles occurred usually only at long intervals. In walking a hundred yards the patient was compelled to stop four times on account of the spasm in the foot.

There is no appreciable weakness either in the arm or the leg. The dynamometer: Right side, 70; left hand, 65. The tongue deviates constantly to the left. Pupils are equal, eye reflexes normal, fundi normal. Knee-jerk: left, increased; right, increased. Achilles jerk: left, normal; right, absent. Plantar reflex: left, difficult to determine, on account of spasm; right, absent. Biceps jerk: left, present, increased; right, present. Triceps jerk: left, present, increased; right, present. Chin jerk: left, present; right, present. Supraorbital reflex: left, increased; right, present.

The muscle irritability to mechanical irritation was very markedly increased in all the muscle groups of the left arm and leg, and to a lesser degree on the right side of the body. Sensation is normal. There is no astereognosis.

A fact of considerable importance in the history is that she has used the left hand since early childhood much more than the right, although she writes with the right hand. It would therefore appear from the symptoms presented in this case—such as the temporary aphasia, with persistent deviation of the tongue, for several years, and temporary loss of power in the right arm—that in the original cerebral lesion the speech fibres were affected. The rapid recovery of the function of speech in the presence of the paralysis of the tongue could be explained in this case, and in another case of persistent and complete hemiplegia in a left-handed person who later learned to use the right hand for writing purposes, and in whom the loss of speech lasted several weeks after the apoplectic attack, by assuming that in such cases there is a fairly well-developed speech centre in both hemispheres. The original speech centre evidently is the main centre, because in the hemiplegic case the patient was aphasic for several weeks after a

right-sided lesion ; and what might be termed the accessory contralateral motor speech centre, developed coincidentally with the education of the hand in writing movements, takes up the function of speech under such conditions only after some additional training and considerable effort on the part of the afflicted individual.

The absence of any period of unconsciousness in the case of hypertonia here reported is of considerable importance. The name suggested for this condition by v. Bechterew and the modification suggested by Spiller both assume an apoplectic attack, which if restricted to its exact meaning would necessarily imply an unconscious period.

The absence of any marked weakness other than that of the tongue is sufficient to differentiate it from other postapoplectic conditions, such as athetosis, chorea, tremor, contracture, etc. The almost if not complete absence of paralysis in this case should stamp it as the most typical example of this affection on record. The case is somewhat different from those described by v. Bechterew in the absence of this initial unconscious period and in the long period intervening between the cerebral insult and the development of the hypertonia. There has as yet been no autopsy in a case of hypertonia apoplectica. The determination of the nature and character of the lesion and its position must, therefore, be open to discussion until such an autopsy occurs. v. Bechterew holds that the lesion is not cortical, on account of the absence of clonic convulsive movements ; that it is not subcortical, on account of the wide extent of the peripheral manifestations necessitating an equally wide-spread lesion in the subcortex ; and that it is probably a lesion of the basal ganglia in the neighborhood of the posterior limb of the internal capsule. The symptoms can best be explained, he thinks, by a small lesion causing direct irritation of the motor tracts or secondary irritation through the tension of a healing cicatrix. This case is reported from the clinic of Dr. Spiller at the Philadelphia Polyclinic.

THE ETIOLOGY OF INFANTILE PARALYSIS.

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MUCH discussion has arisen as to the causation of the disease known as infantile paralysis, but the question is still far from being settled. Our knowledge as to its nature, derived from its clinical aspects and from a careful examination after death of the bodies of its victims, is assisted by the flood of light which recent research has shed on the anatomy, physiology, and pathology of the muscular and nervous sys-

tems. Infantile paralysis is characterized by a loss of muscular power in one or more limbs of acute onset, followed by more or less muscular wasting. We now know that muscle fibres depend for their nutrition on their connection with certain cells in the spinal cord by means of the nerve fibres that supply them, so that destruction of the nerve cell involves destruction of the nerve fibre and its attached muscle fibre. Disease and destruction of the muscle or nerve fibre do not necessarily, however, bring in their train death of the nerve cell; indeed, the nerve fibre may be regenerated by a fresh outgrowth from the nerve cell and form a fresh attachment to the muscle fibre.

Paralysis and degeneration of the muscle fibres may be due to a primary disease of the muscle, the nerve to the muscle, or the cell of which that nerve is a process. Primary disease of muscle fibres is usually chronic in its course, and since it is generally hereditary, is probably due to some congenital defect. Occasionally, it is true, acute degeneration of muscle fibres does take place in certain specific fevers, probably from the action of some toxin. Hence an acute destruction of muscle fibres is more commonly due to some disease either of its nerve fibre or of the nerve cell from which that fibre is derived. The nerve cell and fibre¹ may be attacked separately or together, and the resulting disease may be acute or chronic in its course. Destruction of the nerve fibre does not necessarily mean a permanent wasting of the muscle fibre, since a fresh connection may be established between the nerve cell and the muscle, while destruction of nerve cells, since they are never replaced, always involves the complete degeneration of their muscle fibres. A local injury may separate the nerve cell from the muscle by severing the nerve fibre or may destroy the nerve cell itself. Inflammatory processes may involve the nerve cell or fibre, and so may temporarily or permanently render them useless, and their functions may similarly be temporarily or permanently abolished by some lesion that cuts off their blood-supply. Lastly, the nerve cell or fibre may be acutely disabled by the action of some poison circulating in the blood, and it is possible that such a poison would either only temporarily impair their functions or so seriously damage them that recovery would be impossible. It has been shown during the last forty years that the muscular weakness and wasting of infantile palsy are always due to destruction of the nerve cells in the ventral horns of the spinal cord. Widely different views have, however, been expressed as to how and why the cells are attacked. Clinically there would seem to be more than one disease grouped together under the same name, while the pathologists have regarded the disease as single and due to a single cause. In attempting to ascertain the nature of the complaint it is

¹ Strümpell and Bartholmes. *Deut. Ztscht. f. Nervheilk.*, 1900, xviii. p. 304.

essential to give due weight to both the clinical and pathological evidence.

Clinically there are certain features of the disease which may be considered first. It attacks the very young by preference, being almost confined to the first four years of life, and comparatively rare after the third year. Cases do occur in adult life, but are very infrequent. The disease has a seasonal predilection, being most frequently met with in the hot weather, *e. g.*, the months of July, August, and September. It does not tend apparently to specially attack weakly children, the robust, well-nourished infant being as liable to be affected as one that is feeble and wasted. There does seem, however, to be a special liability to attack in children who have recently suffered from the acute specific fevers of childhood, measles, scarlet fever, and diphtheria.¹ The paralysis is at its maximum in extent within a short time of the onset, and is almost invariably much more extensive at first than it is later on. Recovery of power starts almost at once and progresses gradually over several months; complete recovery, however, does not take place, or, in other words, some of the motor cells are always destroyed.² The permanent paralysis may be limited to a single limb or to a single group of muscles, or even a single muscle, although at first all the limbs may have been nearly powerless. The lower limbs are attacked much more frequently than the upper. Following on the local paralysis, local deformities frequently develop, owing to the unopposed action of the healthy muscles. When there has been extensive paralysis of one limb it is usually colder than its fellow, and there is a general want of development, so that the bones in the paralyzed limb are smaller and shorter than in the healthy one. The loss of power in the permanently paralyzed muscle fibres is absolute from the first, and there is a rapid loss of electrical excitability, as rapid as when the nerve to the muscle is cut. In muscles that are about to recover, the loss of power is either not quite absolute or is only absolute for a very short time. The electrical excitability of these muscles is, too, never quite lost, there being always some reaction to the galvanic current. As in other cord diseases, an acute arthritis sometimes occurs which may be mistaken for acute rheumatism.³

Although nearly all cases of infantile paralysis conform to the description given above, they differ markedly from one another in their clinical aspect during the first few days. One may, indeed, divide them into classes according to these initial symptoms. The most striking cases, although not the most numerous, are those in which a child without any other sign of ill-health becomes suddenly paralyzed. On

¹ Zappert. *Jahrb. f. Kinderheilk.*, liii. Gowers. *Diseases of Nervous System*, vol. i. p. 353.

² Pasteur's cases, given below.

³ Coutts. *Medical Times and Gazette*, 1885, p. 76. Barlow. *British Medical Association*, 1883.

awakening it in the morning the mother may find it unable to move one or more limbs, or the onset may be even more sudden than this, the child tripping or falling in walking or running, and when picked up being found paralyzed in one leg. This sudden loss of power is unaccompanied by any pain or sign of the patient not being perfectly well, and there is not the slightest alteration of sensation to be found. Such a sudden onset is more common in young children, under the age of two years. According to Coutts,¹ in cases which have commenced in this sudden fashion, the initial palsy is usually limited to one limb, and the ultimate persistent paralysis is confined to a few muscles in that limb.

CASE I.—Maud R., aged two years, was found by her mother one morning to have lost the use of the right arm, having been perfectly well the night before. On examination there was flaccid paralysis of all the muscles of the right arm and shoulder; some slight movement of fingers. No other limbs affected.

In another class may be put the more common type of case where there is a good deal of fever and malaise and frequently vomiting for some hours or even a day or two before the onset of the paralysis. The amount of the early loss of power is usually wide-spread, both lower limbs, and both upper limbs and trunk being affected. The permanent paralysis, too, is frequently very extensive. The full extent of the paralysis may appear suddenly, or it may require some hours or even days to develop. Pain and tenderness in the affected limbs are not uncommon symptoms, but no other affection of sensibility seems to occur. As a type of this class one may record the following case:

CASE II.—E. P., aged five years, a bright, lively girl, was noticed to have lost her appetite at supper one night. She vomited after supper and several times during the night. Next day she was found to be unable to stand or even to sit up, and she complained of considerable pain in the back. On admission to the hospital there was flaccid paralysis of all limbs, absence of knee-jerks and superficial reflexes; no anaesthesia and no loss of control over rectal and vesical sphincters or paralysis of cranial nerves. After admission she gradually recovered power to some extent in all muscles except the right deltoid, and at the end of three months could raise herself to a sitting position and could walk with the help of her arms. On discharge from the hospital there was permanent complete paralysis of the right deltoid and partial paralysis of all the muscles of both legs.

The disease is uncommon in adults, but such cases as do occur nearly always resemble that more common type in children which has been last described. Pain in the back and limbs are very prominent symptoms, much more so than in children, and the onset of the paralysis is

¹ Private communication to author. Gowers. *Diseases of Nervous System*, vol. i. p. 357.

much less rapid. The case reported by Williamson¹ may be taken as an example, especially as the clinical diagnosis was confirmed post-mortem.

CASE III.—A man, aged twenty-two years, was attacked with pain in the back, slight pain in the limbs, and at the same time a numb feeling was noticed in the right hand. On the next day there was numbness in the right leg and to a less extent in the limbs on the opposite side. The patient also noticed that the right arm was weak, and this weakness rapidly increased so that the right arm became completely paralyzed, and later the right leg also. On the third day the limbs of the left side became completely paralyzed. The bladder and rectum were not affected, and there was no anæsthesia. Slight improvement began after ten days, and the patient became able to just move his arms and toes. After being ill for five weeks he one day died quite suddenly. At the post-mortem examination an inflammatory condition was found in the ventral horns of the spinal cord similar to that described later on.

Very few opportunities have occurred for the examination of these adult cases post-mortem. But besides Williamson's case, a few others have been reported.²

Cases of infantile palsy generally occur sporadically, and beyond the seasonal prevalence no connection between any two individual cases can, as a rule, be found, but sometimes two or more children of a family are attacked soon after one another. More rarely a large number of cases arise in a small district within a short time, thus constituting a veritable epidemic. In such an epidemic in Vermont, reported by Caverly,³ as many as 140 cases occurred. The clinical symptoms in these epidemic cases resemble the more common type of the sporadic cases, i. e., the cases occur in the summer, there is initial fever and malaise, with headache, pain in the back, and vomiting, and then, after a day or two, loss of power comes on more or less suddenly. There are, however, certain features peculiar to these epidemics. Convulsions and coma are occasional early symptoms, while they are almost unknown in the sporadic cases. Pains in the back and limbs are more severe and more frequent, being almost invariable, and the limb pains may be associated with tenderness over the nerve trunks, and even some anæsthesia in the distribution of the peripheral nerves, showing that there is some accompanying multiple neuritis. Certain cases occurring during the epidemic, and probably arising from the same exciting agent, have

¹ Medical Chronicle, 1890, vol. xii. p. 454.

² Schultze. Virch. Archiv, 1878, vol. lxxiii. Leyden. Archiv f. Psychiatrie, 1875, vol. vi. p. 271. Friedländer. Virch. Archiv, 1882, vol. lxxxviii. p. 84.

³ Caverly. New York Medical Record, 1894, vol. xlv. p. 671. Cordier. Lyons Médical, 1888. Strümpell. Quoted by Goldscheider, Zeitschft. f. klin. Med., 1893, vol. xxiii. p. 554. Newmark. Med. News, 1899. Packard. Journal of Nervous and Mental Disease, 1899, vol. xxvi. p. 210. Zappert. Jahrb. f. Kinderheilk., vol. lili. Chapin. Archives of Pediatrics, November, 1900. Brackett. Transactions of the American Orthopedic Association, 1893, vol. xi. p. 132.

only the clinical aspect of multiple neuritis without any evidence of implication of the spinal cord at all. For instance, in the epidemic of 44 cases in Stockholm, reported by Medin,¹ there were two cases of simple multiple neuritis besides several mixed cases of anterior poliomyelitis and neuritis. Again, during an epidemic of multiple neuritis in Hamburg, reported by Eisenlohr² in 1887, where the illness started with fever, diarrhoea, and vomiting, two cases of anterior poliomyelitis occurred which Eisenlohr considered were due to the same exciting cause as the cases of neuritis. In these epidemics, too, the implication of the cranial nerves, or rather their nuclei, is comparatively frequent. The simultaneous occurrence of anterior poliomyelitis and multiple neuritis in the same patient, and probably due to the same exciting cause, has been noted in sporadic cases by several observers.³ The association, however, is common in epidemic cases and extremely rare in the sporadic. The implication of the cranial nerves, too, has been observed in sporadic cases, but only in one or two instances.

The disease itself seldom kills in the sporadic cases, only two such deaths having been reported,⁴ but a certain proportion of the patients in these epidemics have died. Death in these instances seems to be due to an attack on the respiratory centre in the bulb.⁵ We have evidence, too, that the disease in these epidemics may attack the higher cells in the brain or their axis-cylinders, *e. g.*, Caverly reports that rigidity of muscles was not uncommon in the Vermont epidemic (also Pasteur). In some of the epidemics certain patients who are obviously attacked by the prevailing disease may never show more than a slight temporary weakness of certain muscles, or even may show no muscular weakness at all. For instance, in the interesting family epidemic reported by Pasteur,⁶ all the seven children were attacked within ten days of one another with fever, backache, and headache; two of these recovered without any sign of muscular weakness, one had some muscular tremor and recovered, one had muscular tremors and transient strabismus, one had ordinary flaccid paralysis of the infantile type, one had some permanent paralysis of the infantile type, but with some initial rigidity, and the last one had hemiplegia with marked rigidity. One must confess, in the light of these cases of Pasteur's, that it is possible that the cause of the ordinary sporadic infantile palsy may produce a febrile complaint, with perhaps temporary weakness, to be

¹ Medin. Verhandlungen des X. Internat. Med. Cong., Berlin, 1890, vol. II. pt. VI., p. 87.

² Eisenlohr. Berlin. klin. Wochenschr., 1887, vol. XXIV. p. 781.

³ Strümpell, A. Neurolog. Centblatt., 1884, No. 11, p. 241. Leyden. Archiv. f. Psychiat., 1875, vol. VI. p. 271.

⁴ Drummond. Brain, 1886-88, vol. VIII. p. 14. Dauber. Deut. Zeitschft. f. Nervheilk., 1893, vol. IV. p. 200.

⁵ Medin, Drummond, Dauber.

⁶ Pasteur. Clin. Soc. Trans., 1897, vol. XXX. p. 1438.

followed by a complete recovery. We have, however, no further evidence in favor of this hypothesis.

Two cases recently shown to the Neurological Society by Dr. Coutts and myself well illustrate the general aspect of these epidemic cases:

CASE IV.—A brother and sister, aged respectively seven years and five years, were taken to Margate by their parents in the summer of 1899. On the sixth day of their stay, the girl, after a long walk, was attacked with nausea and feverishness, and then several times during the night with vomiting. The following morning she was found to have lost the use of all four limbs and to be unable to sit up. She complained, too, of pain in the back. On this day, August 12th, the family returned to London. On August 16th, the fifth day of the disease, a peculiar gurgling was noticed in the throat, as though the child was going to choke, but this symptom only lasted a short time and did not return. The mother thought that the child improved and regained some little power in both arms during the next four weeks. On September 14th she was admitted to the East London Hospital for Children. She was there found to be a well-nourished child, with the exception that the muscles generally were rather flabby. There was loss of power in all four limbs and she was totally unable to sit up, crying out with pain on any attempt to raise her to a sitting position. The legs could only be drawn up to a very slight extent and she had considerable difficulty in re-extending them, and the knee-jerks were absent. The grasp of both hands was feeble and she was unable to raise either arm from the side. The movements of the palate and eyes were unaffected. She could swallow well, and there was no affection of the sphincters of the bladder and rectum. There was anaesthesia of both legs below the knees. The muscles generally were very tender when grasped, however gently, especially those of the calves, and for a short time there was distinct tenderness over the popliteal nerves. On electrical examination on October 17th only a very slight response could be obtained to both faradism and galvanism in the lower limbs, but in the upper limbs the muscles responded well to both forms of current, with the exception of the right shoulder muscles. The right deltoid did not react to faradism at all, and gave a well-marked reaction of degeneration with galvanism. In the left deltoid the A. C. C. was greater than the K. C. C., but the reactions were otherwise normal. After admission the girl slowly improved, except during an attack of acute pleurisy. The anaesthesia and tenderness disappeared, and there was some general recovery of power, but when she left the hospital, after three months' stay, she was still unable to walk or raise the right arm from the side. During the next twelve months the improvement continued, but a marked lateral curvature of the spine developed, deflection to the right in the mid-thoracic region, possibly due to weakness of the spinal muscles. On examination at the present time, two years and three months from the commencement of the illness, the patient was found to have still further improved. She could walk, though not well, both legs being weak, but especially the right. The muscles of both lower limbs were rather flabby, especially on the right side, and the right leg and thigh were rather smaller than the left; no muscle was, however, completely paralyzed. The knee-jerk could not be obtained on either side. There was marked lateral curvature of the

spine, but even in this some improvement had taken place. Both upper limbs were weak, but the child was capable of performing all movements, and could even raise both arms above the head. The muscles of the right shoulder were apparently a little weaker than those of the left, but none of them were atrophied. The case, therefore, does not now present the clinical aspect of infantile paralysis, and it is quite possible that considerable further improvement may take place in the future. Not improbably the peripheral nerves have here been attacked much more than the cord.

The boy was quite well up to August 17th, but was attacked suddenly the next day, seven days after his sister was first taken ill, with feverishness and loss of power in all limbs, and was unable to sit up. He was admitted to the hospital on September 13th. He was then found to be a well-nourished boy of weak intellect with internal strabismus of both eyes, which had existed from birth. He complained of pain in the back, which was increased upon raising him in bed. There was weakness in all four limbs. He could just walk when supported; the grasps were feeble but equal on both sides; he could just raise the right arm from the bed, but was quite unable to lift the left arm at all. The knee-jerks were absent. There was no anæsthesia, but there was pain on pressure over the nerves of all the limbs and over the left shoulder, although there was no apparent swelling of the joint. During the patient's stay in the hospital he recovered power over all his muscles, except those of the left shoulder, the tenderness disappeared and the knee-jerks returned. The muscles of the left shoulder wasted rapidly and remained quite powerless. The electrical reactions of the muscles were normal except in the muscles of the left shoulder, which reacted only slightly to faradism and gave a well-marked reaction of degeneration to galvanism. At the present time the boy presents a typical picture of infantile paralysis, the left deltoid, supraspinatus, and infraspinatus having completely disappeared.

The amount of evidence that has been afforded us by the examination of patients after death is extremely small. The first to suggest that the disease involved the spinal cord and not the muscles was Heine,¹ in 1840, but it was not until Cornil² examined the spinal cords of two cases, in 1863, that there was any definite proof of this. In his cases in which death had occurred, in one a year and the other forty-seven years after the onset, he found that the ventral portion of the cord was smaller than normal, and he considered that the lesion was in the ventral white column. Prévost³ found in one case atrophy of the ventral horns of the gray matter. It is to Charcot,⁴ however, that we are indebted for the knowledge that the disease chiefly attacks the cells in the ventral horns. He examined, with Joffroy, a woman, aged

¹ Heine, J. *Beobachtungen über Lahmungs Zustände der unteren Extremitäten*. Stuttgart 1840.

² Cornil. *Compt. Rend. des Seances et mémoires de la Société de Biologie*, 1863, series 3, vol. v. p. 187.

³ Prévost and Vulpien. *Ibid.*, 1865, p. 217.

⁴ Charcot and Joffroy. *Archives de Physiol. norm. et pathol.*, 1870, vol. iii. p. 134.

forty-seven years, who in childhood had been attacked with extensive palsy of the lower limbs with resulting permanent paralysis and wasting. They found that the ventral horns in the lumbar enlargement were smaller than normal and that nearly all the cells had disappeared. There was some increase of neuroglia throughout the gray matter, especially in the ventral horns, but the atrophy of the nerve cells was much more conspicuous than the affection of the connective tissues. Charcot considered that the lesion was inflammatory in its nature, but thought that the cells were first attacked and the connective tissues only secondarily.

Rogot and Damaschino¹ were able to examine three cases within a few months of the onset of the paralysis, whereas in Charcot's case the disease had existed for many years. In these cases they found marked signs of inflammation which were naturally most marked in the most recent case, where death had taken place two months after onset. There was engorgement of the vessels with much exudation of leucocytes, and in places hemorrhages, and there was also destruction of the cells of the ventral horns with atrophy of the ventral roots, but this destruction was considered by Roger and Damaschino as being secondary to the myelitis and not primary. There followed on the publication of these cases a considerable controversy as to the nature of the pathological process, some upholding Charcot's view, that it was primarily a parenchymatous inflammation of the cells, others the view that the interstitial inflammation was primary, the cells being destroyed by involvement in this.²

Leyden,³ in 1875, from the examination of four cases, in three of which the inflammation seemed to have first affected the connective tissue, in the other the cells suggested that the clinical phenomena of infantile palsy might be caused by two different pathological lesions.

Archambault and Damaschino⁴ were the first to describe a really recent case, the previous cases having been attacked many months or years before death. In this instance death took place twenty-six days after the onset of the paralysis. Since that time several still more recent cases have been described, in one of which death took place within six hours of commencement of the illness.⁵ In all these the appearances closely resembled those described by Roger and Damaschino, and the authors considered the disease to be an inflammatory myelitis.

¹ Roger and Damaschino. *Compt. Rend. des Seances et mémoires de la Société de Biologie*, 1871, p. 49.

² Parrot and Joffroy. *Archiv. de Physiol. norm. et path.*, 1870. Roth. *Virchow's Archiv*, 1873, vol. lviii. p. 263. Schultze. *Virchow's Archiv*, 1878, vol. lxxiii. p. 443. Charlwood Turner. *Path. Soc. Trans.*, 1879, vol. xxx. p. 202. Taylor, F. *Path. Soc. Trans.*, 1879, vol. xxx. p. 197. Eisenlohr. *Deut. Archiv f. klin. Med.*, 1880, vol. xxvi.

³ Leyden. *Archiv f. Psychiat.*, 1875, vol. vi. p. 271.

⁴ Archambault and Damaschino. *Revue mensuelle des Maladies de l'Enfance*, 1883, p. 63.

⁵ Drummond. *Brain*, 1885-86, vol. viii. p. 14.

Rissler,¹ who examined four of Medin's cases, three within eight days, and the other seven weeks after the onset, is the only author who has examined recent cases who supports Charcot's view. He found, it is true, hyperæmia and cell infiltration, but considered that the nerve cell degeneration was disproportionately great compared to the amount of interstitial inflammation.

Rissler's views were criticised by Goldscheider,² who, from an examination of two cases of his own, one recent and the other of long standing, came to the conclusion that the disease was inflammatory, but primarily occurred in the distribution of the bloodvessels. He pointed out that in most cases, even including Rissler's, the lesion was not confined to the ventral horns, but spread over to the adjacent white columns or to the base of the dorsal horn.³ The ventral horns receive their blood-supply by the central branches of the anterior spinal artery, and these central branches also supply the adjacent white matter and the base of the dorsal horn. The branches of the central artery course mainly in the long axis of the cord, so that a section of the cord at any level is supplied by branches from several central arteries.⁴ Hence, if one such artery escape attack, the cells in its distribution would escape, and so different groups of cells would be affected at different levels. Kawka and Goldscheider have shown by means of serial sections that this is the case. More recently Siemerling, Dauber, and Matthes⁵ have found that the disease is inflammatory and that the inflammation occurs only in the distribution of some of the branches of the anterior spinal artery. In recent years von Kahlden⁶ has again supported Charcot's original view. von Kahlden's cases were all of long standing—two to sixty years—and so have not the same value for argument as recent cases. He founded his views mainly on the great destruction of nerve cells, with comparatively small signs of inflammation or of destruction of nerve fibres in the ventral horns. While not denying the possibility of a primary atrophy of the cells as a cause for the phenomena of infantile palsy, one may emphasize the fact that the post-mortem evidence is strongly against this view, but it is important to note that the vast majority of recent cases that have come to a post-mortem examination have belonged to the second or more common of the two clinical classes where any account of their onset is given. In

¹ Rissler. *Nordiskt. Medicinskt. Archiv*, vol. xx. Quoted by Goldscheider.

² Goldscheider. *Zeitschft. f. klin. Med.*, 1893, vol. xxiii. p. 494.

³ Roger and Damaschino. *Compt. Rend. de la Soc. Biolog.*, 1871, p. 49. Roth. *Virchow's Archiv*, vol. lviii. p. 264. Charlwood Turner. *Trans. Path. Soc.*, 1879, p. 202. Angel Money. *Trans. Path. Soc.*, 1884. Drummond. *Brain*, 1885-1886, vol. viii. p. 14. Eisenlohr. *Deut. Archiv f. klin. Med.*, 1880, vol. xxvi. Kawka. *Inaug. Dissert.*, Halle, 1889.

⁴ Kadyt. *Ueber die Blutgefässe des menschliche Rückenmarkes*, Lemberg, 1889.

⁵ Siemerling. *Archiv f. Psychiat.*, 1894, p. 265. Dauber. *Deut. Zeitschft. f. Nervheil.*, 1893, vol. iv. p. 200. Matthes. *Deut. Zeitschft. f. Nervheil.*, 1898, vol. xiii.

⁶ von Kahlden. *Beitrag. z. Path. Anat. und z. allgemein. Path. (Zeigler)*, vol. xiii. p. 113.

Matthes¹ case, however, the onset is stated to have been sudden, without previous ill-health, and yet the post-mortem appearances were similar to those of the other recent cases.

A more directly vascular origin for the disease was suggested as early as 1870 by Clifford Allbutt,² who considered that a hemorrhage into the cord might explain the symptoms.³ Allbutt's case, however, of hemorrhage into the cord had but little clinical resemblance to infantile palsy, while it closely resembled the more recently described cases of hæmatomyelia. Hemorrhage in association with myelitis is not uncommon, and small hemorrhages are frequent in infantile palsy, but are there secondary to inflammation.⁴ Primary hemorrhage is, however, extremely rare, and is almost invariably due to injury. Pain in the back and paraplegia, including paralysis of the bladder and rectum, are the most prominent symptoms.⁵ The hemorrhage does not usually take place into the ventral horns, but more commonly into the dorsal horns, and in cases where a vessel in the ventral horn has given way the blood tends to spread toward the dorsal horn and not upward or downward in the ventral horn. Goldscheider and Flatau,⁶ by injecting Berlin blue into the cords of human corpses and of living dogs, found that the ventral horns were specially resistant to such injections, the lines of least resistance for the spread of the injection being along the gray matter of the dorsal horns. Hence it may be said that it has been disproved that primary hemorrhage can be the cause of any cases of infantile paralysis, even those most sudden in their onset.

Embolism need not be considered in the absence of a source for the emboli.

Batten⁷ has recently suggested thrombosis of the vessels. After pointing out the peculiarity of the vascular supply of the cord, particularly in the lumbar region, which Moxon showed was derived from very long narrow arteries in which the resistance would obviously be great, and the blood flow therefore sluggish, he shows that the lesions are confined to the distribution of the anterior spinal artery.⁸ He further states that thrombosis, whether primary or secondary, does occur in all cases and draws attention to the similarity of the appearances found in the ventral horns of recent cases of infantile paralysis, with the red softening which is seen in the brain after thrombosis of

¹ Matthes. *Deut. Zeitschft. f. Nervheilk.*, 1898, vol. xiii.

² Clifford Allbutt. *Lancet*, 1870, vol. ii. p. 84.

³ Gowers. *Diseases of Nervous System*, vol. i. p. 868.

⁴ Charlwood Turner. *Path. Soc. Trans.*, 1879, vol. xxx. p. 202. Drummond. *Brain*, 1886-1886, vol. viii. p. 14. Dauber. *Deut. Zeitschft. f. Nervheilk.*, 1898, p. 200. Chaffey. *Path. Soc. Trans.*, 1896, p. 90.

⁵ Batten. Article on Hæmatomyelia. Clifford Allbutt's *System of Medicine*, vol. vii. p. 44.

⁶ Goldscheider and Flatau. *Zeitschft. f. klin. Med.*, 1897, vol. xxx. p. 175.

⁷ Batten. Private communication to author and *Encyclopedia Medica*.

⁸ Goldscheider and others. *Loc. cit.*

the smaller vessels, and that in cases of longer standing the condition is such as would be left after occlusion of the vessels. Thus there is present, according to Batten, a vascular peculiarity in the normal cord, favorable to thrombosis, and appearances such as would be produced by primary thrombosis are found in cases of the disease. He considers that some morbid condition of the blood just precedes the thrombosis, which condition may be due to a number of causes, *e. g.*, recent specific fever, chill, bacterial infection, etc. This theory certainly explains the sudden onset and the vascular distribution of the disease; but it is difficult to understand why thrombosis of these spinal vessels, which is very rare in adults, should be so comparatively common in children, while thrombosis elsewhere is much rarer in children than in adults. The sudden onset of the disease and its vascular distribution is as well explained by supposing that the thrombosis, which probably always occurs to a greater or less extent, is secondary to a local inflammation. Goldscheider,¹ however, in his case of thirteen days' duration, was unable to find definite thrombosis anywhere.

The remarkable special symptoms of infantile paralysis and its occasional occurrence in epidemic form have naturally led observers to search for some specific organism as the cause, but in spite of careful search, Rissler, Goldscheider, Siemerling, Dauber, and others found no organism to account for the disease, and obtained no growth in cultivation experiments. Two recent observers, however, have succeeded in finding organisms in cases of infantile palsy which were similar in appearance to one another and possibly identical. Schultze² examined some fluid obtained from the meninges by lumbar puncture in a case which he considered to be infantile paralysis. The pressure of this cerebro-spinal fluid was decidedly increased, but the fluid was clear except for a few specks. In the fluid were found diplococci in chains or tetrads, which were very like the Weichselbaum-Jager meningococcus. No growth was obtained, however, of the organism. Two years after Schultze reported his case, Chapin³ described a small epidemic, in Poughkeepsie, of anterior poliomyelitis and polyneuritis. In one of the cases, which after death showed the ordinary appearances of anterior poliomyelitis, some diplococci were found by Brooks in the ventral horn of the cord, but none were found in the meninges or blood. In the second case, similar diplococci were found in the blood during life. Here also no growth was obtained. This discovery of the presence of a definite organism in the diseased parts or in the blood is not sufficient for us to accept such organisms as the cause of the complaint. The discovery, however, is important and suggestive,

¹ Goldscheider. *Zeitschft. f. klin. Med.*, 1893, vol. xxiii. p. 509.

² Schultze, F. *Munch. med. Wochenschrift*, 1898, vol. xxxviii. p. 1197.

³ Chapin. *Archives of Pediatrics*, November, 1900.

and further researches may find that this organism is constantly present in all cases or in all those of a particular type, and may further show that it can be grown outside the body and its life history studied.

From the evidence at our disposal it is impossible to come to any definite conclusion as to the exact nature of the disease. Clinical records are strongly in favor of at least two, and probably three or four, definite diseases being included under the term anterior poliomyelitis, viz.: 1, that class where the paralysis comes on suddenly without previous ill-health; 2, that class where the onset of the paralysis is preceded by general symptoms, such as fever, vomiting, pain in the back, etc.; 3, the epidemic class, and 4, the adult class. The three latter may possibly be the same disease which may sometimes occur sporadically, and at other times in the form of an epidemic. Occasionally cases occur sporadically which are like the epidemic cases, the disease having spread to the nerve centres in the brain and to the peripheral nerves, and in some of these death even has ensued.

The arguments against regarding hemorrhage into the gray matter of the cord as the cause of any of the cases of infantile paralysis have already been given, and the question of thrombosis has been discussed. In spite, therefore, of the fact that a purely vascular lesion would best explain these striking cases where the onset is absolutely sudden without other sign of ill-health, one is compelled to regard such a cause as improbable. Of the two other possible ways by which the cells in the ventral horns might be destroyed, viz.: a local inflammation or a primary degeneration of the nerve cells, it is impossible at present to quite ignore the latter. An acute degeneration of the nerve cells might be brought about by some poison circulating in the blood, and such a poison might be formed by the growth of the micro-organisms which are the causes of such diseases as scarlet fever, or measles, or by the growth of a micro-organism which is specific to infantile paralysis. Such a primary degeneration of the nerve cells is a possible cause for some cases of infantile paralysis, but the evidence in its favor is very inconclusive. The one definite fact obtainable from the post-mortem examination of patients that have suffered from the disease is that in all recent cases there are signs of a local inflammation in the ventral horns. That these appearances are inflammatory and not due to vascular occlusion is rendered the more probable by the fact that the inflammation sometimes involves other parts than the ventral horns. For instance, Schultze¹ found inflammation of the meninges in an otherwise typical case, and he considered from clinical evidence that the association of meningitis with anterior poliomyelitis was not uncommon. The inflammation, as a rule, however, seems to be confined to the distribu-

¹ Schultze. Münch. med. Wochenschrift., 1896, No. 28, p. 1197.

tion of the anterior spinal artery, and doubtless in the majority of the cases thrombosis occurs very early and so explains the sudden or very rapid loss of power. This local inflammation, like so many inflammations not due to direct injury, is probably caused by a local growth of some micro-organism. No such organism has yet been isolated, and, apart from the observations of Schultze and Chapin, there is no direct evidence of its existence; but the clinical aspects of the disease, as well as the post-mortem appearances, are strongly in favor of this theory. It is possible that the hitherto unisolated organisms that cause measles and scarlet fever might start a local growth, and so a local inflammation in the ventral horn, but the majority of cases of infantile paralysis are not preceded by any acute illness or, indeed, anything that can be regarded as an exciting or predisposing cause. The reputed association with the acute specific fevers would be better explained by supposing that there is a weakened resistance to the attack of other organisms left after these complaints, and such a lessening of resistance would also explain the undoubted frequency with which exposure to chill, damp, or fatigue precedes an attack of acute anterior poliomyelitis in the adult. It is probable, therefore, that the organism associated with anterior poliomyelitis is specific to that disease and is not concerned in the causation of any other morbid condition. Since there seem to be two or more definite diseases included under the name of infantile palsy, there would probably be two or more specific organisms. Beyond this it is at present impossible to go.

REPORT OF A CASE OF ALCOHOLIC MULTIPLE NEURITIS.

BY L. W. ATLEE, M.D.,
OF PHILADELPHIA.

CASES of multiple neuritis due to any one of the several etiological factors are not of infrequent occurrence in the attention of those having special facilities for observation in this class of disease; but to the general practitioner they are so rare as to be seldom or never met with, even in an active practice, and unless he has recently been looking up the subject-matter describing the disease he is likely to be much confused and led into error in his diagnosis when confronted with a case. In a great majority of all cases there are very definite symptoms, but the disease presents a very diverse clinical picture, though certain general features stand out with special prominence.

In the following case the initial rheumatoid pains in the extremities were looked upon by the family physician as due to "rheumatism;" later, the numbness and tingling and slight loss of power in the hands

and feet were viewed as being produced by some obscure spinal disease; and later again, as the disease advanced, the ataxic gait seen in this form of pseudo-tabes led to a diagnosis of true tabes; and at a still later stage of the disease the expansive delirium and motor weakness led a specialist to diagnose the disease as general paralysis.

J. C., aged forty-four years; native of Philadelphia; parents Irish. He is a widower and the father of five healthy children. He is an active working member of a morocco leather firm, and alone was possessed of the secrets of the chemical mixtures used in the process. (His being concerned with chemicals is mentioned, owing to the fact that when he was first seen it was a matter for consideration how much influence lead or arsenic might be having in the production of the disease. This was, of course, prior to having elicited a full history of the case.) When first seen by me, a few days after the specialist had diagnosed general paresis, he was in bed; and as he could give no rational replies to my questions it was, to say the least, most difficult to give to each symptom its proper importance and arrive at a diagnosis. The man was fairly well-nourished looking, the skin moist and greasy; the face was flushed and dilated vessels noticeable over the nose and malar regions. His pulse was 104; temperature in the mouth, 99.5° F. His tongue was heavily coated; the gums soft, spongy, and easily made to bleed. Nothing abnormal could be found in the thoracic organs. The liver was very much enlarged and its edge sharp and hard. His urine showed a heavy deposit of urates and gave a decided haze on boiling and with the contact test. The urea was normal in amount, and there were some hyaline tube-casts in the sediment.

The small muscles of the thumb and fingers were markedly atrophied, as shown by the shrunken appearance of the tissue between the metacarpal bones; the muscles at the back of the forearm concerned in extension of the hand and fingers were also atrophied. These muscles in the palm and forearm were tender on deep pressure, as were also the muscles of the calf and of the sole of the foot. The knee-jerk was absent; the cutaneous reflexes were unaffected. The pupil reflexes were normal; the vesical and rectal sphincters were normal also.

The psychical symptoms were remarkable. He did not at all appreciate his condition, and at times was unconscious of his environment, though he recognized the people about him and could express his wants. When engaged in conversation there was noticeable a sort of silliness—a “childish jocularity”—in what he said. He had delusions of various kinds, at times talking loudly to people he believed to be present, particularly to his wife, who had been dead several years; but most of his delusions were connected with his work at the shop, and he was continually giving orders to the workmen. He did a great deal of travelling in his delirium, and when asked “How are you this morning?” would reply, “Oh, I’m all right to-day—only this soreness in my legs,” and then he would branch off suddenly and tell how he had been to Pittsburg or St. Louis to see the “ball game.”

He slept very little at night—it was his most delusional and excitable time of the twenty-four hours—but during the day he dozed a good deal. There were two beds in the room, and he frequently moved from one to the other; at times he sat on the edge of the bed, and sometimes he walked through the hall and into the back room. After a good deal

of persuasion he was induced to stand up and to walk. He could do neither with his eyes covered. His gait was very like that of tabes, but his legs were evidently weak from some motor paralysis, though there was no decided "foot-drop." There was nothing remarkable about the man's articulation or manner of speaking, no "scanning," etc.

The history of this case had to be entirely obtained from the relatives, and at first it was impossible to find out how much alcohol had been imbibed as a constant practice all these years; but by dint of interrogating everybody who knew anything about him it became apparent that the patient had been constantly using alcohol for the past eight years at least, although it was an exceedingly exceptional occurrence for him to become what is commonly known as drunk. He never, in fact, missed a day at his work in all this period until his present illness. Some five months ago he began to lose his appetite and to sleep badly; for this he used more spirits, drinking at night when he awoke. At this time he began to complain of numbness and tingling in his hands and feet, and weakness in his legs; and then the feet and hands commenced to swell when they were left in a dependent position for any length of time. Later he had trouble in getting about from the unmanageability of his legs; in other words, the gait became ataxic, as described by those associated with him. The mental condition now was much changed; he became irritable and his memory defective, as shown in forgetting business engagements and the frequency with which he left in the trolley cars a small grip he carried to and from the shop.

In the early stage of the disease the boring pains in the limbs had been regarded as rheumatic and had been treated as such, and when the parietic symptoms became prominent he had been subjected to "electric treatment." This had greatly increased the pains in the limbs, and had been discontinued a few days before he was first seen by me, and also owing to the fact that at the time the disease had been looked upon as general paralysis, and a conformable prognosis given. The electric reaction of the affected muscles was not taken.

After having had the case under observation for a few days and obtained a more candid history of the man's habits, the condition was looked upon as entirely due to multiple neuritis of alcoholic origin, and a more hopeful prognosis given.

The treatment was directed entirely to the correction of the defective general health. All alcohol was withheld, for up to this time he was allowed it in considerable quantities. It was not difficult to do this, owing to the man's mental and physical condition. He was fed frequently with milk and lime-water, strong broths and beef-juice, and gradually his digestion improved sufficiently to take lightly broiled meats and soft-boiled eggs. Mouth-washes corrected the purulent, ulcerating gingivitis and aphthous condition of the buccal mucosa. Bitter tonics, mineral acids, and strychnine were the principal medicinal treatment. When he became very much excited he was given valerianate of ammonia and bromide of sodium. Hyoscine hydrobromate was tried at first, but produced terrible depression, the respiration becoming very shallow and rapid and the heart action weak. For the sleeplessness trional acted excellently and without any apparent after-effect.

At the end of three months' treatment his general health was quite good. All the animal functions were well performed, the muscular power was greatly restored, though the gait was still somewhat ataxic,

and deep pressure into the calf muscles still elicited painful sensations. His mental condition had also changed. When first seen he frequently remained quiet and apathetic for hours, but now he was very irritable and easily excited. At times he perfectly realized his condition and became much depressed, but much of the time he was under the influence of delusions, particularly of persecution—he was being poisoned, he was being bullied and interfered with in the exercise of his will, strange people were being harbored in his house by his relatives; he was continually searching under the bed, in the closets, etc., for these people. At night he was very violent, and as he had now improved so much in general health and muscular power he was difficult to control, and he became personally abusive to those having the constant care of him, so that it was necessary to send him where he could be under surveillance. For this reason he was sent to an institution for the care of the alienated. The following report of his condition after some two months' treatment at this place is very satisfactory: "I am pleased to inform you that J. C. has very much improved in mind of late, and his present condition gives encouragement that he may get well enough to return to his home in the near future. While his mind is comparatively clear, he is still rather delusional; but if he does not suffer a relapse I am in hopes his mind will continue to clear up. His general health is fairly good."

There are few diseases in which an early diagnosis is of greater importance than in multiple neuritis of alcoholic origin. During the *stadium incrementi* prompt treatment would save many patients from months of suffering and disability, since the removal of the cause insures with certainty, during a gradual onset, a quick removal of the symptoms, whereas its effect on the developed disease is manifested much more slowly (Gowers). In forming our diagnosis, if all the symptoms are considered separately for their individual value, and conjointly for their associated significance, it is not often the observer will remain in doubt.

By far the greater number of cases of this disease are slow in their onset unless some other factor is brought into play, as exposure to cold, etc. In the early stages the tingling and numbness in the fingers and toes, palms and soles, and the deep muscular pains, are frequently attributed to "rheumatism." Later, the symmetrical weakness of the extensor muscles of the hand and foot, with hyperæsthesia and deep tenderness, add strength to the diagnosis of multiple neuritis. From the predominance of particular symptoms the disease is divided into a *motor*, *sensory*, and *tabetic* form. The violence of the disease expresses itself particularly in the distribution of the radial and peroneal nerves, which are what is designated as homologous nerves by physiologists.

We must bear in mind that the disease does not often present itself in a classical, stereotyped form; as, for instance, a case may show the feet severely paralyzed and the hands present but sensory symptoms; and although loss of power is the obtrusive feature, some loss of co-ordi-

nation usually accompanies it, and is often the means by which the attention of the patient is first directed to the disability. He finds a difficulty in standing or in performing the finer movements of the fingers; then he may notice for the first time that he has lost power in extending the hand and fingers, or, when walking, to raise the toes from the ground. Whenever there is weakness of the legs the knee-jerk cannot be obtained. "In very rare cases this may persist in a slight degree."¹

The bladder or rectum are very rarely affected in their innervation in the disturbance caused by polyneuritis. When such symptoms do exist they should suggest involvement of the cord also.

Though the stress of the affection falls on the distribution of the radial and peroneal nerves, the other muscles of the extremities become affected; least and last those about the shoulder and hip. Only in the most extremely severe cases are the muscles of the trunk affected, the diaphragm, and the muscles of the thorax and abdomen; very rarely the facial muscles or those of the tongue; but sometimes the condition of the vocal cords, the rapidity of the pulse, and defective lung circulation suggest affection of the vagus.

Flabbiness and wasting of the muscles are early and marked conditions, and the change in the electric irritability also is early, giving the reaction of degeneration.

The disturbance of sensory nerves is very variable. It is most intense at the ends of the extremities. It usually shows itself in a lessening of the sense of touch and an increase of the sense of pain on stronger pressure. The temperature sense is unchanged.

In the ataxic form (of which alcohol is the most frequent cause) inco-ordination is the chief symptom, and is attributed to affection of the muscle afferent nerves. The inco-ordination so closely resembles that of tabes that it has received the name of *pseudo-tabes* (Gowers), for the pains in the limbs and the loss of the knee-jerk make the resemblance very striking. The ataxia may involve the arms as well as the inferior extremities, but is not nearly so common.

Reflex action from the skin varies much in its condition. In severe cases with much motor and sensory loss it is usually absent.

In prolonged cases we may find trophic changes in the nails, skin, and hairs. These changes are similar to those that occur in ordinary neuritis—glossy skin, arthritic adhesions, and thickening being the most common. Vasomotor disturbances are common, and show themselves in cedema about the ankles, back of the foot, and at the wrist and back of the hand.

The symptoms of multiple neuritis closely resemble those of acute and

¹ Gowers. *Diseases of the Nervous System*, vol. 1. p. 155.

subacute inflammation of the gray matter of the cord—poliomyelitis—but not so much in the alcoholic form as in that due to toxæmias or cold. These symptoms—febrile onset, muscular wasting, and reaction of degeneration, with a tendency to spontaneous recovery, and the initial rheumatic pains—are confusing. In multiple neuritis the localization of the palsy is symmetrical, while in poliomyelitis it is random; in the former we have persistence of the nerve pains, tenderness of the inflamed nerve trunks, and changes in the sensibility—never present in poliomyelitis. The presence of increased myotatic irritability, excessive knee-jerk, and foot clonus would mean disease of the cord.

Pachymeningitis, by damaging the nerve roots, may cause paralysis, wasting, and anæsthesia; but it is rare in this disease to find all four limbs affected. "The legs rarely suffer" (Gowers). The anæsthesia is found in the upper parts of the limbs and the trunk even more than in the distal parts. The nerve trunks are not tender.

In diphtheritic paralysis the disease usually attacks the palate and ciliary muscles first, and there is little if any pain.

The distinction of multiple neuritis from true tabes is often made with great difficulty, especially in that form of neuritis in which the sensory symptoms are predominant and the paralytic symptoms practically absent. It is easy to decide the question when we have distinct loss of power in the extension in addition to the inco-ordination, since there is no actual loss of power in tabes. This should decide the question. In the pseudo-tabetic form of polyneuritis we may have distinct inco-ordination on walking, increased by closure of the eyes, and distinct irregularity in the movements of the feet and legs. These cases perfectly simulate tabes. Pains are common to the two diseases—both acute and prolonged pain and dull rheumatoid—but true "lightning" pains are seldom met with in neuritis. The muscular tenderness is of value as to its being neuritis. In the latter there is no affection of the sphincters, no sense of constriction around the trunk; and lastly, of great importance, the reflex action of the pupil to light is not lost, or so rarely as to be considered so. In neuritis there is no optic-nerve atrophy and no "crises." "Care must be taken not to confound the vomiting of alcoholic gastric disturbance with the gastric crises of tabes" (Gowers). The trophic changes of the two diseases differ. The tabetic enlargement of bones and diseases of the joints are unknown in neuritis, while in the latter we have the "glossy skin" and arthritic adhesions, not found in tabes.

In distinguishing multiple neuritis from hysterical palsies the loss of power of extension of the wrist is of great importance, as it is never seen in hysteria.

Multiple neuritis should not give rise to difficulty in its diagnosis from "general paralysis." The peculiar distribution of the motor and sensory symptoms and the loss of the knee-jerk in neuritis are conclu-

sive, the only confusing symptom being the "expansive delirium" sometimes seen in the alcoholic cases.

Severe forms of alcoholic origin when seen late, with damaged kidneys, severe chronic gastric catarrh, and mental torpor, may at first give rise to diagnostic difficulties, as in these cases uræmic symptoms or chronic cerebral meningitis may exist as complications; but time and the bearing of this in mind, and the critical weighing of the history and symptoms, should usually lead to a correct appreciation of the condition.

NOTE.—This man has since returned to his home, and is entirely recovered from all symptoms of this disease.

HEPATIC LESIONS IN INFANCY.

BY MARTHA WOLLSTEIN, M.D.,

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THE pathology of the liver in infants and young children has not been fully elucidated. A study of 370 consecutive autopsies, made chiefly at the Babies' Hospital, with a few from the Infants' Hospital, on Randall's Island, offers some interesting facts. The ages of the cases studied ranged from two days to four years, but the majority (193) were under six months old; 287 were less than one year of age, and only 31 were over eighteen months. The lesion most frequently encountered was fatty infiltration, which was apparent on gross examination in 214 cases, or 58 per cent., including 45 cases which were tuberculous as well as fatty. Of the remainder, 22 showed tubercles without fatty change, 2 were cirrhotic, 85 deeply congested, and only 47 were normal to the naked eye.

The condition of the infants was noted at the autopsy table as follows: 80, or 22 per cent., were well nourished; 93, or 25 per cent., were fairly nourished; and 197, or 53 per cent., were emaciated.

Fatty Livers. Leaving the 45 tuberculous cases for consideration later, we have 169 simple cases of fatty liver, of which 133 were ordinarily fatty, and 36 extremely so; 80 cases, or 48 per cent., occurred in emaciated infants; 51, or 30 per cent., in fairly well-nourished children, and 38, or 22 per cent., in well-nourished ones. It will be seen, therefore, that the cases of fatty liver occurring in non-tuberculous, emaciated subjects, was 10 per cent. less than the whole number of fatty livers in the autopsy series. Of the extremely fatty livers, 39 per cent. occurred in well-nourished children, and an equal number in emaciated infants; while only 18 per cent. of the ordinarily fatty ones were well nourished, and 50 per cent. were emaciated. So that an extreme degree of fatty infiltration was decidedly more common in

well-nourished subjects, and an ordinary degree of fatty change was somewhat more common in emaciated cases.

Of the extreme cases, 10 were under three months of age, 29 under one year, and 1 over two years. Of the ordinary cases, 26 were less than three months old, 109 were under one year, 19 between one and two years, and 4 over two years.

It is interesting to note the clinical and anatomical diagnosis in these cases. Of *marasmus* there were 97. Considering these in detail, we find that in 22 instances in which no gross anatomical lesion was present at autopsy, the liver was not the seat of fatty change; that in 30 other cases no fatty change had occurred in spite of the presence of pneumonia, 17; enterocolitis, 10; and atelectasis, 3. The pneumonia in these cases was, however, of the hypostatic variety only, and superficial in distribution. The intestinal lesion was merely a mild catarrh, and in no case accompanied by ulceration nor deep-seated infiltration. On the other hand, fatty livers were found in 45 *marasmus* cases, 46 per cent.; accompanied by bronchitis, 3; malaria and vulvovaginitis, 1; enterocolitis, 14, and bronchopneumonia, 27. These figures bear out Freeman's¹ statement that simple *marasmus*, unless complicated by an acute disease, is not accompanied by fatty change in the liver.

There were among the whole number of autopsies 67 cases of *tuberculosis*, from two months to three years of age, 29 being under one year and six over two years. Of these, 45 had livers showing both fatty infiltration and tubercles in smaller or larger number; while in 22 no fatty change had occurred. Thus, 67 per cent. of the tuberculous cases were accompanied by fatty liver, while only 55 per cent. of the simple cases showed this lesion. These numbers differ somewhat from Dr. Freeman's; he found that fatty livers do not occur more often with tuberculosis than with other conditions. Among the tuberculous cases were 21 well-nourished infants, of whom 16, or 76 per cent., had fatty livers; 13 fairly nourished children with 8 fatty livers, or 62 per cent.; and 33 emaciated subjects with 20, or 61 per cent. These conditions are directly opposed to those of the simple cases in which the well-nourished infants showed the smallest per cent. of fatty livers, and emaciated infants the highest.

Eleven livers were extremely fatty, 55 per cent. of them occurring in well-nourished cases, and 36 in emaciated ones. Of the ordinarily fatty ones 29 per cent. were well nourished, and 49 per cent. were emaciated. As in the simple cases, the emaciated infants were more prone to fatty change of the ordinary degree, and the well-nourished tuberculous children were rather more liable to extreme fatty infiltration of the liver.

¹ Transactions of the American Pediatric Society, vol. II., 1899.

Four of the 10 cases of *rhachitis* were accompanied by fatty liver, the cause of death having been bronchopneumonia in 3, and enterocolitis in 1. While in the remaining 6, which were without fatty change, bronchopneumonia was found three times, bronchitis once, enterocolitis once, and enterocolitis with pachymeningitis interna hæmorrhagica once.

In 14 cases of *congenital syphilis* the liver was fatty in 8, all of which were complicated by acute bronchopneumonia, accompanied in 1 case by multiple gummata throughout both lungs. In 1 case, three months old, with erysipelas of the face and pneumonia, the liver was found to be dark-colored and rather soft; it contained a number of small, round, lighter, depressed areas surrounded by a zone of congestion. Microscopically there was intense congestion, with consequent pressure atrophy of many liver cells, and scattered areas of young, cellular connective tissue throughout the organ. Hochsinger¹ says the typical form of syphilitic hepatic disease in infancy is an acute diffuse infiltration with young cellular connective tissue, and that true nodular gummata are rare at this age. No hepatic gummata were encountered in our cases. Only 3 of the 14 cases reached the age of seven, thirteen, and nineteen months, respectively, all the others being under the age of four months—again bearing out Hochsinger's statement that the fatal cases die early. Five of his 16 were less than three months old.

Of 68 cases of *pneumonia*, 45, or 66 per cent., had fatty livers, a decided increase over Freeman's figures; he found only 35 per cent. so affected.

Grouping all the various intestinal lesions under the head of enterocolitis, there were 57 cases with 36 fatty livers, or 63 per cent.

With the acute suppurative inflammation of the serous membranes, fatty infiltration of the liver was very common, occurring in 5 of 7 cases of simple meningitis, 3 of 4 cases of empyema, 2 of 4 cases of peritonitis, and 1 of 2 cases of pericarditis. In 3 cases of pyæmia, and 1 of furunculosis the livers were fatty.

In 2 of 3 cases of measles, all dying of pneumonia, the fatty change occurred. Areas of necrosis were not present. In 1 of the 2 fatal diphtheria cases, 2 cases of influenza, and 2 of malaria (1 dying of acute bronchopneumonia, the other of marasmus) it was also present.

Two cases of sclerema were accompanied by very marked fatty infiltration of the liver.

Thus in the present series fatty livers occurred most often with suppurative inflammations, next in frequency with tuberculosis, pneumonia, and the intestinal diseases. It was never found in uncomplicated marasmus and was inconstant both in syphilis and rhachitis, of which no uncomplicated cases are among our records. Neither disease seemed

¹ Wien. med. Wochens., 1892, Bd. 46.

to cause a predisposition to fatty infiltration of the liver. It is to the acute infections that the change in this series of cases must be ascribed.

Cirrhosis. One liver was the seat of hypertrophic cirrhosis. It was the case of a female infant, three months old, with a typical syphilitic eruption, fissured lips, and snuffles. She was fairly well nourished, and entered the hospital for vomiting and diarrhoea. The liver extended 3 cm. below the free border of the ribs, and the spleen was also enlarged. There was no icterus at any time. At the autopsy the liver weighed 205 grammes, was decidedly enlarged, and cut with difficulty, owing to the presence of an increased amount of connective tissue. Microscopically there was a decided increase in the interlobular connective tissue and less of the intralobular. This connective tissue was partly of the cellular, and partly of the older, fibrous variety. The liver cells were granular, and, in places, the seat of pressure atrophy. The gall-ducts were normal in structure and not increased in number. The walls of the small bloodvessels were thickened, the adventitia and the intima being involved. The spleen weighed 150 grammes; it was firm, and the capsule showed chronic perisplenitis. Microscopically hyperplasia was marked.

The only other case of cirrhosis encountered in this series of autopsies was one due to obstruction of the bile-ducts in an infant dying at the age of three months. It is reported elsewhere.¹

No case of waxy degeneration occurred in these young children.

THE SACROCOCCYGEAL DIMPLES, SINUSES, AND CYSTS.

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THE purpose of this paper is to present a preliminary consideration of the congenital dimples, sinuses, and dermoid cysts found in the median line of the back over the coccyx and lower part of the sacrum, together with a more or less complete review of the literature of the subject to the year 1901.

The presence of these congenital defects occurring in a far larger number of cases than is ordinarily believed has received as yet comparatively little attention from clinicians, having been remarked only

¹ Archives of Pediatrics, March, 1902.

as a rule when some surgical complication, to which the deeper of these structures (the sinuses and cysts) are especially liable, has made a careful examination necessary.

The French authors were the first to regard the differences in development of the sacral region of the trunk, and, therefore, to note those longitudinal furrows or rounded depressions of greater or less depth, found in a large proportion of babies and children and smaller proportion of adults.

A simple examination of adults or children, but especially of babies, by the mere separation of the buttocks, will often show over the coccyx

FIG. 1.



Representing the most common appearance, the shallow dimple. Drawn from ten-days-old baby. Corresponds to A, Fig. 4.

above the anus, some form or degree of dimpling or indentation of the skin, either the longitudinal furrowing or rounded depression, with less frequently a sharply marked deep pit indicating the mouth of a fistula or sinus. (See Figs. 1, 2, and 3.) Nor is it very uncommon to find several members of one family showing the same condition. Such cases have been reported by Madelung and Dunlop.¹

The integument within and immediately about these little defects usually shows slightly more gloss, is pinker than that over the rest of

FIG. 2.



Representing the longitudinal furrowing. Drawn from baby about one week old.

FIG. 3.



Representing the external appearance of the less frequently found sinus. This may be only a centimetre deep or several centimetres, and may or may not be sacculated at its end. Corresponds to the external appearance of B, C, or D, Fig. 4.

the part, and is moister. From the deeper ones, indeed, clothing in contact with the skin may be decidedly dampened, as instanced in some of the cases of J. M. Warren¹ and others. Pulling upon the skin about these depressions or putting it upon the stretch with the finger demonstrates more than the usual resistance to its gliding on the deeper structures, and at the same time causes an exaggeration of the dimpling, showing an adherence or tacking down to the less movable parts beneath. Hodges,² among others, has written of this. It can often be shown where there is but the merest dimpling present, one so slight as to escape ordinary observation. It is to be regarded as quite characteristic of these depressions.

In size and depth they may range from the faintest indentation through sharper and deeper pit-like formations to sinuses of many centimetres depth, possessing the finest orifice or one so large as to be mistaken for the anus itself, as happened in a case reported by Lannelongue.³ Or we may find over this sacrococcygeal area a completely sequestered pouch, an epidermoid cyst, with or without outward mani-

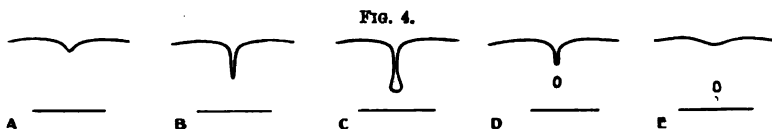


FIG. 4.
Diagrammatic sketch of the dimples, sinuses, and cysts in coronal section, and showing the more usual varieties met with. Straight line indicates bone beneath either of sacrum or coccyx; curved line, the skin surface of the buttocks.

festation of the same. Often one or more fistulae will be found to lead into a cystic formation or sacculated pouching, such as described by Wendelstadt.⁴ In fact, "we can demonstrate all possible transitions from the open inversions of the external skin, such as not infrequently occur in the neck in the form of cleft or sinus-like dermoids, to such as have only a very narrow communication with the skin, and finally such as are wholly cut off, as was first demonstrated by Heschl."⁵ (See Fig. 4.)

The occurrence of these congenital defects observed in a study of a great number of subjects shows the largest proportion to exist in young babies, and the least in adults. Lannelongue gives 20 to 25 per cent. of all young children. Despres,⁶ 30 to 33 per cent. at birth. Heurtaux,⁷ 4 to 5 per cent. of adults on a basis of 960 cases examined.

¹ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1854; Philadelphia Medical Times 1878; Boston Medical and Surgical Journal, 1877, and "Surgical Observations."

² Boston Medical and Surgical Journal, 1882.

³ Bull. Méd., Paris, 1889; Bull. Mém. Soc. de Chir., 1882-1886.

⁴ Dissertation, Bonn, 1865.

⁵ Prager Vierteljahrschr., 1860.

⁶ Bull. de la Soc. Anat., 1874; Bull. et Mém. Soc. de la Chir., 1889, vol. iv.

⁷ Bull. et Mém. Soc. de Chir., 1882.

Lawson Tait,¹ 23 per cent. of women examined at the Birmingham Hospital for Women, which would certainly seem a high percentage for adults. Peyramaure-Duverdier,² about 3 per cent. of persons examined. Kulm and Wendelstadt give a somewhat higher percentage. In a series of 300 consecutive births observed in this city, a well-marked dimple or sinus occurred in 100 out of the 300 babies. The series was an absolutely consecutive one, and therefore gives very accurately the true proportion of cases presenting these anomalies.

As regards their situation, it may be noticed anywhere from the anus to well up on the sacrum and always exactly in the middle line. Usually they are situated over the lower part of the coccyx, rarely closer than 1 centimetre to the anus. Mallory³ gives the situation in order of frequency as :

1. Near tip of coccyx.
2. Sacrococcygeal region.
3. Over lower part of sacrum.
4. Between coccyx and anus.

Nicaise⁴ says they occur in three principal places, dorsum of sacrum, sacrococcygeal junction, and tip of coccyx. Lannelongue found in 95 observations that 29 were situated over the sacrum, 38 at the junction of the sacrum and coccyx, and 28 at the tip of the coccyx. Despres believes that they are commoner between the coccyx and the anus, while Lawson Tait believes he has seen the greater number at the upper end of the fold between the nates over the sacrum.

In the series here reported the average distance was 1.66 centimetres above the anus, agreeing more closely with the statistics of Mallory. The nearest to the anus was 1 centimetre, the most distant 3½ centimetres.

There is more often but one dimpling or sinus noted, but occasionally two, more rarely three or more. Lannelongue reports those with three openings. Lamadrid mentions one with three openings, while Heurtaux reports one with three and another case with four congenital openings of sinuses in the middle line of the back. One depression was found ninety-two times, two depressions eight times. Those with three or four were not noted in the above series.

The "depression" of the French, or dimple, or fovea, is by far the most frequent and simplest of the three abnormal formations, i. e., foveæ coccygeæ, fistulæ coccygeæ, and cysticæ coccygeæ.⁵

The sinus is much less frequent. In the 100 cases referred to the foveæ occurred eighty-nine times, while the fistulæ occurred but eleven

¹ Publication, Dublin.

² Thèse de Paris, 1882.

³ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1892.

⁴ Tribune Médicale, 1893.

⁵ Wette's Classification, Langenbeck's Archiv, 47.

times. Of the relative proportion of the cyst occurrence we can as yet say nothing for lack of sufficient data and histological examination.

By the older writers, before the days of microscopical examination and before any serious attempt at explanation for their occurrence was made, but when the surgical complications of certain of these structures formed subject-matter for literature, they were usually described as "pilous cysts," or "pilonidal sinuses;" or they were styled "furuncles," "abscesses," or "phlegmons," according to the nature of the pathological process occurring and in which they were seen.

The study of these foveæ, fistulæ, and cysticæ is of comparatively recent date, beginning about the year 1868. Courand,¹ reviewing the literature of the subject to the year 1883, declares that he found but fourteen reported cases.

The tumors of this region, on the other hand, were studied many years previously. Meckel and Himly, in 1818, wrote upon fetal inclusions and parasitic monsters. Ammon, in 1842, Wernher, in 1843, and Velig, at Strassbourg, 1846, Lotzbeck, in 1858, etc., contributed to the literature. One of the first reported cases of sinuses was that of Anderson,² in 1847, under the heading, "Hair Extracted from an Ulcer."

Gross,³ in 1882, speaks of a "congenital pilocystic tumor" sometimes met with upon the coccyx in the vicinity of the anus. "Its origin is apparently connected with a sebaceous follicle, which, during its development, intercepts a small tuft of hair very much as in certain sebaceous formations on the head and face." Vaughan,⁴ in 1865, considers their origin involved in obscurity. He reports three cases of "pilous cysts." Lamadrid,⁵ 1872, considers them due to imperfect coalescence of the lateral halves of the body. "It is loose hairs which work their way down these preformed sinuses, which cause the irritation and inflammation often observed." J. M. Warren, in his *Surgical Observations*, also considers their origin obscure. Hodges, in 1880, believed as did Lamadrid, that hairs from the surface were the primary source of trouble. Heurtaux, in 1882, believes that epithelial detritus and sebaceous accumulations take place in these congenital infundibula. "This retention can become the cause in certain cases of suppurative inflammation of the skin which forms the bottom of the sinus; the skin becoming ulcerated and perforated, pus gradually infiltrates the subcutaneous tissue and gives rise to the fistulous tract."

What bothered the older observers was the presence of the coil of hair or accumulation which they usually found at the bottom of the sinuses and sacculated dilatations in their suppurative cases. As the

¹ Thèse de Paris, 1883.

² Boston Medical and Surgical Journal, 1847.

³ System of Surgery, vol. II.

⁴ St. Louis Medical and Surgical Journal, 1865.

⁵ Philadelphia Medical Times, 1878.

epithelial, sebaceous, and hair-bearing lining was destroyed by the inflammation and often impossible of identification, they had to imagine the strands of hair working down from outside, quite in such a way "as hair-balls are formed in the stomachs of cattle," one writer states. Hodges gives three conditions as essential, "a congenital dimple or sinus, pilous development, and lack of cleanliness."

If no hair was found it was the sebaceous and epithelial accumulation, with little or no means of exit, that caused the trouble. Walker,¹ in 1875, reported a case of "Monstrosity by Inclusion." A sinus over the coccyx was laid open and a coil of light hair found therein. Fere,² in 1882, thinks the dimples and sinuses due to incomplete closure of the "posterior umbilicus," i. e., the lower end of the medullary tube. Terrillon,³ in 1882, considers them due to a special form of spina bifida involving the skin only. Lawson Tait, suggested the "coccygeal dimple" as the "cicatrix of the spina bifida by which the human tail has been lost." Lannelongue, in 1882, believes that "after the medullary canal is formed, the mesoblast passes back between the vertebral column and the external epidermis, except in the region of the sacrum, where little of this tissue is interposed, so that this region is reduced to epidermis and bone. Consequently the superficial layer, the epiblast, joined at a later period to the mesoblast, preserves closer relations with the bone, and at one or more points the skin may be bound down to the bone, and later, when the subcutaneous tissue is developed around these places, a depression will be formed. If deep and narrow enough, the orifice may close up and a dermoid cyst will be the consequence." Wendelstadt, in 1885, expresses the same idea and opinion.

As these congenital dimples and sinuses were not confined in their occurrence to the sacrococcygeal region, but were not infrequently observed in the same localities as the dermoid cysts of regions in which embryonal parts joined, such as the neck, face, roof of the mouth, back of the head, and along the spine, and even in conjunction with these cysts (see the cases of Guzman,⁴ Sutton,⁵ Stelwagon,⁶ Barling,⁷ Wagstaffe, etc.), the idea of imperfect closure of the lateral halves of the body or epiblastic involution was usually adduced to explain them. This was the generally accepted explanation up to the time of the article by Tourneux and Herrmann,⁸ in 1887, upon the remains of the spinal cord or medullary vestiges in this region.

Tourneux and Herrmann, finding remnants of the spinal cord rudiment behind the coccyx in the fœtus, in a study of this region, believed

¹ Virginia Medical Monthly, 1875.

² Bull. et Mém. Soc. de Chir., 1882.

³ Journal of Anatomy and Physiology, London, 1886.

⁴ Philadelphia Medical Times, 1883.

⁵ Journ. de Anat. et Phys., Paris, 1887.

⁶ Bull. de la Soc. Anat., Paris, 1878-1882.

⁷ Thèse de Paris, 1883.

⁸ Journal of Anatomy and Physiology, London, 1886; Annals of Surgery, 1889; Lancet, London, 1889.

⁹ Annals of Surgery, 1889.

them of sufficient importance to account for these structures. Borst¹ gives the salient features of the argument: "At the beginning of the third month the spinal cord reaches to the third last coccygeal vertebra and is continued to the lowest coccygeal vertebra as a bundle of irregularly arranged nerve fibres in which the cells are embedded in groups and rows apparently without distinct regularity, and there is no central lumen. This bundle of fibres is connected with the deep layers of the cutis at the lower end of the spinal column, and these structures form a swelling and a cavity, the latter partly lined with polyhedral epithelium and partly with a form more on the cylindrical order. Owing to the unequal growth of the outer coverings of the spinal column and of the spinal cord, the growth of the soft parts being slower than that of the spinal column, a loop-like formation of this terminal portion of the spinal cord gradually develops, which bends toward the dorsum of the foetus (horseshoe form of the terminal end).

"The concavity of this loop is directed backward and upward; the two ends of the loop terminate, the one anteriorly and deep seated (descending), and the other posteriorly and superficial (ascending), the latter lying immediately beneath the skin in the deep tissues behind the last coccygeal vertebra. At the end of the third month the conus of the spinal cord is on the level of the junction of the third and fourth sacral vertebra, and below this there is only an epithelial tube surrounded by a thin layer of longitudinal nerve fibres, which structure broadens along the lower coccygeal vertebræ (the descending end) and terminating below in the ascending end as mentioned above; the filum terminale developing out of the upper sacral portion of this cord, while the lower gradually disappears. So the so-called 'ascending' of the spinal cord is not only due to an unequal growth of the spinal cord and spinal column, but also to the fact that a portion of the former undergoes retrograde changes and disappears.

"The deeper end of the before-mentioned loop, as intimated, undergoes retrograde changes by atrophy during the fourth month. But the upper end of the mentioned loop, which at this time lies over the second last coccygeal vertebra under the skin, continues to grow up to the fifth month, then reaching its maximum development ($\frac{3}{4}$ millimetre along the fourth and fifth coccygeal vertebræ); in the formation of strands and heaps of polyhedral cells with irregular cavities lined with cylindrical, cuboid, or polyhedral epithelium, the whole extending from below anteriorly to above posteriorly, and connected with the tip of the coccyx by means of the so-called caudal ligament; this ligament accompanies the entire medullary remnants throughout their whole extent.

¹ Centralblatt. f. Allgem. Path. u. Path. Anat., 1896.

"These mentioned medullary remnants also begin to atrophy after the fifth month; but traces of the same, it is said, can be found in the fully developed newborn.

"At the middle of the sixth month only two or three of the mentioned heaps of cells remain, each possessing a cavity, and in the wall of the latter small, round cavities are found between the cylindrical or flat cells.

"Of the cells lining the central cavities of these structures (Perman') some become flat, resembling squamous epithelium (as that of the superficial skin), which might indicate a return to the former ectodermal type; others become elongated and resemble the cylindrical cell of the ependyma.

"The cells forming the mass of the medullary remnant retain their spherical or polyhedral shape. Ciliated epithelium has not been found in the medullary remnants." These researches introduced a new element in the discussion, and the persistence of these structures was considered by many as productive of sinus and cyst formation in this region.

Sutton, in 1889, considers it more satisfactory to regard these sinuses and cysts as "slight defects in the coalescence of the superficial portions of the medullary folds in the sacrococcygeal region."

Masse,¹ in 1889, agrees substantially with Lannelongue. Mallory, in 1892, working independently of Tourneux and Herrmann, after an examination of the bodies of six fetuses by section through the sacrum and adjacent tissues, confirms their results. He attributes everything in the way of dermoid structure found over the coccyx and sacrum to these medullary remnants, neural vestiges, or "vestiges coccygiennes" of Tourneux and Herrmann. "These cases show," he says, "that in fetuses of three to six months there is frequently present over the coccyx a canal lined with epithelium, in some cases connected with the skin, in others not; in some, situated near the skin; in others, near the coccyx. The question naturally arises as to their origin. They may be due either to an extension inward of the epidermis or to the remains of some canal. If due to an extension, or as Lannelongue assumes, to the skin being bound down to the coccyx, why do they not contain the glands and hair follicles with which the epidermis in that region is studded. As regards the extension inward of the skin, why should it occur here so often and nowhere else? It seems much more likely that they are due to incomplete obliteration of a former canal, and extending, as they all do, upward and posteriorly to the coccyx, the medullary canal seems to be the most likely origin.

"The branchial clefts are closed by the eighth week. As before stated, the medullary canal has been open as late as the ninth week,

¹ Laugenbeck's Archiv, 1895.

² Bull. et Mém. Soc. de Chir., 1889; Bull. gén. de Therap., 1885.

consequently the obliteration of the clefts in the one case, and of the medullary canal in the other, must take place at about the same period of intra-uterine life, with this difference, that the growth is more rapid and perfect in the upper part of the body, and hence more favorable to the closure of the clefts. If, notwithstanding this, sinuses and cysts occur in the neck and about the ears, there is at least an equal chance that they may occur at the lower end of the medullary canal.

"It would seem, from a study of the sections from these fetuses, that obliteration of the medullary canal takes place at first and most completely at the lower end of the sacrum, and extends from this point in both directions. As is well known, the spinal cord at first extends the whole length of the vertebral canal; but, as the latter grows the more rapidly in length, the cord rises and the filum terminale is stretched, thus favoring obliteration of the medullary canal at the lower part.

"The obliteration of the medullary canal between the end of the vertebral canal and the skin apparently frequently takes place in an irregular manner; but, for that matter, the medullary canal in the spinal cord shows frequent irregularities, sometimes existing as a distinct canal, sometimes double, as in Fœtus VII., and often showing in sections only as a very irregular clump of cells. Undoubtedly, the majority of these remnants of the medullary canal become obliterated; only the larger, especially those in which glands and hairs are present, persisting as the depressions, sinuses, and cysts of extra-uterine life; and in all probability it is only the congenital sinuses and cysts which give rise to the suppurating sinuses."

Nicaise, in a clinical lecture reported in 1893, stated that his belief in the origin of these affairs agreed with that of Lannelongue and the majority of the French and German observers. This idea of epiblastic evolution, he says, was mentioned as far back as 1852 by Verneuil.

Ritschl,¹ in 1892, holds extreme views, believing that not only cysts situated upon the dorsum of the sacrum and coccyx, but cysts lined with stratified squamous epithelium, resembling that of the skin, and situated upon the ventral surface of the coccyx as well, are due to these same medullary remnants of Tourneux, Herrmann, and Mallory.

Aschoff,² in 1895, believes that the median agglutination may be the cause of sinuses and dermoid cysts of the dorsum of the sacrum and coccyx. He also says that the caudal extremity of the spinal cord lying on the dorsum curves upward and backward, forming a U-shaped loop, one end of which is connected with the skin. Gradually the loop portion which connects the ends disappears, and at the last the dorsal portion (les vestiges coccygiennes of Tourneux and Herrmann) does the same. The point at which connection with the skin exists, and which

¹ Beiträge zur klin. Chir., 1892.

² Cysten in Lubarsch's Ostertags Ergib., 1895, vol. II.

is connected with the coccyx by fibrous strands, is the fovea coccygea. This idea is again referred to by Stolper.¹ Borst, in 1898, is of the opinion that not only the depressions and sinuses, but the cyst formations and many of the more complex tumors of this region are due to these invaginations and agglutinations of the ectoderm. He believes with Lannelongue, Wendelstadt, Aschoff, and others, that the lower portion of the spinal column being the last portion of the posterior opening of the spinal canal to close, that, therefore, the "separation between the external skin and incurved corneal layer of the blastoderm is more difficult, and fibrous agglutinations with the skeleton are more apt to occur." That these fistulæ and depressions are not always connected with the spinal column by the fibrous bands mentioned by Fere and subsequently by Lannelongue (who denies this as an invariable rule), Wendelstadt, Molk,² Trendelenburg, and others can be explained upon the basis of their "subsequently becoming looser." He finds that these congenital depressions, sinuses, and cysts of the sacrococcygeal region "show the presence of the external skin in their walls." It is true, he says, as Wette³ notes, that there are frequent deviations from the normal skin structure, such as thinning of the epithelial covering, absence of the development of papillæ, lack of true corium, poor development, or entire absence of hair, sebaceous and sweat glands; but this is no more than might be expected with such disturbances in development. Furthermore, due to complex conditions of development of this part of the body, these abnormal agglutinations can and often do occur, and "portions of the ectoderm become displaced beneath the surface, and there continue to develop."

Born, he says, having gone over all the work done in this line (Nagel, Mihalkovicz, Keibel, Reichel, Retterer, Tournoux, etc.), states that with the manifold and complicated processes of invagination and agglutination which occur here, epithelial germs can easily become separated and lead to the formation of cysts. Nor is it astonishing that cysts may occur in the perineal region as well, lined either with cylindrical epithelium or epidermis, as the ectodermal plate is able to produce epidermis as well as mucous membrane.

Stolper, in 1899, believes that Mallory and others "go far beyond the indications" in ascribing to the medullary vestiges the formation of these sacrococcygeal defects. "I am of the opinion that for the formation of fistulæ and true dermoid cysts we must also drop the frequently urged theory, or, at least, modify it very much. Particularly in these structures we miss the presence of nerve tissue as a principal constituent." The idea is a less radical one; it is not the medullary vestiges themselves, "but the peculiarities of growth associated with

¹ Deutsche Zeitschrift f. Chir., 1899.

² Thèse de Strasbourg, 1868.

³ Arch. f. klin. Chir., 1894.

their development and atrophy are of interest in the development of fistulæ and cysts." It is the connective-tissue strands accompanying the ascending arm of the U-shaped tube, and which extend from the tip of the coccyx and later form the ligamentum caudale, which "are not without significance in the development of the mentioned depressions and furrows." "Even if the extra-vertebral nerve mass disappears after birth in the physiological way, the easily imagined bilateral continued growth of the accompanying connective-tissue strands may result in the formation of depressions and furrows, which, owing to comparatively early intra-uterine closure of overgrowing epidermis may result in canals. This becomes all the more probable when we consider that the medullary remnants have reached their maximum development at the beginning of the fifth month and begin to atrophy after the sixth month." He believes the epidermoid fistulæ and the sacral dermoid cysts to be a "continued development of the much more frequent and similarly explained foveæ coccygeæ." Both the fistulæ and the cysts are structures of true epidermis and with an epidermoid lining which is continuous with that of the external skin, and both are connected beneath with either the sacrum or the coccyx.

Wette's observations upon the often found relatively incomplete development of the skin lining these structures are also referred to. Owing, however, to the known irregularities in the formation of hair and the hair follicles and sebaceous and sweat glands in the different portions of the external skin, we should not be surprised to find similar irregularities in these complex malformations as seen elsewhere. Two cases are reported in which histological examination of the tissue removed was made. The first case, that of a man, aged thirty-one years, and suffering from an acute inflammation of one of these fistulous tracts (Clinic, Dr. Wagner, Konigschutte) was cured by radical excision of the whole tract. "It was found at several points that the wall of the fistula presented almost exactly the same histological structure as the external skin, except that the layer of epidermis was not so thick and had less of a corneal layer." There were no hair follicles found, but glandular structures very closely resembling sebaceous formations lay deeper in the skin than they are usually found. Disregarding the lesions of the acute inflammatory process and those due to previous curettings, "we had to deal with an open canal, open below, closed above, the wall of which was composed of epidermis throughout." The second case was that of an acute inflammatory process in a congenital sac showing similar conditions. Cure also resulted after radical extirpation. Microscopical examination of the sac here showed a mucous-membrane-like structure without hair follicles and with scant glandular structural development. The tract leading to the dilated portion was lined with granulation tissue, and no direct transition from the skin to

the wall of the fistula could be made out, "but the undisturbed portion showed epidermoid lining."

This last case illustrates the almost completely sequestered pouch, communicating with the exterior of the body by a narrow fistulous tract only. Some of these invaginations are at the start of considerable size, while others dilate from the closure of their fistulous outlets, the resulting accumulation often augmented by inflammatory products forming a tumor. This class must be grouped with the dermoid cysts.

We have, then, in this sacrococcygeal region three abnormal formations—depressions, sinuses, and cysts—usually communicating with the external surface of the body, sometimes not, and which are lined with a tissue identical with that covering the external surface, namely, skin. Morris¹ has recently reported another case.

That the "vestiges coccygiennes" of Tourneux and Herrmann and Mallory are the remains of the medullary canal there does not seem to be any doubt, and no attempt is made to deny the fact. It is even quite certain that in many cases they form a part of and give rise to the more complex congenital malformations, the tumors of monogerminal origin of this region. But it would certainly seem that here their significance ceases; they are to be regarded merely as a part of the original way of communication between the medullary canal and the skin surface, and are destined for obliteration. Nor have, in all probability, the connective-tissue strands of such importance to Stolper any causative relation with our dimples, sinuses, and cysts. If we are to regard the sinuses and cysts as more advanced forms of the dimple, as is undoubtedly the case, it is difficult to see how the dilated ends of these sinuses, and more particularly the sacculated pouches and the more extensive cyst formations, may be produced by traction of a few strands in this manner. And, too, would these binding strands tend to loosen as age advances, and when we know connective tissue thickens and becomes less mobile? We find, it will be remembered, our largest number of dimples in infancy and childhood.

It would seem that if the medullary remains were earlier or primary forms of our coccygeal defects, we should sometimes find in the latter evidences of the fact in the form of anomalies of the lining skin; the presence of cuboidal, columnar, or intermediate forms of cells approaching the squamous variety of the skin and transitions, one to the other. Likewise, in our medullary vestiges we might expect to find at times irregularities; portions of stratified squamous epithelium or transitional forms with or without persistence of the structure as a whole. Furthermore, as has been mentioned, nerve tissue is notably absent in the walls or in the tissue surrounding our sinuses and dermoid cysts. Histolog-

¹ Annals of Surgery, 1900.

ical examination shows us that in our epidermoid structures nothing but true skin has ever been found, except in those instances where it has not been possible to identify any lining tissue on account of inflammatory changes. As regards "the cells lining the central cavities of these structures (medullary vestiges), some become flat, resembling squamous epithelium (as that of the superficial skin), which might indicate a return to the former ectodermal type. Others become elongated and resemble cylindrical cells of the ependyma. The cells forming the mass of the medullary remnant retain their original spherical or polyhedral shape. Ciliated epithelium has not been found." Mallory mentions columnar and cuboidal epithelium. Tourneux and Herrmann found stratified epithelium of the prismatic type and pavement type. They approach either the cells of the medullary tube or those of the epidermis in their evolution. That the elements of the medullary vestiges, instead of atrophying, may take on a lawless growth, just as occurs in tissue with tumor formation elsewhere in the body, we have reason to believe, as has been said, because tumors of this region have been described containing cysts accompanied by or embedded in nervous or neuroglia tissue. Such are the cases of Gutzeit,¹ Weigert,² Wasle,³ Perman, Schonborn, etc. The cysts in many cases were lined with cuboidal, cylindrical, and stratified squamous epithelium, often one form merging with the other. Such a case was Perman's. "The cysts lined with pavement epithelium not infrequently show the general character of a dermoid cyst; there may be a cornification of the epithelium (Hamel⁴); a development of papillæ (Nasse⁵); hair (Hamel, Lutkemuller,⁶ Hildebrand⁷); and of sebaceous and sweat glands (Lutkemuller, Nasse, Hildebrand)."

It is not unusual to find fistulæ and dermoid cysts occurring in conjunction with these complex tumors of this region. Nor is it to be regarded as evidence against their formation from the ectoderm of the surface. Agglutinations and involutions in these cases occurring in similar manner as in uncomplicated cases, while other displacements of ectoderm, or part only of the same, together with medullary remnants and other foetal structures by proliferation, form the bulk of the tumor mass. (See Schmidt's⁸ case.) He found in a three-and-a-half-year-old girl a number of fistulæ in the sacrococcygeal region which communicated with a dermoid containing hair. This was connected to a complex tumor which had a pedicle connection with the spinal column.

Furthermore, we have to consider such cases occurring elsewhere; in the lumbar region or higher (cases of Muscatello,⁹ Kaufmann,¹⁰ Wette,

¹ Dissertation, Königsberg, 1896.

² Dissertation, Wiesbaden, 1896.

³ Langenbeck's Archiv, 1893-1896.

⁴ Arch. f. klin. Chir., 1896.

⁵ Archiv f. klin. Chir., 1894.

⁶ Virchow's Archiv, 1875.

⁷ Dissertation, München, 1895.

⁸ Oesterr. med. Jahrb., 1875.

⁹ Dissertation, Greifswald, 1889.

¹⁰ Dissertation, Berlin, 1883-1893.

etc.), where there are no medullary vestiges. We find, therefore, except in the large and progressively growing tumor formations on the one hand, medullary remnants, and upon the other, formations of true skin. There are no gradations in these smaller defects. So that it would seem that the epidermal lining of our dimples, sinuses, and cysts was derived from a tissue or cellular structure already differentiated and fixed and subject to no possibility of any other outcome. If this is not so, we will have to believe that the ectoderm of the distal portion of the tube becomes fully developed epidermis, or nothing at all, and disappears. If it is true that we have these dermoid structures, because the surface type of epithelium is developed, then we have a right at times to expect an irregularity in development or an irregular disposition of this epithelium. So far in many cases we have yet to see squamous epithelium of the surface in conjunction with cuboid, spherical, or polyhedral shaped and disposed cells. In the utter lawlessness prevailing in tumor formations in this region, however, this is to be expected, and is found.

Again, we have to consider the cases where two, three, or even four congenital fistulous tracts are seen. It is difficult to see how the ascending end of the vestigial loop can account for these.

The fact that we can demonstrate dimples, sinuses, and cysts along the spinal column, in the neck, face, back of head, and median line, in mouth, etc., where there are no spinal cord remains, but where embryonal parts do meet and fuse, and where involutions or abnormal curvings and agglutinations may occur, seems certainly to establish an analogy in formation with those of the median line in the sacrococcygeal region. Cases of White,¹ Abraham,² Sutton, Wagstaffe,³ etc., are referred to.

Further study of anatomical material upon the embryology of this region will form the basis of a subsequent paper.

Of the dermoid cysts, we speak of the true congenital dermoid lined with squamous epithelium and found usually upon the dorsal surface of the sacrum and coccyx, and which never reaches a size larger than that of the "fist," and "usually does not assume such size until later in life," and even then they are soft and can be "moulded" on pressure (Streit). Sometimes there is a fistulous communication with the skin. Their etiology is that of the depressions and sinuses met with in this region. Stolper considers them together with the dimples and sinuses, which he regards as the simplest forms of abnormality occurring as the result of "disturbance in the approximation of the embryonal component parts at the lower end of the axis skeleton." On the ventral

¹ Transactions of the Pathological Society, London.

² Journal of Anatomy and Physiology, London, vol. xv.

³ Transactions of the Pathological Society, London, 1873.

side of the coccyx and sacrum, sometimes above, but oftener below, the levator ani diaphragm, we also find cystic tumors, "which must be classed as true dermoids." These are usually found of larger size than those of the dorsum. In Deahna's¹ case, pressure ulceration of the vagina and bladder actually happened, the cyst filling the pelvis. In Biernacki's,² a similar one prevented childbirth. They seldom, however, reach the size of the teratoid mixed tumors. Sanger³ believes that this latter class are not truly congenital, but are present "simply as a germinal layer at birth," and must be considered a separate species of tumor. That dermoid cysts of this region are rare is shown by his collection of only eleven cases (three between rectum and coccyx, one between rectum and sacrum, three subperitoneal, in cavity of pelvis under levator ani, three behind rectum in cavity of pelvis, right and left side, one behind and below Douglas' cul-de-sac, but above recto-vaginal septum).

They are not visible as tumors, and are generally met "together with anomalies in the structure and differentiation of the uro-genital apparatus and the anus." They sometimes simulate ovarian or intraligamentary cysts or spina bifida with meningocele. (Death has resulted in two recorded instances from tapping the meningocele cysts mistaken for vaginal cysts and teratoid tumors.) They may rupture after supuration into the bowel, and their contents—débris, pus, and hair—be passed by rectum. They may be mistaken for periproctitic abscesses. (Collected cases of Sanger, Meyer,⁴ von Bergmann, Nasse, etc.)

These dermoid cysts are not confined to the regions immediately in front of and behind the sacrum and coccyx, but are found also in other regions of the pelvic connective tissue. They are noted for their simple construction, Sanger says, and show "no relation to the ovaries, the external skin, and spinal column." He distinguishes those situated high up and those that are low down. The former are more complex structures, generally containing mesodermal and ectodermal tissue, and, therefore, hardly belong to the class of simple, true dermoid cysts, but rather to the teratoid class.

To return to the simple cystic structures of true dermoid character. "The simplest forms of these disturbances, which at the same time tend to throw light on the origin of the larger real dermoid cysts, occur in the form of flat depressions in the skin in the region of the coccyx, or as fistulæ in the same region." Frequently at the end of these fistulæ coccygeæ, lined with epidermis and often containing hair and sweat glands, we find dilatations or cavities, or "a series of epidermoidal sacs (Schmidt⁵) containing atheromatous material, and which may without

¹ Archiv f. Gynäk., 1876, vol. vii.

² Dissertation, Berlin, 1887.

³ Archiv f. Gynäk., 1890.

⁴ Dissertation, Greifswald, 1896.

⁵ Virchow's Archiv, 1888; Arb. aus der Chir. Poliklin. zu Leipzig.

doubt be considered as dermoid cysts formed from the segmented or strangulated ends of such fistulæ;" also these epidermoid fistulæ themselves may, owing to closure of their orifices, dilate and then resemble, and, in fact, constitute, as has been said, dermoid cysts. These cysts may or may not present a dimple or sinus superficially. This character of dermoid is noted for its very slow growth. In this connection the case of Kutz¹ is of interest. He mentions a certain tumor, the size of two fists, situated upon the posterior surface of the sacrum, in a woman, aged fifty-three years, and which was noticeable only as a small nodule at birth. (Cases, also, of Lannelongue, etc.) The simple dermoids of this nature situated upon the ventral surface possess the same character, and may or may not have a dimple or fistulous opening at this site. Usually there is nothing to suggest their presence externally. They are, as a rule, situated between the anterior surfaces of the sacrum and coccyx and the rectum in the loose connective tissue. Birkett's² case was probably one of this kind; Sanger³ classifies it as such, although histological data are scanty. The origin of these cysts is explained upon somewhat different grounds than those given for the structures on the dorsum and probably formed by agglutination of the ectoderm with the skeleton. They stand in very intimate relation "to the cloaca formations and the secondary processes resulting therefrom." Born believes that during and succeeding the cloaca stage, with the processes of invagination and fusions that happen, epithelial germinal cells can become separated and subsequently develop cysts. Aschoff thinks it not astonishing that we find here cysts lined with epidermis or cylindrical epithelium, as the cells of the ectodermal plate are capable of producing epidermis as well as mucous membrane. He instances in this connection the "manifold character" of the urethral mucous membrane which is derived from the urogenital plate. Also, the ectodermal structures concerned in the formation of the cloaca may be used in explanation of the cysts lined with mucous membrane found here. There is, as has been demonstrated (Spee⁴), a "direct transition of entodermal structures into offshoots of the ectoderm" through the normally early obliterated canalis neurentericus, connecting the hind end of the medullary canal and the post-anal intestine. Borst speaks of cysts lined partly with squamous epithelium in this region, which he considers formed from this remnant.

The complex tumors of this ventral region also present cysts lined with stratified, squamous epithelium and mucous membrane and transitional varieties. True dermoid cysts are found forming a part of these complex masses or next to tumors of complex construction. Borst, also, while thoroughly doubting Ritschl's view of the derivation from

¹ Dissertation, München, 1895.

² Berl. klin. Woch., 1884.

³ Guy's Hospital Reports, 1859.

⁴ Archiv f. Anat. und Phys., 1889.

the coccygeal vestiges, says that inasmuch as we have sometimes such great disturbances in this region, possibly in stunted conditions of the lower end of the axis skeleton, "portions of the vestiges coccygiennes may be transposed to the ventral side and give rise to tumors."

We find transitions from the simple congenital dermoid cyst to the larger and more complex dermoids, and from these to the simpler mixed or teratoid tumors monogerminal in origin, and from these again to the elaborately complex masses due to a secondary germinal implantation or true foetal inclusion.

We may regard the foveæ coccygeæ as the simplest of the abnormal disturbances and the mixed teratoid tumors the most complex that occur in this region. All the transition forms are observed. Both Hamel and Masse saw stratified pavement epithelium merge into the ciliated form. Borst and Hildebrand saw cysts that were lined with epithelium resembling that of the intestine, with a basilar border and mucous glands, in which there was a transition to stratified pavement epithelium.

The tumors situated upon the dorsum of the sacrum and coccyx show more or less firm connection with the bone beneath, while those upon the ventral surface have but slight connection, if at all, occasionally being attached to the periosteum. This is what one would expect to find, considering our ideas of origin. That inflammatory changes are prone to occur in the deeper of these structures, the fistulæ, in the pouch-like dilatations often found at their terminations and in the cysts, we have seen. The sebaceous accumulations, proliferated epithelium, and especially hair, products of the lining epidermis, having no outlet, or, at best, a greatly narrowed one, are apt to be retained and lead to irritation and suppurative inflammation. If there be a pre-existing outlet, the pressure of the retained material may re-open it and the result be a persistent chronic or intermittent discharge of pus and débris. If none existed, or the pressure of the accumulation be insufficient to open it, having become sealed through maceration of the skin at the outlet, or if the opening show great disproportion in size, retention and abscess formation result, with the usual consequences: tumor formation with subsequent burrowing of pus and the establishment of secondary fistulous tracts. These fistulous tracts are always to one side of the median line, never in it.

Traumatism of one nature or another is very often the exciting cause for inflammatory change. A blow or nothing more severe than a long period of rest in bed may be sufficient. Wette found seven cases in 15,000 sick persons. He also mentions a case occurring subsequent to the formation of a bed-sore in a typhoid patient. Stolper's first case followed confinement to bed (typhoid). Direct traumatism is, however, more often the exciting cause, as in the case of Routier and

Delens,¹ Goodsall,² Anderson,³ J. M. Warren,⁴ etc. Abscess formation followed in periods of time varying from a few weeks to several months or years. The *secondary* sinuses were situated at points close to the median line, well out on the buttock, and from near the sacro-iliac synchondrosis to the upper posterior and inner surface of the thigh on either side. (Goodsall, in the analysis of his cases, thinks the first rupture occurs, usually from one-quarter of an inch to one-half of an inch on the left side of the median line.) Furthermore, these discharging sinuses have frequently been considered to have their origin in a focus of *tubercular disease* occurring in the sacrum or coccyx, with the result that one or the other of these bones has been curetted nearly away. The cases of Beall,⁵ Goodsall, Madelung,⁶ Gussenbauer⁷ and others contain such examples.

The treatment that these cases have usually received has been incision of the abscess formation and curetting of the discharging sinus, followed by the use of carbolic acid or silver nitrate as caustics to destroy the lining membrane or to facilitate healing. The only plan, however, promising complete success is the radical, complete extirpation of the whole tract down to the periosteum of the bones as soon as practicable.

A CASE OF TRANSITORY CYSTINURIA ASSOCIATED WITH DIAMINURIA.

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AND

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IN a previous paper on cystinuria one of us collected all the cases reported up to midsummer, 1899. Including Simon's⁸ own case, the number was then 107. Since that time three additional cases have been reported by Warburg,⁹ Cammidge and Garrod,¹⁰ and Reid.¹¹ We

¹ Bull. et Mém. Soc. de Chir., 1838.

² St. Bartholomew's Hospital Reports, 1838.

³ Boston Medical and Surgical Journal, 1847.

⁴ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1854; Philadelphia Medical Times, 1873; Boston Medical and Surgical Journal, 1877.

⁵ Atlanta Medical and Surgical Journal, 1889.

⁶ Centralbl. f. Chir., 1885.

⁷ Prag. med. Woch., 1893.

⁸ Cystinuria and its Relation to Diaminuria. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1900, vol. cxix.

⁹ Ueber Cystinuria. Dent. med. Zeit., 1898; cit. in Centralbl. f. d. med. Wiss., 1899, No. 13, p. 222.

¹⁰ On the Excretion of Diamines in Cystinuria. Journal of Pathology and Bacteriology, 1900, p. 327.

¹¹ A Case of Cystinuria Ending in Recovery. New York Medical Journal, 1901, vol. lxxiii, No. 16.

have further found the record of a case by Lafleur,¹ which had been previously overlooked, and Dr. Garrod has kindly written us of two other instances observed by himself and Dr. Cammidge, an account of which has not as yet been published. To this number we can add another case, so that the total now reaches 112.

Warburg's case occurred in a young woman, aged twenty-two years, who had suffered a great deal from articular rheumatism from her sixth to her fourteenth year. A short time before her admission to the hospital she had a chill, and complained ever since of lassitude and general malaise. Physical examination showed no abnormality barring some swelling of the cervical and inguinal glands. The urine was cloudy and contained blood and albumin. On microscopical examination large numbers of leucocytes and red blood-corpuscles were found, but casts were absent. Of crystalline elements many crystals of cystin were seen. Two days later high fever again appeared, associated with severe pains in the hip joints. With a gradual improvement in the general condition the amount of cystin diminished and finally disappeared. Two weeks later a similar attack occurred, and with it the cystin reappeared. With a return to the normal the cystinuria ceased. Attacks of colic (renal) had at no time occurred. The urine of the members of the family contained no cystin. An examination for diamins was not made.

The patient of Cammidge and Garrod was a male, aged twenty-two years, who had been admitted to St. Bartholomew's Hospital, London, with a cystin calculus. The urine was examined during two periods of twelve and twenty-nine days, respectively, with an interval of thirty-eight days. On two days only no cystin was seen on simple microscopical examination, but the addition of acetic acid quickly caused a copious precipitation of typical hexagonal plates. Cadaverin was found on two occasions only, though thirty examinations were made in all. Putrescin could not be obtained from the urine, but was apparently present in the feces; and it is interesting to note that on the first day on which diamin was separated from the urine nothing was obtained from the feces, and the only yield from the feces was obtained in the midst of a long period, during which the urine yielded no diamin in spite of careful daily examinations. Leucin and tyrosin were absent, although acicular tufts were obtained with Moreigne's² method. But the writers find that what Moreigne regarded as tyrosin was in all likelihood the hydrochlorate of cystin.

Cammidge and Garrod also examined bouillon cultures of various

¹ Proceedings of the Montreal Med. Chir. Society, May 9, 1898; cit. in Philadelphia Medical Journal, 1898, vol. 1. p. 910, and reported in the Montreal Med. Journ., by E. M. Eberts, September, 1898.

² Arch. de Méd. expér. et d'Anat. pathol., 1899, first series, vol. xl. p. 254.

micro-organisms which had been isolated from their patient's feces, but in no instance were diamins found.

The writers also report that from an alkapton urine no diamins could be obtained by the method of Stadthagen and Brieger,¹ but on benzoylating the urine a crystalline product resulted, which was soluble in alcohol, insoluble in water, and which melted at 196° to 198° C. The nature of this product is unknown. A pure solution of homogentisic acid, however, did not give rise to anything corresponding; but, very curiously, it was obtained from a specimen of urine to which a solution of the acid had been added.

Reid's case occurred in a man, aged thirty-two years, who for over six years had been suffering with painful micturition, and had been treated for gravel and muscular rheumatism by his family physician. Examination of the urethra revealed ulceration, and in the urine cystin was found. Subsequently the cystin apparently disappeared, and Reid regards the favorable outcome as referable to the medicinal treatment which was employed. An examination for diamins, or of the urine of the patient's family for cystin, was not made.

Lafleur's patient was a chlorotic female, twenty-three years old. There was no history of urinary trouble apart from occasional "scalding" during micturition, extending over a period of five months. There was no family history of calculus, and careful examination of the patient's bladder was negative. The urine developed a peculiar faint, greenish-yellow tinge on standing for twenty-four hours, with a distinct odor of hydrogen sulphide. An examination for diamins was not made.

Of the remaining two cases Dr. Garrod writes that in one an examination for diamins could not be made, as the patient declined to co-operate; but in the second what appeared to be pure benzoyl-putrescin was obtained, with a melting-point of 175° to 176° C. The excretion of six days was examined, but the diamin was present only in one specimen.

At this place we also reproduce the brief report of Böttker's² work, which Garrod gives in his paper, as the original is published in Norwegian: During the first two months of the period in which the cystinuria patient was under observation Böttker succeeded in obtaining from the urine, by the process of Udranszky and Baumann, benzoyl compounds, which were shown by their melting-points and by analysis to be those of putrescin and cadaverin, but the yield was small. Five specimens of feces which were examined yielded no diamins. After the first two months the diamins could no longer be found in the urine,

¹ Berlin. klin. Woch., 1899.

² Norsk. Mag. f. Laegevidensk. Christiania, 1892, vol. vii. p. 1220.

although the excretion of cystin continued as long as the patient remained under observation.

The history of our own case, which occurred in the practice of Dr. W. M. Lewis, is as follows:

The patient is a married woman, white, aged forty-six years, and the mother of six children. With the exception of a miscarriage about five years ago, which was followed by tedious convalescence, owing to pelvic inflammatory disease, she had suffered no serious illness up to the time of the present attack. At various times, however, during the past ten or fifteen years she had complained of attacks of "neuralgia" affecting the head and epigastric region, which were occasionally associated with nausea and vomiting. Her husband contracted syphilis early in life, and the children present evidence of the inherited disease. Her family history is bad: Two brothers and a sister died of phthisis; the mother died, at an advanced age, of "asthma;" the father was an alcoholic, and died as the result of injuries which were sustained while intoxicated.

Her present illness began on January 15, 1900, with a sense of oppression about the chest, severe headache, nausea, loss of appetite, pains in the extremities, constipation, and painful and difficult micturition. She was not seen, however, until two weeks later, when the following note was made: The patient is a spare, slightly emaciated woman, of a dark, sallow complexion and anxious countenance. The skin feels moist and clammy. The tongue is covered with a whitish fur. The pulse varies between 80 and 96 per minute, is intermittent, and of low tension. The temperature is 101° F. The respirations are 25, shallow, and attended by a frequent, dry, hacking cough.

Examination of the chest revealed nothing of importance excepting a few scattered râles in the dependent portions of both lungs behind. Abdominal examination also was negative. There was some tenderness about the region of the spleen, but the organ was neither palpable nor its area of dulness increased.

During the following days attacks of severe pain developed in the region of the heart, which extended backward beneath the scapula and down the left arm. These attacks were attended by extreme dyspnoea and associated with a markedly intermittent pulse, which became quite frequent, ranging between 110 and 120. The temperature varied between 101° and 103.4° F. The tongue was dry and covered with a brownish fur. The skin was moist and hot. There was a tendency to delirium. Expectoration was scanty and tenacious. At this time there was dulness on percussion at the left base, associated with distant tubular breathing and a few fine, crackling râles. The respirations numbered 30, and were associated with severe pain in the chest and back. During the attacks of pain about the heart a systolic murmur was also noted, which was audible over the entire præcordia, but heard with maximum intensity at the base. Upon the subsidence of the attacks, which usually lasted from four to six hours, the murmur would disappear, to recur with each following paroxysm. Later in the disease the pulse-rate became much diminished, falling as low as 40 per minute, and it was noted that with the gradual development of the bradycardia the intermittent character of the pulse disappeared. The temperature gradually reached the normal, the area of dulness at the left base disappeared,

the pulse-rate returned to normal, and by April 10th no symptoms of the recorded illness remained.

A specimen of urine voided on the day of the first examination was highly colored, strongly acid in reaction, and of a specific gravity of 1.025. Albumin was present in considerable amount; the uric acid was much increased; the amount of indican was apparently below normal; sugar was absent. On standing, an abundant, granular-looking, and glistening white sediment collected, which contained a few red corpuscles, leucocytes, and epithelial cells from the genitalia. Casts were then not found. The most interesting feature was the presence of large numbers of colorless hexagonal plates, occurring both singly and in groups, which were soluble in hydrochloric acid and ammonia and insoluble in acetic acid, and which were accordingly regarded as cystin. The amount was not estimated, but was considerable. Daily examinations of the urine were then made for about one month, and it was noted that during the first four or five days crystals of cystin were uniformly present, but never again in such large numbers as on the first day. After the fifth day, very curiously, the substance could no longer be demonstrated either in the sediment of the native urine or after the addition of acetic acid (two years have now elapsed), and to the present day it has not reappeared. Previous to the disease cystin had never been seen in the woman's urine, although Dr. Lewis had made microscopical examinations on several occasions. Attempts on four successive days to isolate diamins from the collected urine of twenty-four hours led to no definite result. On benzoylating the urine according to Baumann's method a small amount of a gummy, brownish substance was obtained, which was soluble in alcohol and insoluble in water. The material was lost, however, during subsequent attempts at crystallization.

As it was thus likely that diamins, if present at all, were to be found only in traces, it was decided to collect the urine in bulk and to examine large amounts at one time; 12,000 c.c. in all were obtained and benzoylated as usual. After separation from the phosphates the alcoholic solution was concentrated to a small volume and poured into thirty times its volume of water. The precipitate which was thus obtained consisted of the same brown, gummy material as before, but in correspondingly larger amount. The solution in alcohol and precipitation with water was repeated twice, but even then no crystals could be obtained. Thinking that the presence of so much of the brown pigment prevented crystallization, the gummy material was again dissolved in alcohol and the solution filtered through animal charcoal. In this manner the greater portion of the pigment was removed, but at the same time a not inconsiderable fraction of the benzoylated material was lost. Efforts to recover this were abandoned, owing to the difficulty of separating the benzoylated material from the pigment. The alcoholic filtrate was now concentrated to a very small volume and then filtered directly into the water while the latter was actively stirred. A tendency on the part of the benzoylated substance to separate out as gummy material was then still observed, but was effectually combated by allowing the alcoholic solution to flow into the water, drop by drop, and stirring all the time. In this manner a milky looking fluid was finally obtained from which no gummy material whatever separated out. On standing for about thirty minutes a grayish, granular-looking sediment began to form, which on microscopical examination was seen

to be composed of delicate, almost colorless platelets and fine, acicular crystals. The total amount of substance which was thus obtained weighed 0.3827 gramme. The crystals were readily soluble in alcohol, insoluble in water, and very sparingly soluble in ether; their melting-point was 129° C., corresponding to that of cadaverin. Putrescin was not found.

Tyrosin could not be demonstrated in the urine according to common methods; but, like Garrod, we were able to show that from pure cystin, when treated according to Moreigne's method, fine crystals of the hydrochlorate can be obtained, which on microscopical examination alone might be mistaken for tyrosin.

A single examination of the feces for diamins led to no result.

The urine of the patient's children and of a brother contained no cystin, nor could a sulphur reaction be obtained.

Our case of cystinuria is thus the seventh in which diamins could be demonstrated in the urine.¹ Both cadaverin and putrescin together have thus far only been found by Baumann and Udranszky and Bödtker; cadaverin alone by Stadthagen and Brieger, by Cammidge and Garrod, by Simon, and in the present instances, and putrescin only by Cammidge and Garrod in their second case.

From the feces of Baumann and Udranszky's patient both diamins were obtained. Putrescin was found by Cammidge and Garrod in one of their cases, and cadaverin by Simon in his first case. In the remaining cases the results were negative.

In his first paper Simon suggested that in future cases of cystinuria repeated examinations should be made before drawing conclusions as to the absence of diamins, and that amorphous precipitates, which may be obtained on pouring the alcoholic solution of the benzoylated products into water, should be purified with special care, so as to favor crystallization of any benzoylated diamins that might be present. Experience has since shown that, as a matter of fact, diamins may be apparently absent from the urine for days, weeks, and possibly months, even though the cystinuria continues; or if not absent, then present in so small amounts as to escape detection with the available methods if the urine of only twenty-four hours is examined. In the present case we obtained no definite result on four occasions on which twenty-four-hour lots were examined, while we were successful with larger quantities. It might, of course, be argued that in our case the diamins were present on one or two occasions only while the urine of a number of days was collected, and absent on others. But of this we are not convinced. We believe

¹ The first case was that of Udranszky and Baumann (*Zeitschr. f. physiol. Chem.*, 1889, vol. xlii. p. 562; see also Garcia, *ibid.*, 1898, vol. xvii. p. 577, and Pfeiffer, who quotes a further examination of this case by Baumann, *Centralbl. f. d. Krankh. d. Harn. u. Sex. Organe*, 1897, vol. viii. p. 173). The second and third cases are those of Stadthagen and Brieger (*Berlin. klin. Woch.*, 1889, vol. xxvi. p. 844). The third is the case of Bödtker (*loc. cit.*); the fourth is Simon's first case (*loc. cit.*); and the fifth and sixth are those of Cammidge and Garrod (*loc. cit.* and above).

that they were uniformly present, though in small amounts, and are prompted in our supposition by the fact that even during the first four days a small amount of benzoylated material was obtained, which was soluble in alcohol and insoluble in water. This was lost during repeated attempts at purification by solution in alcohol and reprecipitation with water. But it will be recalled that later, when working with larger amounts, we obtained this same material apparently, but succeeded in bringing it to crystallization.

But while we believe that every case of cystinuria is associated with diaminuria, we have convinced ourselves that the diaminuria does not necessarily coincide in point of time with the cystinuria, and that, like the cystinuria, the diaminuria also may be a temporary event.

Temporary cystinuria appears to be more common than was once supposed, and renders the condition even more interesting. In our case the elimination of cystin apparently only existed during a number of days, and appeared to be in some manner connected with the patient's illness. In Ebstein's¹ case the condition persisted for about ten days and then disappeared.

In Smith's² two cases the cystin crystals were only found on one occasion, and Reid reports that in his case also the cystin disappeared and remained absent even after a year had elapsed from the time of the first observation. But we cannot regard this disappearance as evidence of a cure brought about by the administration of medicines, as Reid supposes.

As regards the origin of cystinuria, we still maintain the views expressed in a previous paper, viz., that the condition is essentially referable to a metabolic anomaly and not due to a specific microbic infection of the digestive tract. We may further add that our supposition concerning the relation existing between cadaverin and lysin on the one hand, and between putrescin and ornithin on the other, has since been firmly established. Our own investigations in this direction could, unfortunately, not be continued for reasons which lay beyond our control; but Ellinger³ has shown that through the activity of micro-organisms, at any rate, the transformation of ornithin to putrescin, and of lysin to cadaverin can actually be effected.

It has further been shown by Mörner⁴ and Embden⁵ that on hydrolytic decomposition of keratin considerable amounts of cystin can always be obtained, and that of the true albumins egg albumin, serum albumin, and edestin, at least, yield cystein under similar conditions. It

¹ Deutsch. Arch. f. klin. Med., 1879, vol. xxlii. p. 138.

² Practitioner, 1898, vol. i. p. 475.

³ Zeitsch. f. physiol. Chem., 1900, vol. xxix. p. 334.

⁴ Cystin ein Spaltungsprodukt d. Hornsubstanz. Ibid., 1899, vol. xxviii. p. 595.

⁵ Ueber d. Nachweis v. Cystin u. Cystein unter d. Spaltungsprodukten d. Eiweisskörper. Ibid., 1901, vol. xxxli. p. 91.

accordingly appears that in addition to various other groups the albuminous molecule also contains a cystein group or even one of its dithio-compounds. In such an event it would not be necessary to imagine a substitution of an HS group for a hydrogen atom of phenyl-alanin, which we previously suggested as the mother substance of cystein; but we may suppose that the entire group is split off directly, and then escapes oxidation, owing to the activity of some factor of which we are as yet in ignorance, but which probably represents the direct cause of the cystinuria. The diamins, of course, suggest themselves in this connection; but it is to be noted that Baumann and Udranszky¹ failed to produce cystinuria in dogs by the administration of diamins. On the other hand, it might be argued that under normal conditions the diamins would be immediately destroyed, and that in cystinuric individuals a certain insufficiency may exist which prevents their destruction. Of the actual existence of such an insufficiency we believe there can be but little doubt, but we are nevertheless scarcely prepared to admit that a causative relation exists between the cystinuria and the diaminuria, and we still believe that both are the outcome of a third factor which is still unknown. Whether or not any one of the tissue ferments may here be of moment future researches will show.

THE CAUSES AND VARIETIES OF CHRONIC INTERSTITIAL PANCREATITIS.

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LITTLE systematic study has been devoted to the varieties and to the causes of chronic inflammation of the pancreas. Since the lesion is relatively uncommon, and, though affecting an organ having functions of great importance, is rarely associated with symptoms by which it can be recognized during life, it has, as might be expected, failed to attract the attention given to corresponding alterations of the liver and kidneys. The causal relationship of pancreatic disease to diabetes mellitus and to disseminated fat necrosis now recognized, has emphasized the importance of lesions of the organ.

Changes in the gland, notably chronic inflammation, are seldom primary, but are associated with and usually the result of changes in other organs, the adjacent part of the gastro-intestinal tract, the liver and the bile passages. Inflammatory irritants may reach the gland by

¹ Weitere Beiträge z. Kenntnis d. Cystinurie. Ibid., 1891, vol. xv. p. 77.

the ducts, by the lymphatics, and by the bloodvessels. Obstruction of the pancreatic duct is followed by chronic inflammatory changes, retention of the secretion being associated with alterations of the gland cells and proliferation of the connective-tissue stroma. Opening upon the mucous membrane of the duodenum in company with the common bile duct, the pancreatic duct is exposed to the invasion of bacteria from the intestines and from the inflamed biliary passages. Changes in the bloodvessels, arterial sclerosis, and venous stasis by altering the nutrition of the parenchyma are believed to cause chronic inflammation, while a more important factor in its production is the action of various toxic substances brought by the blood, syphilis, tuberculosis, and alcoholic excesses being assigned as causes.

Carnot,¹ who produced chronic pancreatitis experimentally by a variety of means, designates the condition as (a) mechanical, produced by obstruction of the duct, and as (b) toxic or (c) infectious, caused by the action of toxic substances or of bacteria carried to the gland by the blood or by the lymph or by way of the duct. As he points out, such a classification cannot be applied to the varieties of chronic pancreatic inflammation observed in human cases after death, since in many instances the etiological factors concerned are no longer demonstrable.

From the clinical records of the Johns Hopkins Hospital and the autopsy protocols of the pathological laboratory of the University and Hospital twenty-nine cases of chronic interstitial pancreatitis are available, and in all of these cases material has been preserved for microscopical examination. In the present study the attempt has been made to determine the factors concerned in the production of the lesion, as well as its character. Of twenty-nine cases seventeen occurred in males and twelve in females. The following table indicates the age of the individuals in whom the lesion was observed :

| | | | | | | | |
|----------------|---|---|---|---|---|---|----------|
| 10 to 20 years | . | . | . | . | . | . | 1 case. |
| 20 " 30 " | . | . | . | . | . | . | 2 cases. |
| 30 " 40 " | . | . | . | . | . | . | 2 " |
| 40 " 50 " | . | . | . | . | . | . | 9 " |
| 50 " 60 " | . | . | . | . | . | . | 11 " |
| 60 " 70 " | . | . | . | . | . | . | 2 " |
| 70 " 80 " | . | . | . | . | . | . | 2 " |

Over two-thirds of the cases occurred between the ages of forty and sixty years.

In ten of the cases chronic inflammation followed partial or complete obstruction of the pancreatic ducts. In one case complete occlusion of the portal vein was accompanied by moderate proliferation of the inter-

¹ Thesis, Paris, 1896.

stitial tissue; in one case the lesion was associated with a condition of wide-spread pigmentation, described by von Recklinghausen under the name *hämochromatosis*. In the remaining seventeen cases the etiology of the condition was somewhat more obscure, but is in most of them discoverable. In a preceding report¹ I have described the histology of chronic interstitial pancreatitis and attempted to distinguish the anatomical varieties which occur. Two types of chronic inflammation are distinguishable: (a) *interlobular*, where the newly-formed connective tissue is in great part between the lobules, invading them only secondarily and implicating the islands of Langerhans only when the sclerotic process has reached a very advanced grade; (b) *interacinar*, when the process is diffuse, invading the lobules, separating individual acini, and implicating the islands of Langerhans.

When the interlobular tissue is chiefly affected, the pancreas is densely hard in consistence and has a nodular or granular surface; on section the normal loose *aveolar* tissue uniting the lobules is replaced by dense sclerotic bands, so that the gland is more compact in structure, the lobules being firmly held together. When the interacinar tissue is chiefly affected the lesion may not be recognized macroscopically; the gland is firmer than usual, but is tough rather than hard. With both varieties of inflammation the stroma may be abundantly infiltrated with fat, which may contain small foci of necrosis.

In the present study, based upon a much larger number of cases (including those formerly studied), I have found the two types named. In twenty-one cases the lesion was interlobular, in eight interacinar. The attempt will be made to determine not only the etiology of the inflammatory change, but the variety of the lesion associated with a given etiological factor.

OBSTRUCTION OF THE PANCREATIC DUCTS. The usual causes of duct obstruction are calculi within the duct of *Wirsung* or in the adjacent terminal part of the common bile duct and carcinoma compressing or invading the pancreas. Ligation of the ducts in animals is followed by sclerosis of the gland. Arnozan and Vaillard² have studied the progress of the lesion in rabbits, and advance the suggestion that it is caused by the ferment present in the retained secretions. Carnot thinks that a variety of factors are active in its production. The retained secretion, he thinks, has a toxic action upon the glandular cells, while the presence of bacteria invading from the duodenum the pent-up secretion has an important influence on the changes which follow. Reflex nervous stimuli are no longer capable of exciting normal unctinal activity, and, he suggests, no longer exert an influence on the

¹ *Journal of Experimental Medicine*, 1901, vol. v. p. 397.

² *Arch. de Phys. norm. et path.*, 1884, 3 s. vol. iii. p. 287.

secreting cells, which, like voluntary muscle cells after section of their motor nerve, atrophy. One or more of these conditions are doubtless always present, but their relative importance is difficult to determine. In many of the cases to be described evidence of infection was present, and it is often impossible to determine how far it was responsible for the lesion.

The pancreas is usually provided with two anastomosing ducts, of which the lower opening with the common bile duct into the duodenum is the larger. Occasionally the upper duct or duct of Santorini is the larger and its orifice is widely patent, but in the majority of cases the smaller duct is functionally a branch of the larger and its duodenal orifice, which is usually patent, is too minute to act as an outlet for the entire pancreatic secretion.

PANCREATIC CALCULI. In two cases which have been previously reported¹ with greater detail, advanced chronic inflammation was referable to the presence of numerous calculi in the pancreatic ducts.

CASE I.—W. H., male, aged forty-three years, had used alcohol (beer) in large quantity. His father died with tuberculosis. Ten months before his death he had had an attack of jaundice.

Clinical Diagnosis. Pulmonary tuberculosis.

Anatomical Diagnosis. Chronic pulmonary tuberculosis; cirrhosis of the liver; pancreatic calculi; chronic interlobular pancreatitis, with fat infiltration; parapancreatic fat necrosis.

CASE II.—H. R., male, aged fifty years, states that he has been accustomed to drink whiskey in large quantity.

Clinical Diagnosis. Diabetes mellitus; pulmonary tuberculosis.

Anatomical Diagnosis. Pancreatic calculi; chronic pancreatitis; chronic pulmonary tuberculosis; chronic diffuse nephritis.

In both cases dense sclerotic tissue, in one instance containing fat in large quantity, had in great part replaced the parenchyma of the gland. The islands of Langerhans had, in part, resisted the sclerotic process and had undergone disintegration only when, as in Case II., they were wholly isolated in dense bands of scar-like fibrous tissue. When small patches of parenchyma consisting of small groups of lobules persist, the tissue is remarkably well preserved, and there is little if any thickening of the stroma between the acini. Diabetes mellitus present in Case II. was of mild type and disappeared when the patient was put upon a diet poor in carbohydrates.

BILIARY CALCULI. The relationship of chronic inflammation of the pancreas to cholelithiasis has excited much interest since Riedel² directed attention to the induration of the head of the gland at times observed during operations undertaken for the removal of gallstones, and readily mistaken for malignant growth. In three of one hundred and twenty-two such operations he noticed this condition and suspected the pres-

¹ Loc. cit.

² Berliner klin. Wochenschr., 1896, vol. xxx. pp. 1, 82.

ence of carcinoma, but the subsequent history disproved this supposition, and an autopsy obtained in one case demonstrated the presence of chronic inflammation of the gland. Mayo Robson¹ has described two cases in which at operation for gallstones in the common bile duct the head of the pancreas was found so indurated that a diagnosis of malignant growth was made. In one of these cases autopsy demonstrated the existence of chronic interstitial pancreatitis, and since in the second case the subsequent history disproved the diagnosis of carcinoma, Robson thinks that here also chronic pancreatitis was present.

The following cases give additional evidence of the association of chronic inflammation of the pancreas and cholelithiasis, and demonstrate a mechanism by which this change is produced.

CASE III.—The patient, male, aged sixty-three years, states that he has drunk whiskey in considerable quantity, but otherwise his personal history is unimportant. Two years and four months before his death he had an attack of jaundice lasting five months. A year later he was admitted to the service of Dr. Halsted with jaundice, associated with repeated chills, and an operation was performed, but owing to the density of the adhesions present it was found impossible to explore the bile passages. After the operation his condition improved, and he was fairly well until two and a half months before his death, when he was admitted to the service of Dr. Osler with jaundice and ascites; the liver dulness was decreased in extent. The abdomen was tapped three times and on each occasion about eight litres of fluid were withdrawn. The patient died with symptoms of a general infection, and a culture made from the blood before death demonstrated the presence of *Streptococcus pyogenes*. The urine at no time contained sugar. The autopsy was performed by Dr. W. G. MacCallum.

Anatomical Diagnosis. Cholelithiasis; contraction of the gall-bladder; calculus in the common bile-duct; dilatation of bile-ducts; cirrhosis of the liver; chronic interlobular pancreatitis; serofibrinous peritonitis; chronic nephritis; arterial sclerosis; diverticula of duodenum.

The common bile duct is greatly dilated and enters the duodenum beside a small diverticulum of the intestinal mucosa. Near its termination, but just above its junction with the duct of Wirsung, is a large oval gallstone (1.5 x 0.8 x 0.8 cm.) firmly wedged into the common duct in such a position as to compress the terminal part of the pancreatic duct which, as it enters the diverticulum of Vater, passes immediately below the stone. The pancreas is firm in consistence and compact in texture. The duct of Santorini joins the duct of Wirsung, of which it appears to be a branch, and approaches the wall of the duodenum, terminating in a minute papilla which is not demonstrably patent. The liver has a rough granular surface and is tough in consistence.

Microscopical examination of the pancreas shows that the loose interlobular areolar tissue is replaced by bands of dense sclerotic tissue containing many collections of lymphoid cells with a smaller number of plasma cells and polynuclear leucocytes. Within the lobules there is relatively little increase of the interstitial tissue.

¹ *Lancet*, 1900, vol. II, p. 235.

A small calculus I have shown in a previous report¹ may lodge at the orifice of the diverticulum of Vater and occluding it may convert the common bile duct and the duct of Wirsung into a continuous channel, thus causing the penetration of bile into the pancreas and consequent acute hemorrhagic pancreatitis. A larger calculus present in the diverticulum of Vater, or in the common bile duct, or slightly above its point of junction with the pancreatic duct, may temporarily or permanently occlude the latter and produce chronic inflammatory changes resembling those which follow ligation of the duct in animals. Doubtless bacterial infection often plays a part in the production of the lesion, and in the two following cases the bile passages containing gallstones are the seat of an acute suppurative inflammation which has been propagated along the duct of Wirsung.

CASE IV.—Female, aged fifty years.

Anatomical Diagnosis. Cholelithiasis; calculi in gall-bladder, cystic, hepatic, and common bile ducts; adenocarcinoma of gall-bladder; metastases in liver; jaundice; suppurative cholangitis; chronic interlobular pancreatitis; peripaucreatic fat necrosis.

The liver is deeply jaundiced and is the seat of numerous small abscesses. The gall-bladder, whose wall is thickened by a carcinomatous growth, is contracted over a number of faceted gallstones. The hepatic, cystic, and common bile-ducts near their point of junction are dilated and contain two faceted calculi. The remainder of the common duct is not markedly dilated, but the walls are somewhat thickened and fibrous. The pancreas is very firm in consistence and is infiltrated with fat containing small opaque white foci of necrosis.

Microscopical examination of the liver shows numerous foci of necrosis and suppuration. The normal lobulation of the pancreas is much accentuated by a marked increase of the interlobular tissue. The medium-sized ducts are moderately dilated and contain polynuclear leucocytes in small number.

CASE V.—The patient, female, aged seventy years, was admitted to the service of Dr. Osler, complaining of swelling of the abdomen and legs. Her present illness began eight months before her death, and since this time she has had chills every second or third day. The abdomen and legs have been swollen for two months. She has not been jaundiced and there has been no nausea or vomiting. The bowels have moved not more than once a week. On admission to the hospital, a month before her death, she was emaciated and general anasarca was present. The leucocytes numbered from 33,000 to 27,000. For three weeks before her death the temperature was continuously subnormal. The urine contained numerous casts and albumin in abundance, but no sugar.

Anatomical Diagnosis. Cholelithiasis; suppurative cholangitis; cirrhosis of the liver; chronic interlobular pancreatitis; chronic diffuse nephritis; amyloid kidneys; brown atrophy of the heart.

The gall-bladder, adherent to adjacent organs, contains pale, turbid bile and two faceted calculi. The common bile duct is patent. The

¹ Johns Hopkins Hospital Bulletin, 1901, vol. xii. p. 182.

liver, of which the interstitial tissue is increased, contains small abscesses. The pancreas is large and very firm, and in the adjacent fat are minute spots of fat necrosis. Its ducts are dilated and contain viscid material. Microscopical examination of the pancreas shows a marked proliferation of the interlobular tissue, which is dense and fibrous, but rich in lymphoid cells. The duct of Wirsung and its branches are dilated and contain polynuclear leucocytes and red blood-corpuscles, while their walls are infiltrated with polynuclear leucocytes. The interstitial tissue of the liver, much increased about the portal spaces, is dense and sclerotic and contains polynuclear leucocytes. The small ducts are often greatly distended and filled with polynuclear leucocytes.

In Case III. a calculus in the common bile-duct occupied a position in which it compressed the pancreatic duct; the lesser pancreatic duct was not demonstrably patent. Chronic interlobular pancreatitis was the result of duct obstruction, with perhaps secondary invasion of bacteria. In Cases IV. and V. no calculus was found compressing the pancreatic duct, but in Case IV. one may have occupied such a position at some previous time. In Case V. the history gives no evidence of the passage of a calculus along the duct, and chronic inflammatory changes in the pancreas are presumably the result of an ascending infection originating in the acutely inflamed bile passages. In support of this conclusion there exists an acute inflammation of the pancreatic ducts.

MALIGNANT GROWTH. In five cases chronic interstitial pancreatitis was caused by a malignant growth compressing or invading the organ. In the following case, which may serve as an illustration of this condition, a primary carcinoma of the pancreas arising in the head of the gland compressed the duct of Wirsung and produced chronic interstitial inflammation accompanied by the formation of cysts.

CASE VI.—The patient, female, aged forty-one years, became jaundiced nine months before her death. She gives no history of having had syphilis. A tumor mass was palpable in the umbilical region. The autopsy was performed by Dr. Flexner.

Anatomical Diagnosis. Adenocarcinoma of the pancreas; compression of the bile and pancreatic ducts; jaundice; chronic interlobular pancreatitis; metastatic carcinoma of the retroperitoneal lymph glands.

The tumor, which arises from the pancreas, consists of two masses, between which the remains of the head and part of the body lie compressed. A fibrous capsule separates the tumor from the gland parenchyma. Numerous cysts about the size of a walnut occupy the body and tail, and are present, but less numerous, in the duodenal end. A probe can be passed along the compressed duct.

Microscopical examination shows throughout the gland a moderate increase of the interstitial tissue between the lobules and in less degree within them. Cysts lined by a single layer of cubical or flat epithelium are abundant in the body and tail.

In the second case (Case VII. of the present series) previously described,¹ advanced chronic interstitial inflammation followed the development of a carcinoma of the bile papilla and diverticulum of Vater. The carcinomatous tissue was removed at operation, and the biliary and pancreatic ducts were transplanted into the duodenum. At autopsy the duodenum was the seat of an extensive carcinomatous ulcer, into the base of which the pancreatic duct opened. Marked dilatation of the pancreatic duct gave evidence of its partial obstruction. Infection from the ulcerated surface upon which the duct opened was with great probability an important factor in producing the advanced chronic interstitial inflammation which resulted.

In three cases (Cases VIII., IX., and X.) chronic inflammatory changes were associated with the invasion of the gland by a carcinomatous new-growth of the stomach. When the head of the gland is invaded there is a diffuse interstitial change; but where, as in two cases, the body is invaded, while the head is not in contact with the tumor, inflammatory alterations occur only in the part of the gland distal to the point at which the duct is compressed. The carcinomatous tissue, moreover, acts as a local inflammatory irritant, and in its immediate neighborhood there is a proliferation of the gland stroma replacing the parenchymatous elements.

ASCENDING INFECTIONS FROM THE DUODENUM. Occlusion of the duct and stagnation of the pancreatic secretion produce conditions favorable for the invasion of bacteria. Under certain circumstances bacteria from the duodenum or from inflamed biliary passages may invade the pancreatic ducts in the absence of duct obstruction. In Case V. the inflammatory process was transmitted to the pancreas from the inflamed bile passages, though there was no evidence that the pancreatic duct had been occluded.

The following case is described in order to show that acute interstitial pancreatitis may accompany an acute inflammation of the stomach and duodenum, and is doubtless the result of an ascending duct infection:

CASE OF ACUTE PANCREATITIS. The patient, female, aged thirty-eight years, was admitted to the service of Dr. Osler eighteen days before her death. She had had four children born at term and six miscarriages, the last a little more than a year ago. During her pregnancies micturition has been frequent and painful. Following the last her feet became swollen, and urination has been very frequent. Her appetite has been good, but she has been troubled with indigestion and has vomited frequently after meals. During her stay in the hospital the oedema of the legs, arms, and dependent parts increased. The urine contained a considerable amount of albumin (0.3 to 0.4 per cent.), and hyaline and epithelial casts. The patient died in uræmic coma on the nineteenth day after her admission.

¹ Journal of Experimental Medicine, 1901, vol. v. p. 409.

Anatomical Diagnosis. Chronic diffuse nephritis; small, granular kidneys; hypertrophy of the heart; general arterial sclerosis; chronic passive congestion of the viscera; acute and chronic gastritis and duodenitis; acute interstitial pancreatitis.

The stomach contains clear, viscid mucus adherent to the mucosa, which shows deep scarlet-red injection. In the duodenum, particularly upon the summits of the valvulæ conniventes, similar injection is seen. The pancreas is very firm in consistence. Microscopical examination of the stomach wall shows the superficial bloodvessels of the mucosa deeply injected, while between and within the glands polynuclear leucocytes are abundant. Between the glands plasma cells are present in large number. The alterations of the duodenal mucosa are of the same character, though slightly less marked. The interstitial tissue of the pancreas is oedematous and infiltrated with polynuclear leucocytes, often forming collections of considerable size. The ducts contain products of secretion and polynuclear leucocytes, often in abundance.

In the preceding case acute interstitial pancreatitis of moderate severity accompanied inflammation of the stomach and duodenum, which, associated with chronic nephritis and chronic passive congestion, had for several months before the patient's death caused frequent vomiting. The ducts of the gland contained inflammatory products, and the lesion was evidently the result of an ascending inflammatory process, having its origin in the gastro-intestinal tract.

Doubtless many cases of chronic inflammation are the result of a primary acute lesion, and represent a late stage of the process. This condition is illustrated by the following specimen, obtained from an autopsy not performed in the laboratory, and unaccompanied by clinical history or autopsy notes. It is described in order to show that proliferation of the interstitial tissue may follow an acute suppurative process. Beginning proliferation of the interstitial tissue accompanies acute inflammation, and the lesion may be said to be in a transitional or subacute stage. The process is doubtless caused by bacterial invasion, and numerous bacilli are demonstrable in the ducts and in the acutely inflamed interstitial tissue.

Specimen of Acute Interstitial Pancreatitis The interlobular tissue is oedematous, and contains scattered accumulations of polynuclear leucocytes, often in great number, among which are lymphoid and young fibroblast cells. The interlobular fibrous bands are slightly thickened, and here and there occur collections of lymphoid cells. The lumina of the acini are often dilated and contain polynuclear leucocytes. In specimens stained with methylene blue, short bacilli, varying in size, are found both in and between the acini where leucocytes are abundant and in large numbers within the lumina of the ducts.

Korte¹ has produced chronic inflammation of the pancreas by injecting *Bacillus coli* into the pancreatic duct, and has obtained a similar

¹ Berliner Klinik, 1896, No. 102.

result by injecting fecal material. By an ingenious method Carnot¹ produced conditions by which an ascending infection from the duodenum results. A thread was inserted into the pancreatic duct and through its orifice into the duodenum, and left fixed in this position. Advanced sclerosis of the gland resulted, and the walls of the ducts were thickened and infiltrated with leucocytes, while the interstitial tissue of the gland was much increased. In another experiment Carnot produced suppurative inflammation by injecting colon bacilli into the pancreatic duct.

CHRONIC INTERLOBULAR PANCREATITIS ASSOCIATED WITH PERSISTENT VOMITING. In four cases advanced chronic pancreatitis has been found in cases which during life suffered with persistent vomiting. Two of these cases occurred in young women, and there was no history of syphilis, alcoholic excess, nor any other condition which has been assigned as a cause of chronic pancreatitis, and at the autopsy the ducts of the gland were found unobstructed. In one of these cases vomiting accompanied pregnancy, the clinical diagnosis being *emesis gravidarum*. The third case occurred in a man, aged forty-nine years, who, though once addicted to alcohol, had for ten years led a temperate life; chronic gastritis was found at autopsy. In the fourth case the condition accompanied constriction of the terminal part of the duodenum. In all of these cases the clinical history affords evidence of continued gastric or gastro-intestinal disease, namely, epigastric pain, nausea, and vomiting, the latter repeated and severe.

CASE XI.—The patient, a deaf mute, female, aged thirty-one years, entered the service of Dr. Osler complaining of heartburn and dyspepsia. She states that at the age of twenty-four years she had "inflammation of the stomach," and was sick for eight weeks. She has had one living child and three miscarriages. She is at present pregnant, and has not menstruated for twelve weeks. For six or seven years she has had dyspepsia. Four days before her admission she began to vomit, the vomiting has persistently continued and has occurred irrespective of the taking of food. There has been no blood in the vomitus. She complains of a constant burning pain in the epigastrium, which is increased after eating. Physical examination disclosed nothing of importance. During the day, after her admission, vomiting was almost continuous, often occurring three or four times an hour. Examination of the vomitus showed a total acidity of 11, free hydrochloric acid 9; no lactic acid; hæmin crystals were present. The red blood-corpuscles numbered 8,612,000, the white corpuscles 17,000, hæmoglobin 125 per cent. In the afternoon of the following day, the patient becoming much weaker and semi-conscious, the vomiting ceased.

She was transferred to the surgical side, and an exploratory laparotomy was performed, but nothing was found to account for the symptoms. The patient's condition after operation was improved, and there

¹ Loc. cit.

was no return of vomiting until twelve days later, when it recurred with its previous persistence. The pulse was rapid and irregular, 112 to 152 to the minute. The vomiting ceased for a short time, but recurred a week later.

The patient was transferred to the obstetrical ward, and it was decided to empty the uterus. The cervix was dilated, and an ovum two and a half months old was removed. The patient rallied from the operation, but died on the twenty-second day after her admission. The urine contained a trace of albumin and a few hyaline casts, but at no time sugar.

Anatomical Diagnosis. Endometritis of puerperal uterus; anomalous artery, a branch of the aorta penetrating the substance of the right lung; rupture, with formation of cavity containing blood-clots; chronic interstitial pancreatitis.

The condition of the uterus and of the right lung will not be described. The stomach is normal, save for the presence of a few submucous ecchymoses; the duodenum is normal in appearance. The pancreas, weighing 75 grammes, is almost board-like in consistence; the surface is nodular and irregular, while on section the texture is homogeneous, the normal loose interlobular tissue being replaced by dense stroma. This interlobular tissue is dense and fibrous, and contains many spindle-shaped, lymphoid eosinophile and plasma cells. Entire lobules are at times partially destroyed, a few scattered acini remaining in the proliferated stroma, but in general the sharply defined lobules are not invaded by the process.

CASE XII.—H. B., female, aged twenty-eight years, was admitted to the hospital, complaining of bleeding from the vagina, weakness, and pain in the abdomen. She had had chills and fever at the age of twenty years, and two years ago smallpox, but otherwise she had been healthy. Her present illness began ten months before her admission, with a sudden, profuse hemorrhage from the vagina, and since this time she had discharged clots of blood. An operation, the nature of which she does not know, was performed five months before, and was followed by much nausea and vomiting. Previous to her admission she has been much nauseated, and states that even a small quantity of water may cause her to vomit, so that though often hungry she is afraid to eat. The bowels are constipated. She has colicky pains in the epigastrium, and during the examination she frequently puts her hand to the umbilical region and complains of cramps. The patient was much emaciated and anæmic. Vaginal examination demonstrated the presence of an inoperable fungating carcinoma of the cervix. While in the hospital the patient's condition gradually became worse, and death occurred. The urine contained no sugar.

Anatomical Diagnosis. Carcinoma of the uterus, with metastases in the broad ligaments, pelvic and lumbar lymphatic glands, liver, and rectum; double pyelonephrosis; chronic interstitial pancreatitis.

The pancreas is firm in consistence, and on section very compact in texture, the interlobular tissue being replaced by denser stroma. There are no noteworthy changes in the stomach, intestine, or bile passages. Microscopical examination of the pancreas shows an abundant proliferation of the interlobular tissue, which is dense and fibrous, but often contains lymphoid and plasma cells in considerable number. It separates sharply the lobules, which are usually rounded as though compressed by the new tissue.

CASE XIII.—Male, aged forty-nine years, was admitted to the service of Dr. Osler, complaining of rheumatism, cough, shortness of breath, and indigestion. He had had three attacks of inflammatory rheumatism before the age of twenty-one years, but since has had fairly good health. He denies having had syphilis; he admits having formerly used alcohol in excess, but says that for the last ten years he has not been a heavy drinker, only taking a drink occasionally. He has been short of breath on exertion for five years; but indigestion, with which he suffers, troubles him, he states, even more than the shortness of breath. For a year past he has had attacks of vomiting without any apparent cause and with no relation to the taking of food, occurring sometimes in the morning before breakfast. He describes the vomitus as consisting in great part of phlegm. On admission, the patient is a well-nourished man, slightly cyanosed. The heart is hypertrophied, its action is feeble, and at the apex is heard a systolic murmur. On one occasion he passed a small amount of blood by the rectum. His condition gradually became worse, and he died on the fourth day after admission. The temperature while in the hospital was subnormal; the urine contained a trace of albumin, hyaline and granular casts, but no sugar.

Anatomical Diagnosis. Chronic endocarditis of the mitral valve; dilatation and hypertrophy of the heart; cardiac thrombi; chronic passive congestion of the viscera; infarcts of lungs; right hydrothorax; acute and chronic gastritis; ulceration of the ileum and colon; chronic pancreatitis with lipomatosis.

The mucosa of the stomach, covered by a thick layer of tenacious mucus, has a puffy appearance and is red in color, studded with small submucous ecchymoses. Superficially the pancreas appears to be a mass of fat, glandular tissue being nowhere visible; it weighs 152 grammes. On section the gland tissue is found to be in very great part replaced by fat. The duct of Wirsung is patent throughout, and about its branches can be seen small masses of reddish-yellow gland tissue embedded in fat, and forming, except in the tail, a relatively small proportion of the cut surface.

Microscopical examination shows that groups of lobules are widely separated by adipose tissue, while within these groups individual lobules are separated by thickened strands of dense stroma containing many lymphoid cells. In places are seen lobules in process of disintegration, dilated acini composed of cubical cells, being separated by new-formed stroma, but usually the new-formed tissue does not invade the lobules.

The cases just described demonstrate the occurrence of chronic interlobular pancreatitis in individuals who during life have suffered with persistent vomiting. They indicate the existence of some relationship between the underlying gastro-intestinal disturbance and the chronic lesion of the pancreas. It is improbable that persistent vomiting was caused by the pancreatic diseases, since in many cases the latter has existed unaccompanied by this symptom. Persistent vomiting, even though dependent upon disturbances of a reflex nervous mechanism, or upon the evacuation of toxic products into the stomach, is indicative of some profound change in the organ, and has apparently produced conditions

favoring an ascending infection of the ducts. The type of the accompanying pancreatic lesion favors this conclusion, since it is typically interlobular and resembles that observed in the preceding cases where infection of the pancreas had followed lesions of the gall ducts, with or without obstruction of the pancreatic duct. This explanation is further confirmed by the following case, in which persistent vomiting was the result of partial occlusion of the terminal part of the duodenum:

CASE XIV.—Mrs. F. G., aged fifty-one years, entered the service of Dr. Osler seven weeks before her death, complaining of nausea, vomiting, and loss of weight. Seven years before her uterus had been removed by Dr. Kelly for carcinoma of the cervix; she has since enjoyed good health. About six months before her admission she had noticed that she was losing weight. Later she was troubled with severe pain in the abdomen and back, uninfluenced by taking food, and became constipated. The pain disappeared, and she felt well until two months before entering the hospital, when she was attacked with very severe vomiting, occurring often five or six times a day and unaccompanied by nausea, but preceded by slight pains in the chest. Food caused no pain. Occasionally she passed several days without vomiting. Physical examination disclosed what appeared to be a tumor mass, irregular in outline, crossing the upper half of the umbilical region. While in the hospital there were frequently repeated attacks of vomiting, usually at intervals of twelve to eighteen hours, the vomitus consisting of greenish-yellow fluid material, varying in amount from a few cubic centimetres to a litre; it contained no blood. The patient gradually became weaker and died. The temperature occasionally rose to 100° F. The urine contained a small amount of albumin and a few hyaline and granular casts, but no sugar.

Anatomical Diagnosis. Recurrent adenocarcinoma of the retroperitoneal lymphatic glands and of the peritoneum; indurated retroperitoneal carcinomatous tissue, constricting the duodenum and left ureter; multiple abscesses of kidney; cystitis; bronchopneumonia; fatty degeneration of the liver; fibrous myocarditis.

There is no tumor mass in the pelvis, but in front of the sacrum, near the promontory, are a few enlarged firm lymphatic glands which, on microscopical examination, are found to contain an epithelial new-growth of the type of adenocarcinoma. On either side of the aorta occurs in moderate amount indurated tissue. Similar tissue, scar-like in character, is found at the base of the mesentery where the jejunum emerges, and upon the neighboring peritoneal surface are several small, soft nodules. Sclerotic tissue invading the wall of the duodenum at its termination has contracted the lumen to a diameter of twelve millimetres. The constricted area is not more than one centimetre in length, and above and below the intestinal wall is soft. From the base of the mesentery indurated tissue, which is found to consist of alveoli of epithelial cells embedded in dense stroma, extends into the mesentery of the transverse colon and in the retroperitoneal tissue as far as the left ureter, which is constricted by it. A narrow band of similar tissue extends between the pyloric end of the stomach and the pancreas, but does not invade the latter. The pancreas is very firm, and upon the surface individual lobules are sharply defined. The

ducts are not dilated. Microscopical examination shows an interlobular inflammation of moderate intensity, the lobulation being accentuated by thickened fibrous bands containing lymphoid, plasma, and eosinophile cells in fair abundance. The acini are dilated, the islands of Langerhans unchanged.

The four cases described show that chronic interlobular pancreatitis may occur as the result of alterations in the stomach and duodenum, which during life are manifested by long-persistent vomiting, and is presumably the consequence of an ascending infection of the duct. The mechanical effect of vomiting upon the ducts and their contents is obviously difficult to determine.

ALTERATIONS OF THE BLOODVESSELS. In the cases already considered, chronic pancreatitis has been secondary to alterations occurring in the ducts; in another group of cases the ducts are unchanged, and the lesion is referable to the bloodvessels or to toxic substances brought to the gland by the blood. In the pancreas, as in other organs, general arterial sclerosis has been thought to be the cause of fibroid induration, and G. Hoppe-Seyler¹ and Fleiner² have described cases of chronic interstitial pancreatitis attributed by them to obliterating endarteritis. Both writers think that changes in the vessels are followed by nutritive disturbances which cause degeneration of the parenchyma and its replacement by fibrous tissue. The condition, Fleiner suggests, is analogous to the contracted kidney, which is at times associated with general arterial sclerosis, and to changes in the liver, heart, and brain following arterial diseases. In three cases of arterial sclerosis Kasahara³ found a moderate increase of the interstitial tissue of the pancreas, but in other cases of the same disease found nothing more than thickening of the bloodvessels. In none of the cases of chronic interstitial pancreatitis here considered has arterial sclerosis, general or local, been a conspicuous feature of the condition, and in only four cases was arterial sclerosis of moderate grade noted in the anatomical diagnosis. In two cases not included in the series the arteries of the pancreas were greatly thickened, while only in their immediate neighborhood was there proliferation of the interstitial tissue.

A second alteration of the bloodvessels assigned as a cause of chronic pancreatitis is chronic passive congestion. Friedrich⁴ states that the lesion is not infrequently the result of long-continued venous gorging occurring in chronic diseases of the heart, lungs, and liver. The changes, he says, are usually slight, and do not cause destruction of the glandular elements, but between the acini are formed small tracts of thickened connective tissue, giving the gland an increased toughness.

¹ Deutsch. Archiv f. klin. Med., 1893, vol. lli. p. 171

² Berliner klin. Wochenschr., 1894, vol. xxxi. pp. 5, 83.

³ Virch. Arch., 1896, vol. cxliii. p. 111.

⁴ von Ziemssen's Practice of Medicine (trans.), vol. viii. p. 551.

Small interstitial hemorrhages occur, and are changed later into collections of rust-colored pigment.

In the following case the pancreas was the seat of an extreme grade of chronic passive congestion following complete occlusion of the portal vein by a primary carcinoma of the liver.

CASE XV.—Anatomical diagnosis. Primary carcinoma of the liver, invading and occluding the portal vein; chronic passive congestion of the spleen, pancreas, stomach and intestines; ascites.

A large, carcinomatous mass occupies the right lobe of the liver and invades the branch and main trunk of the portal vein, which is greatly distended and wholly occluded. A plug of new-growth extends into the splenic vein. The pancreas is compressed by the distended vein behind it, and is firm and compact in texture and of a uniform dull brownish-red color.

In the head and body of the gland strands of carcinoma cells are found within the largest venules, at times wholly obliterating their lumen. In the immediate neighborhood fibrous tissue is increased in amount, and thickened strands extend between the lobules. At a distance from the carcinomatous tissue there is a very slight diffusely distributed increase of the interlobular tissue, which is denser than usual. Among the acini in places occur irregular patches of interstitial tissue poor in cells. Small hemorrhages have in places occurred into the interlobular tissue, and both here and within the secreting cells is found brown pigment. The bloodvessels are widely dilated.

In the preceding case the pancreas was the seat of a slight generalized increase of the interstitial tissue between the acini as well as between the lobules, but it serves to illustrate how small is the influence of chronic passive congestion in causing proliferation of the pancreatic connective tissue. In two additional cases of the present series chronic passive congestion dependent upon a valvular lesion of the heart accompanied chronic inflammation of the pancreas. One of these cases (Case XIII.) has already been described. In the following case the inflammatory change cannot with any certainty be attributed to chronic congestion, since at least one other possible factor existed, namely, lead poisoning.

CASE XVI.—H. D., a brass moulder, aged thirty-eight years, was admitted to service of Dr. Osler with symptoms of chronic lead poisoning and mitral stenosis. He disappeared from observation, and died four years later.

Anatomical Diagnosis. Mitral stenosis; dilatation and hypertrophy of the heart; chronic passive congestion of viscera; bronchopneumonia with gangrene; chronic interlobular pancreatitis, with fat infiltration; peripancreatic fat necrosis.

The frequency of chronic congestion of the abdominal viscera, and the relative infrequency of chronic interstitial inflammation of the pancreas, is evidence that the former condition is not commonly a cause

of the latter. Chronic passive congestion may doubtless produce slight proliferation of the interstitial tissue, but is an unimportant factor in the production of chronic pancreatitis.

In fourteen of the twenty-nine cases there is reason to believe that chronic inflammation has been the result of changes in the duct, obstruction, or ascending infection. In most of the remaining cases it is probable that the alterations present are like corresponding changes in the liver and kidney: cirrhosis, and chronic interstitial nephritis caused by the action of toxic substances present in the blood. Various conditions presumably capable of producing such substances have been assigned as the cause of chronic pancreatitis, and of these those notably worthy of consideration are tuberculosis, syphilis, and alcohol, named in the probable order of their increasing importance.

TUBERCULOSIS. Carnot¹ has recently directed attention to the occurrence of chronic pancreatitis in cases of tuberculosis. The specific lesion of tuberculosis is, he thinks, relatively uncommon in the gland, and in only one case has he observed acute miliary tuberculosis. Diffuse chronic interstitial inflammation of the pancreas, associated with tuberculous lesions of other organs is, he states, much more common, and a number of the older writers—Ancelet, Valpian, Arnozan and Morache—have cited such cases. Carnot examined the pancreas in patients dying with tuberculosis, and while in the majority no lesion was demonstrable, in seven cases he found a variable, usually moderate grade of chronic inflammation, causing in most cases an increase of the connective tissue normally present about the vessels, the ducts, and between the lobules. In five cases the lesion was associated with pulmonary tuberculosis, in one case with multiple tuberculous foci, presumably of the joints or of bone (“tuberculoses chirurgicales multiples”), accompanied by prolonged suppuration; while in one case a localized sclerosis of the splenic end of the pancreas accompanied an extensive tuberculous caseation of the left kidney. Carnot was able to produce chronic inflammatory changes in the pancreas of dogs, somewhat varied in extent and character, by injecting suspensions of tubercle cultures into the duct or into the parenchyma of the organ, but it was necessary to inject a very great quantity of the culture. A caseous abscess resulted in one case, but in the other experiments the lesion presented none of the specific characters of tuberculosis, and tubercle bacilli were not demonstrable in the tissues. In a single case localized sclerosis was produced by injecting into the parenchyma tuberculin extracted from the bodies of the dead bacilli.

In the two following cases chronic interstitial pancreatitis was found at the autopsy upon individuals dead with advanced tuberculous lesions.

¹ Loc. cit.

CASE XVII.—H. G., male, aged thirty-one years, was admitted to the service of Dr. Osler, complaining of cough and weakness, with which he had suffered for about a year. Four months before his admission, for a week he had had much pain in the left side, and during the same time vomited every morning. He states that he has not had syphilis. The physical signs of an effusion into the left chest were present, and clear straw-colored fluid was removed by tapping. Evidence of partial consolidation of the left lung was obtained, and tubercle bacilli were found in the sputum. The patient rapidly became weaker, and died a month after admission. There had been no sugar in the urine.

Anatomical Diagnosis. Chronic pulmonary tuberculosis; tuberculous pleurisy, with hydrothorax on left side; tuberculous ulceration of larynx and ileum; bronchopneumonia; acute nephritis; chronic interlobular pancreatitis; fat necrosis about pancreas.

Upon the pancreas and in the omentum are a few opaque, white areas of fat necrosis. Microscopical examination of the pancreas shows a moderate grade of interlobular pancreatitis, and the lobules, more clearly defined than usual, are held together by thickened fibrous bands of compact tissue containing relatively few lymphoid cells.

CASE XVIII.—H. B., female, aged seventeen years, was admitted to the service of Dr. Kelly. The present illness began a year before her death, with a profuse ill-smelling discharge from the vagina. Several months later she had fever, and since has not been well, complaining of pain in the back and abdomen. Vaginal examination, followed by curetting and histological examination of the material removed, demonstrated the existence of tuberculosis of the cervix uteri. The abdomen was later opened, but owing to numerous adhesions and abundant hemorrhage it was not possible to remove the uterus. Signs of tuberculous consolidation of the lungs subsequently developed and the temperature ranged between 99° and 103°. She died seven weeks after her admission. Sugar was not present in the urine.

Anatomical Diagnosis. Tuberculosis of the cervix uteri, endometrium, and Fallopian tubes; miliary tuberculosis of the spleen, kidneys, and liver; solitary tubercles of the cerebrum; tuberculous pleurisy; tuberculous ulcer of the rectum.

The pancreas is large and firm in consistence. There is a diffusely distributed increase of the interstitial tissue, most marked between the lobules, which are more clearly defined than usual. Lymphoid cells, plasma cells, and eosinophile leucocytes are fairly abundant in the new-formed stroma. In one section is found a miliary tubercle with a partially caseous centre.

In Case XVIII. the specific lesion of tuberculosis was found in the pancreas, but in Case XVII. tubercles did not occur in the specimens studied. In three additional cases in which tuberculous lesions were found in the pancreas there was no general proliferation of the interstitial tissue. The two cases described support the conclusions of Carnot that in a few instances generalized tuberculosis is accompanied by chronic pancreatitis, but they afford no explanation of its occurrence. The condition may be dependent upon the proximity of a tuberculous lesion, as in Carnot's case, where only the splenic extremity of the

gland in contact with a tuberculous kidney was affected, and a similar explanation may be applicable to the cases of Arnozan, in which chronic pancreatitis accompanied tuberculous peritonitis.

Since tuberculosis is, as is well known, a frequent complication of diabetes, its relationship to alterations of the pancreas are of much interest. In the cases just cited diabetes did not occur, and the lesion of moderate severity is interlobular, a type of inflammation accompanied by diabetes only when far advanced. There is, it appears, no reason to suppose that diabetes may be caused by pancreatitis resulting from tuberculosis.

SYPHILIS. The autopsy records of the Pathological Laboratory furnish no instance in which chronic pancreatitis has been associated with visceral syphilis, and, though the literature demonstrates that the two conditions occur in conjunction, acquired syphilis is certainly not the most common cause of the lesion, as Hansenmann,¹ Kasahara,² and other writers believe. In only one of the present series of cases was a history of syphilis obtained, and in this case chronic pancreatitis with formation of cysts was caused by primary carcinoma of the pancreas compressing the duct. In a case described by Drozda,³ the pancreas was represented by a mass of indurated tissue in which glandular structures were recognizable only in the head, while here and there occurred caseous gummata embedded in the fibrous stroma. The liver was the seat of syphilitic cirrhosis, and the stomach contained an indurated scar. In a case of visceral syphilis, described by Chvostek,⁴ the tail of the pancreas was penetrated by several sclerotic bands of tissue, giving it a lobed appearance. In these cases the alterations are analogous to syphilitic lesions of the liver, and are characterized by the occurrence of gummata or irregular, scar-like bands of tissue penetrating the parenchyma, and differ from the more common lesion in which there is a diffusely distributed increase of the interlobular or interacinar tissue. To the latter type belongs apparently the case of Dieckhoff,⁵ in which, though there was a history of syphilis, coexisting carcinoma of the stomach and pulmonary tuberculosis may be regarded as possible etiological factors. In two cases of chronic interstitial pancreatitis associated with diabetes, Hansenmann obtained evidence of syphilis. Kasahara examined the pancreas in six cases of acquired syphilis, and in two found a moderate increase of the interstitial tissue, in two cases merely thickening of the bloodvessels, and in two cases no alterations.

Syphilis is, no doubt, a cause of chronic inflammation of the gland, and the cases of Drozda and Chvostek indicate the existence of a syph-

¹ *Zeitsch. f. klin. Med.*, 1894, vol. xxvi. p. 191.

² *Wiener med. Presse*, 1880, No. 81.

³ *Festschrift f. Thierfelder*, Leipzig, 1895.

⁴ *Loc. cit.*

⁵ *Wiener med. Blätter*, 1879, p. 791.

ilitic pancreatitis characterized by the occurrence of scar-like bands of tissue which may contain gummata. The etiological relationship of syphilis to a diffusely distributed interacinar or interlobular inflammation is more doubtful.

ALCOHOL. The common cause of cirrhosis of the liver is excessive use of alcoholic drinks. Cirrhosis is relatively infrequent when compared with the prevalence of alcoholic indulgence; but, as has been pointed out here, as with most diseases, other conditions are necessary in order that the disease results. Alcoholic excess is likewise regarded as a frequent cause of chronic interstitial pancreatitis, and in many instances a history of alcoholic indulgence can be obtained. Friedreich found at autopsy upon a drunkard chronic pancreatitis, cirrhosis of the liver, and granular kidneys. Chvostek, Dieckhoff, and Oser¹ have described cases in which cirrhosis of the liver and chronic pancreatitis were associated in alcoholics. In eight of the present twenty-nine cases a history of alcoholic excess was obtained, but in three of these cases (Cases I., II., and III.) chronic inflammation of the organ had followed obstruction of its duct by biliary or pancreatic calculi, and was indirectly, if at all, referable to the use of alcohol.

In the following case chronic interlobular pancreatitis, unaccompanied by cirrhosis of the liver, was found at autopsy in an individual dying with delirium tremens.

CASE XIX.—J. M., male, aged fifty-three years, was admitted to the service of Dr. Osler with delirium. His wife, from whom a satisfactory history was not obtained, stated that he had been drinking very heavily for a month, and on the morning before his admission became delirious and feverish. The temperature was 101.5° F. The patient was in a talkative delirium, and there was tremor of the hands. The abdomen was distended and tympanitic. The patient became completely unconscious, the temperature rose to 105.4° F., and he died on the second day after admission.

Anatomical Diagnosis. Bronchitis and beginning aspiration pneumonia; acute diphtheritic and hemorrhagic colitis; fatty degeneration of the liver; chronic interlobular pancreatitis; fat necrosis near the pancreas.

The pancreas, of normal size, is very firm in consistence, and the interstitial tissue appears to be indurated. The ducts are normal. Numerous small foci of fat necrosis occur about the pancreas and in the transverse mesocolon. Microscopical examination shows a typical chronic interlobular pancreatitis, and the interstitial tissue contains numerous lymphoid cells.

Few reported cases of chronic inflammation of the pancreas not dependent upon lesions of the duct are described with sufficient detail to determine the presence or absence of an alcoholic history, and a review of them would afford statistics of little value. Alcohol being

¹ Die Erkrankungen des Pankreas. Nothnagel's Spec. Path. u. Ther., Vienna, 1898, vol. xviii.

the common cause of cirrhosis of the liver, the association of chronic pancreatitis with this lesion is significant.

ASSOCIATION OF CHRONIC PANCREATITIS WITH CIRRHOSIS OF THE LIVER. The two conditions are frequently dependent upon similar etiological factors, since in the present twenty-nine cases of chronic pancreatitis cirrhosis of the liver was present in eight. In three of these cases pancreatitis was indirectly, if at all, related to the lesion of the liver, and had followed readily demonstrable changes in the duct of Wirsung. The relationship of the blood-supply of the liver to that of the pancreas suggests the possibility that venous obstruction might play a part in the production of pancreatic sclerosis, but the small influence which it exerts has already been pointed out.

Chronic inflammation of the liver and pancreas have been found associated by Friedreich, Chvostek, Hansenmann, Dieckhoff, Kasahara, Oser, and Lefas.¹ In six cases of chronic interstitial hepatitis Kasahara found a marked increase of connective tissue of the pancreas, in two cases a slight increase, and in two none at all.

Of considerable interest are the observations of Lefas, who studied alterations of the pancreas accompanying different varieties of hepatic cirrhosis. With the so-called atrophic or Laennec's cirrhosis he finds that the weight of the pancreas is often increased to 120 or 130 grammes, and the new-formed tissue, which is poor in cells, is uniformly intralobular, penetrating the parenchyma and isolating groups of acini. The point of origin seems to be the capillaries, but at an advanced stage there is a moderate grade of proliferation about the small veins and arteries. With hypertrophic biliary cirrhosis (of Hanot) there is no increase in the volume of the pancreas, but the interlobular tissue is increased in amount and in density, and, containing numerous cells, appears to have its origin about the ducts. The cases of interstitial pancreatitis which I have observed in part confirm the observations of Lefas, though a relationship of the newly-formed tissue to the blood-vessels and ducts was not evident.

In the following cases, previously reported² in greater detail, chronic interacinar pancreatitis accompanied by diabetes was associated with cirrhosis of the liver of the so-called atrophic type of Laennec.

CASE XX.—The patient, male, aged forty-nine years, gave no history of alcoholic excess nor of syphilis.

Clinical Diagnosis. Cirrhosis of the liver, with congestion of the portal circulation and ascites; diabetes mellitus.

Anatomical Diagnosis. Atrophic cirrhosis (of Laennec); chronic interacinar pancreatitis; acute peritonitis, with effusion; splenic tumor.

The liver, which weighs 1370 grammes, has a rough, coarsely granular

¹ Arch. de Méd., 1900, N. S. 3, p. 539.

² Journal of Experimental Medicine, 1901, vol. v. p. 414.

surface, and on section shows a coarse network of thickened fibrous septa. The pancreas is small and firm in consistence. Microscopical examination shows a diffuse interacinar proliferation of the stroma invading the islands of Langerhans.

CASE XXI.—The patient, male, aged forty-seven years, gives a history of having used whiskey and beer in great quantity. For six months there have been symptoms of diabetes mellitus.

Anatomical Diagnosis. Atrophic cirrhosis (of Laennec); chronic passive congestion of the spleen; ascites; chronic interacinar pancreatitis; peripancreatic fat necrosis; general arterial sclerosis; gangrene of the leg.

The liver, diminished in size, is markedly hobnailed in appearance, and on section islands of parenchyma are isolated by thick strands of fibrous tissue. The pancreas, which weigh 108 grammes and is firm, presents microscopically a condition similar to that of the preceding case.

Numerous cases in which diabetes accompanied cirrhosis of the liver are recorded, and the relation of diabetes to diseases of the liver has been much discussed. It is probable that in such cases diabetes, as in the preceding cases, is dependent upon the coexistence of chronic pancreatitis, and cases with autopsy report described by Dieckhoff and Pusinelli¹ support this conclusion.

An excellent illustration of the dependence of chronic lesions of the liver and of the pancreas upon the same etiological factor is furnished by the disease of pigment metabolism, first described by von Recklinghausen as hæmochromatosis. In this condition an iron containing pigment is deposited in the cells of the liver, pancreas, and various other organs, and its presence is associated with death of parenchymatous cells and consequent proliferation of the stroma. The liver is the seat of an interstitial inflammation characterized by an increase of the connective tissue at the periphery and in the centre of the lobules. Though the liver is hypertrophied the histological picture resembles more closely the atrophic cirrhosis of Laennec than the so-called hypertrophic type described by Hanot. In Case XXII. of the present series (described in a previous article²) hæmochromatosis had resulted in the production of cirrhosis of the liver and chronic interacinar pancreatitis, resembling, though less advanced, the variety observed in the two preceding cases, but accompanied by deposition of an iron containing pigment, both in the secreting cells and in the newly-formed stroma. In the greater number of recorded cases of hæmochromatosis death has occurred with symptoms of diabetes mellitus, described by French writers as bronzed diabetes, since pigmentation of the skin is a conspicuous feature of the general pigmentation. In the present case diabetes mellitus was not

¹ Berliner klin. Wochenschr., 1891, vol. xxx. p. 739.

² Journal of Experimental Medicine, 1899, vol. iv. p. 279.

present, doubtless because the lesion of the pancreas had not reached a sufficient grade of intensity.

In the following case hypertrophic cirrhosis of the type of Hanot was accompanied by interlobular pancreatitis.

CASE XXIII.—The patient, male, aged fifty-six years, admitted to the service of Dr. Osler, gives a history of having used whiskey in excess, usually taking from fifteen to twenty-five drinks a day. He denies having had syphilis. For the last year, until a short time ago, he has had attacks of vomiting, coming on without any relation to the taking of food; at one time about a year ago the vomiting was very severe. During the last six months he has become weak and short of breath. The conjunctivæ are slightly jaundiced. The liver is enlarged, the abdomen contains fluid, and the superficial veins are dilated. Three litres of bile-stained fluid were removed from the abdomen. Death occurred with increasing weakness nine days after admission.

Anatomical Diagnosis. Hypertrophic cirrhosis (of Hanot); ascites; chronic passive congestion of spleen; chronic interlobular pancreatitis; cholelithiasis.

The liver is greatly hypertrophied, weighing 2880 grammes; the surface is in places smooth, in places uneven and marked by low elevations, but without a hobnailed appearance. The tissue is firm and cuts with difficulty. Irregularly-distributed bands and patches of fibrous tissue, rich in cells, separate ill-defined islands of parenchyma and penetrate between the cell columns and between individual cells. The gall-bladder contains many small faceted calculi. The pancreas, weighing 60 grammes, is very firm in consistence and evidently sclerotic. The duct of Wirsung in the body and tail of the gland is dilated and contains solid wax-like material. Microscopical examination shows that the lobules, particularly in the body and tail, are separated by dense sclerotic tissue showing little tendency to penetrate between the acini.

In the case just described so-called hypertrophic cirrhosis (of Hanot) was accompanied by interlobular pancreatitis, as in the cases observed by Lefas. In an additional case interlobular pancreatitis of moderate grade accompanied beginning cirrhosis, and, though the hepatic lesion corresponded to the so-called atrophic or Laennec's cirrhosis, the alteration of the pancreas differed from that observed by Lefas and found in Cases XX. and XXI. It is noteworthy, however, that the lesion was associated with tuberculous peritonitis, while a variety of chronic lesions coexisted in other organs.

CASE XXIV.—E. C., female, aged twenty-two years, was admitted to the service of Dr. Kelly with a fecal fistula following the removal of an abdominal tumor, the nature of which the patient did not know. An anastomosis was made between the sigmoid flexure and rectum. The patient died eight months later.

Anatomical Diagnosis. Invagination of the sigmoid flexure into the rectum; chronic periproctitis; fibrous adhesions uniting loops of ileum, with perforation of adjacent loops; perforation of a loop of the ileum

adherent to the abdominal wall, with formation of a fecal abscess and fistula; fibropurulent peritonitis; tuberculous peritonitis; tuberculosis of retroperitoneal lymphatic glands; chronic and acute endocarditis; chronic nephritis; cirrhosis of the liver with fatty degeneration; chronic interlobular pancreatitis.

INTERACINAR PANCREATITIS OF OBSCURE ETIOLOGY. In the present series of twenty-nine cases there are five instances of interacinar pancreatitis whose etiology is wholly obscure; in all of them the lesion is of very slight intensity. In four cases the islands of Langerhans were the seat of a remarkable hyaline change, and diabetes mellitus had resulted. I have already described one of these cases¹ in which, though the islands of Langerhans were almost uniformly affected by the degenerative change, increase of the interacinar stroma was very inconspicuous. Hyaline degeneration of the islands of Langerhans in association with diabetes has since proved to be of much more frequent occurrence than was at first suspected, and, in addition to the subsequent examples which have come to my notice, cases have been recently observed by Wright and Joslin,² and by Hertzog.³ In one of two cases of Wright and Joslin, and in both cases of Hertzog, hyaline degeneration of the islands of Langerhans was associated with proliferation of the interstitial tissue of the gland.

In the following case the islands of Langerhans were almost wholly unchanged, but the pancreas was the seat of a slight interacinar pancreatitis, and diabetes was absent. The clinical and anatomical diagnosis in this case is as follows:

CASE XXIX.—Male, aged fifty-eight years.

Clinical Diagnosis. Inoperable carcinoma of the rectum; œsophageal diverticulum. Inguinal colostomy was performed, and later the œsophageal diverticulum, which compressed the trachea, was removed. Death followed the latter operation.

Anatomical Diagnosis. Carcinoma of rectum; metastases in the retroperitoneal lymphatic glands, liver, and lungs; colostomy wound; wound of operation for œsophageal diverticulum; bronchopneumonia; mucous polyp of the colon; chronic interacinar pancreatitis (slight).

Macroscopically the pancreas appeared unchanged, but microscopical examination shows the occurrence of thickened strands of connective tissue between the acini. The increase of interstitial tissue is, however, slight, and in many places is entirely absent. Only rarely are the islands of Langerhans implicated in the chronic inflammatory process.

Of eight cases of chronic interacinar pancreatitis, six were accompanied by diabetes mellitus. In those in which diabetes was absent,

¹ *Journal of Experimental Medicine*, 1901, vol. v. p. 527.

² *Journal of Medical Research*, 1901, vol. vi. p. 360.

³ *Transactions of the Chicago Pathological Society*, 1901, vol. v. p. 15.

Case XXII., with hæmochromatosis, and Case XXIX., just described, the lesion was little advanced and the islands of Langerhans were only slightly affected.

CONCLUSIONS. 1. Chronic interstitial pancreatitis is slightly more frequent in males than in females. Two-thirds of the total number of cases occur between the ages of forty and sixty years.

2. The most frequent cause of chronic pancreatitis is obstruction of the duct of Wirsung, due to pancreatic calculi, to biliary calculi in the terminal part of the common bile-duct, or to carcinoma invading the head or body of the gland. Duct obstruction may be followed by the invasion of bacteria, which take part in the production of the resulting lesion.

3. Ascending infection of the unobstructed duct of Wirsung may follow an acute lesion of the duodenum or of the bile passages, and may cause chronic inflammation. In cases which have given a history of long, persistent vomiting, chronic diffuse pancreatitis may be found at autopsy, and is probably the result of an ascending infection of the gland.

4. General or local tuberculosis is occasionally accompanied by chronic diffuse pancreatitis, affecting chiefly the interstitial tissue of the gland.

5. Chronic interstitial pancreatitis is not infrequently dependent upon the same etiological factors, notably alcohol, which produce cirrhosis of the liver, and in about one-fourth of the cases the two lesions are associated.

6. Following duct obstruction and ascending infection the lesion affects principally the interlobular tissue, only secondarily invading the lobular tissue and sparing the islands of Langerhans. Diabetes results only when the lesion is far advanced.

7. Accompanying the so-called atrophic or Laennec's cirrhosis of the liver, the pancreas is at times the seat of a diffuse chronic inflammation, characterized by diffuse proliferation of the interacinar tissue, which invades the islands of Langerhans. A similar lesion accompanies hyaline degeneration of the islands of Langerhans and the condition known as hæmochromatosis.

8. Interacinar pancreatitis is usually accompanied by diabetes mellitus. When diabetes is absent the lesion is of such slight intensity that the islands of Langerhans are little implicated.

A NEW CABINET FOR MICROSCOPIC SLIDES, DESIGNED BY
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POLYCLINIC HOSPITAL.

BY MARY B. KIRKBRIDE,
POLYCLINIC LABORATORIES, PHILADELPHIA.

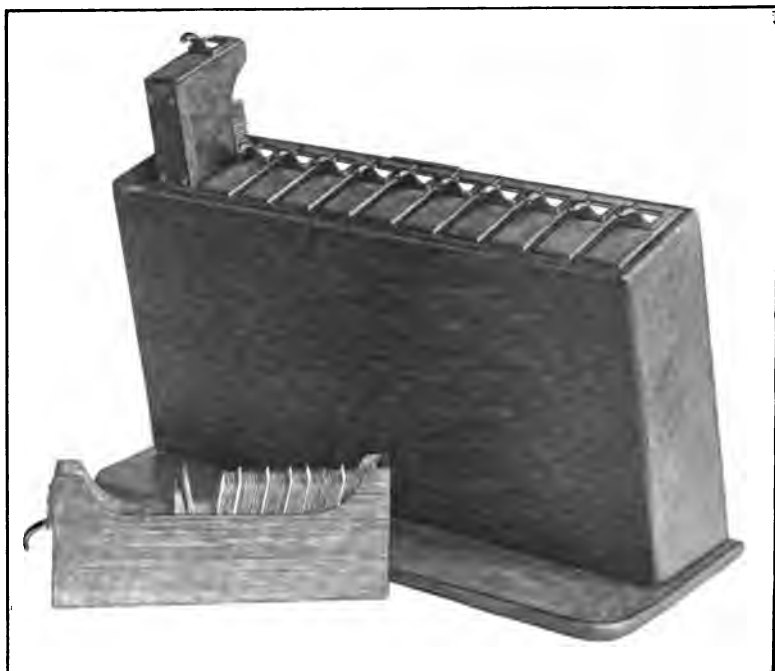
As scientific methods have come more and more into general use, the vexed question of how best to store and classify microscopic slides has become one of personal interest to an increasingly large number of physicians, as well as to all pathologists and biologists. What is needed is, first, a safe and durable receptacle for microscopic slides—compact, yet allowing of expansion without rearrangement, and so convenient that the slides may be used practically as well as theoretically for ready reference. And next, a working classification, easy to understand and use, yet comprehensive and uniform. Though new devices for holding slides and new methods for their indexing appear from time to time, there seems to be no entirely satisfactory solution of the problem.

The cabinet designed by Dr. Kirkbride attempts to meet some of the difficulties, and at the same time to adapt the methods of the card index system to the slides themselves. It consists of an oak case containing a row of narrow drawers or trays just wide enough to hold a slide in an upright position. On the front of each drawer is a combined label-holder and handle of brass. The slide box is patterned after the regular card index cases which are now found in almost every laboratory or office. It is strong throughout—notwithstanding the small size of the trays, which necessitates great care in their construction—and its general finish makes it a distinct addition to the laboratory outfit. The cabinet is made in two sizes of twelve and eighteen drawers, and as each drawer will contain about 110 slides of medium thickness, the cabinets hold (without guide cards) 1320 and 1980 slides, respectively. This is extremely compact, as the drawers are but 24 cm. (9½ in.) deep, and the length of the entire twelve-drawer case is not more than 49.5 cm. (19½ in.). When the large capacity and excellent workmanship are taken into account, the slide box will be found to compare very favorably in cost with others now on the market.

As shown by the illustration, the shell containing the drawers slants forward at an angle of 76 degrees. This makes the trays easier to handle and removes all danger of the slides falling out backward when the trays are taken out or put back, the front of the shell being used

as a kind of pivot upon which the drawer is steadied. The box is so arranged that the slides lie perfectly flat except while in use, and the thickness of the cardboard labels placed at both ends of the slides is just sufficient to prevent the slide above from touching the cover-glass of the slide below. The labels are made of the best quality of white bristol board, which gives an excellent writing surface; for fastening them to the slide, liquid fish glue has been found entirely satisfactory, as it hardens almost at once and holds permanently. The glue comes in convenient bottles, with a brush, and should be kept at hand on the

FIG. 1.



investigator's desk. It need hardly be added that the labels must be placed at both ends, or the slides would lie unevenly (see Fig. 2).

Since the cardboard is glued about 2 mm. from the ends of the slides, each at the top is separated from the next by a small space equal to the thickness of the label, which greatly facilitates "fingering over" the contents of a drawer. When the drawer is out the slides, as in the regular card index cases, slant slightly backward, so that by pushing forward the slide or slides in front the upper label can be read without taking the slide from the drawer. This slant, caused by the triangular block at the back of the drawer, is counterbalanced by the slope of the

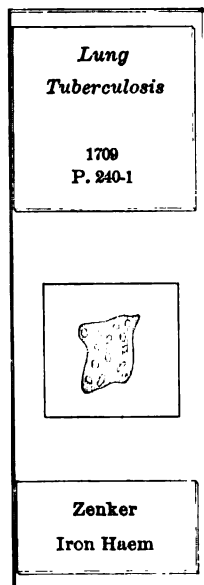
shell, so that when the tray is in place the slides are flat. Besides the triangular block behind there is a second block in the front of each tray which is adjustable; thus it is possible to have the drawer partially filled, or to keep an approximate space for the final slides in any given classification.

The slides are their own index. Briefly, it is a "card index" system in which glass slides are substituted for cards, and the now unquestioned advantages of the card index apply with equal force to the slides; for no matter what classification is used, the collection may expand indefinitely, new slides being added in their proper places, or old ones removed without necessitating any rearrangement. Hence, it becomes a very simple matter to keep the collection "up-to-date" and in good order. Any specimen may be found at once, the label on the drawer and the guide card showing just where to look for it, and the necessity of a card index or reference book is avoided. This adaptability to rapidly growing collections makes the cabinet especially desirable for large laboratories.

The slide case lends itself to any system of indexing, such as decimal, alphabetical, or consecutive numbering. Dr. Kirkbride had spent much time and thought on the classification of pathological specimens, and at the time of his death was at work on a classification which he expected to publish in conjunction with the description of his cabinet. Unfortunately his plan was not perfected, but some general suggestions may prove helpful, the details being left to individual preference.

An alphabetical classification primarily under organs and secondarily under pathological conditions may be used in connection with the cabinets as follows: guide cards or slips are used of the best quality of bristol board, durable but light in weight, to economize space. Main divisions and subdivisions are distinguished by different colors. The slips are the same width as the slides, but project 5 mm. above them, so that the name of the division which each represents can be seen at once. White cards are used for the main or anatomical classification. Immediately behind each of these are slides showing normal conditions of the organ. Then follow blue guide cards in alphabetical order, similar to the white, but giving the pathological subdivisions. For example: Kidney (white card), Amyloid Degeneration (blue card), Inflammation (blue card), Tumors (blue card), etc. The same pathological changes may be present in almost all organs; therefore, the blue

FIG. 2.



guide cards will follow each main division in practically the same sequence. This uniform arrangement adds materially to the ease with which a slide is found. One slide, however, may present two or more pathological conditions, or several changes in a tissue, and cross indexing becomes necessary. Thus a section of kidney with both amyloid degeneration and inflammation should appear in two places. To obviate this difficulty a buff-colored cross index slip is used. The slide itself is placed under "Amyloid Degeneration," while its position and number are given on the cross index slip under "Inflammation." This same card gives references to all sections of kidney classified under other pathological changes, but also showing inflammatory processes. Or it may be expedient at times to keep together slides from a special case, such as an autopsy, even though they are from several organs and show various diseased conditions; here, again, the cross index card is used. In laboratories where the slides are consulted by a number of different persons, and there is danger of their being lost or mislaid, whenever a slide is taken from the collection for reference a salmon-colored removal slip should be put in its place. This gives the date when taken out and by whom, and any other necessary facts. Should a decimal classification be desired, the anatomical divisions would naturally be represented by the whole numbers, the pathological by the decimals.

Sets of blank cards are made by the manufacturers of the cabinet and may be obtained in packages containing fifty cards of one color. A simple but comprehensive working classification giving the main or anatomical divisions is in preparation and will shortly be printed on the white cards. The subdivision cards (blue) are left to be filled out as the character of the collection may require. To supplement the data given on the slides a regular card index is useful. The fixation, embedding, staining, etc., of each specimen are given and a short description of the finished section. Cards for this purpose have been specially ruled for the Polyclinic Laboratories; both these and the slide labels can be had from the makers of the cabinet.

The slide cabinet has now been in use in the Polyclinic Laboratories for over two years, and during that time it has been found thoroughly satisfactory. Thanks are due to Mr. W. E. Parker and Miss M. S. R. James, of Boston, whose interest and valuable suggestions have helped materially in the successful carrying out of Dr. Kirkbride's idea.

FURTHER STUDIES OF GRANULAR DEGENERATION OF THE ERYTHROCYTE.¹

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In a previous communication, read at the meeting of the Association of American Physicians, held at Washington, April 29, 1901, and published in *THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES*,² we presented our investigations of granular degeneration of the erythrocyte and in particular the facts bearing on the relations of this condition to lead poisoning. The material which we then accumulated gave us the opportunity to discuss the morphology of the condition and also its probable etiology. The direct dependence of the degeneration upon lead poisoning was shown by the regular occurrence of the granules in cases of lead poisoning admitted to the hospital, by the invariable discovery of the granules in men working in various capacities in lead works, by the discovery of granules soon after the administration of therapeutic doses in healthy individuals, and by the typical appearance of the corpuscles in dogs subjected to subacute or chronic lead poisoning. Further, we feel prepared to express a positive belief that the granular degeneration is the result of protoplasmic changes rather than the consequence of a nuclear fragmentation. We could see no relation between the granular condition and polychromatophilia excepting a frequent coincidence of the two conditions. Our belief was and remains that this granular condition is a true degeneration of an independent kind. We did not at that time feel prepared to state whether it occurs in lead poisoning as a result of the direct action of lead, or whether it is the consequence of anæmic conditions occasioned by the lead; nor did we then arrive at any conclusion as to the place of origin of the changes found in the circulating erythrocyte. The present communication has for its purpose: in the first place a further discussion of the probable nature of the granules, and in the second place the report of some observations made to determine, if possible, the location in which the changes take place.

To determine the place of origin of the changes, we studied the blood taken from different parts of the circulation as well as scrapings and preparations from the bone marrow and splenic pulp. The material was taken from four animals that had been kept under the influence of

¹ Read at the meeting of the American Medical Association, June, 1901.² September, 1901.

lead for a considerable length of time. First, blood was taken from an artery and vein of the stomach, intestines, liver, spleen, and heart of etherized animals two hours after they had been given large doses of acetate of lead. The animals in each case had been under the influence of lead for some time, and granular erythrocytes were present in the circulating blood. The purpose of this experiment, which was prompted by Grawitz's view regarding the gastro-intestinal origin of the granular degeneration, was to determine whether erythrocytes showing this degeneration occur more abundantly in the blood of the portal vein and its tributary circulations than elsewhere. In only one of our experiments could we recognize an unequal distribution of granular cells. In this case more granular erythrocytes were found in the veins of the spleen, liver, and intestines than elsewhere. We, of course, cannot pretend that this single experience is of much weight in deciding the matter under discussion.

The bone marrow and spleen were examined in two ways: (a) By preparing spreads made from scrapings of the cut surface of the organs, and (b) by embedding portions of the organs, hardened in various ways, in paraffin, and sectioning. In the spreads granular erythrocytes, both nucleated and non-nucleated, were easily demonstrated, but were not more abundant than in the circulating blood. Fixation by heat and in mixtures of ether and absolute alcohol and in absolute alcohol and 1 per cent. formol gave the best results, heat being by far the most satisfactory. The sections did not in any case show granular cells, as the red corpuscles were more or less crenated, whatever the method of fixation. We could not, therefore, be certain of the presence or absence of granular erythrocytes.

The result of these experiments then gave us no indications of the probable place of origin of the granular degeneration. In no instance did we find more abundant granular cells in the bone marrow or spleen than in the peripheral blood.

Some experiments seemed desirable to determine whether the granular cells existed in the circulating blood or whether, on the other hand, they are artefacts. The characters of the granules, the regularity of their occurrence in the blood in certain diseases, as well as their discovery in scrapings of tissues had convinced us that the granular corpuscles are not artefacts, and the question has not been seriously raised. Nevertheless, we thought it advisable to practice injection of certain stains to determine whether the fresh blood-corpuscles after such injections would show granular cells. Dogs that had been kept under the influence of lead until the blood showed granular erythrocytes in abundance were etherized, and solutions of methylene blue or neutral-roth in 0.8 per cent. NaCl solution were injected into a vein. Specimens of blood were taken at various intervals of time from the

ear and examined fresh or after drying and fixation. The injections of methylene blue gave the only positive results. The blood in the successful cases showed nucleated and non-nucleated erythrocytes containing pale blue granules. The appearances of these granular cells were in every respect identical with those of the granular cells seen in stained spreads of the blood obtained before the injection of methylene blue, excepting that the staining of the granules was a little less intense. We could not confirm the observation of Sabrazes, Bourret, and Legér that the granules are eccentrically situated and show a tendency to extrusion.

The dried specimens of blood prepared as above described showed exactly similar conditions.

We have made a few attempts at artificial production of granular degeneration outside the body, and refer to them here, though we are aware of their unsatisfactory character. Quantities of human blood and of dog's blood were mixed with blood serum of dogs that had been under the influence of lead for some time. In no case did we reach positive results. The rapid disintegration of the red corpuscles under such circumstances naturally renders the experiment of little practical value. Equally unsatisfactory results attended the mixture of human blood and dog's blood with various solutions of acetate of lead. Sabrazes, Bourret, and Legér attempted to produce the degeneration by injecting solutions of lead into a ligated vein of a dog's leg, but without success.

A study of the various pathological conditions in which granular degeneration of the erythrocytes occurs seemed to us of possible importance in elucidating the question of the place of origin of the degeneration, and perhaps also its nature. Such a study is of course of even greater and more immediate interest in determining the diagnostic value of this reaction of lead poisoning. We therefore studied a considerable series of cases representing various diseases, and will refer to our results under the headings—chlorosis, pernicious anæmia, leukæmia, and miscellaneous medical and surgical diseases. The results obtained show that no explanation of the nature of the degeneration or its place of origin could be reached in this way.

CHLOROSIS. Contrary to the experience of other observers, we have found granular erythrocytes quite frequently in this disease. Among 18 cases examined by us there were 11 in which the granular cells were abundant. In 6 of the 11, the granules were fine; in the other 5 they were fine and occasionally coarse. In only 1 of the cases were the granules numerous. In this case the hæmoglobin was only reduced to 50 per cent., while in most of the cases a far greater reduction was noted. In all of the 11 cases polychromatic cells were seen, but these seemed to bear no relation in intensity or number to the granular cells.

PERNICIOUS ANÆMIA. Judging from the seven cases which we observed, and from the reports published by others, granular degeneration is a constant condition in advanced stages of pernicious anæmia. We found the granules fairly numerous and at times very large and conspicuous. Though, however, in the number and the character of the granules the blood of pernicious anæmia was usually distinguished from that of other diseases, we did not often find in pernicious anæmia the extraordinary number and variety of granules, coarse and fine, seen in nearly all of the cases of lead intoxication which we have examined; and the other characters of the blood would render a differentiation easy.

LEUKÆMIA. We examined ten cases of this disease and found granules in every one, though they were usually fine and not present in large numbers. We shall have occasion to refer again to this matter, as the occurrence of the granules in leukæmia is of particular importance in connection with the determination of the nature of granular degeneration, nucleated erythrocytes being so abundant in this disease and nuclear degenerations of all kinds so frequent. We should look then to this disease more than in any other for a confirmation of the view that granular degeneration results from fragmentations of the nucleus.

MISCELLANEOUS MEDICAL AND SURGICAL DISEASES. In addition to the 35 cases above referred to, we have examined the blood in 105 patients in the medical and surgical wards of the University Hospital, and have discovered granular erythrocytes in 34 of these cases. In the 34 cases the diagnoses were as follows: typhoid fever, 3 cases; valvular heart disease, 3; peritonitis, 3; septicæmia, 3; tuberculous arthritis, 2; malaria, 2, and pertussis, heart disease and nephritis, lobar pneumonia and pleurisy, phthisis, malignant endocarditis, aneurism and nephritis, nephritis and anæmia, splenic anæmia, secondary anæmia, pseudoleukæmia, chronic diarrhœa, phlyctenular conjunctivitis, orchitis, carcinoma of stomach, lymphoma of neck, sarcoma of neck, empyema, osteomyelitis, each 1 case.

Several observers have reported the discovery of granular red cells in cases of sepsis, and, though the direct bearing of septic infections upon the conditions has not been established, it is of interest to note that this condition was present in some of the cases enumerated above. Perhaps half of the above 34 cases were more or less septic. In the other half of the cases there was no such element, but the high grade of anæmia may have been alone operative in these. In several of the cases no clue whatever to the causation of the granules could be found. We might speak of them as accidental occurrences, but this would not serve as an explanation. In none of the 34 miscellaneous cases were the granules abundant or conspicuous. Two instances in which the

blood picture approached that found in lead poisoning were a case of phlyctenular conjunctivitis in a boy, and a case of chronic diarrhoea in a man who had possibly taken acetate of lead in the treatment of his condition before he came under our observation. Even in these two cases we could easily distinguish the blood from that of lead poisoning, and in the remaining 32 cases the comparative sparseness of the granules and their finer character made the distinction very easy.

There were 71 cases in which we did not find granules. Some of these may be mentioned for the sake of showing that the classes of diseases in which the granules occur or do not occur give no indication of the probable source of the granular cells; and also to show that the condition of the blood as to infection or anæmia will not alone explain the degeneration.

We failed to find any granules in the following cases: 5 cases of valvular heart disease, 4 of phthisis, 4 of appendicitis, 7 of typhoid fever, 5 of sepsis, 6 of grippe, 3 of carcinoma of the stomach, 3 of diabetes, 2 of pneumonia, and in one case each of malaria, splenic anæmia, glanders, actinomycosis, and tetanus.

With the single exception of lead poisoning, no condition that we know of regularly causes this change, though other conditions operate to this end in occasional cases. As far as lead is concerned, we may refer again to our previous studies. In each of seven clinical cases of plumbism we found enormous numbers of the granular cells; and we may add six recent instances. In twenty-one men working in lead works, but showing no symptoms of lead poisoning, the granules were always found, but in varying numbers. Two of these men had worked in the lead works only four days. Moritz found the granular cells in six men working in lead works, but having no symptoms; and other observers, as Strauss, Grawitz, Behrendt, Sabrazes, Bourret, Legér, and Hamel (the last in 25 cases) found the granules in cases of lead poisoning. Strauss also discovered them in atropine poisoning and in rabbits and frogs poisoned with pyrodine. Kaminer and Rohnstein found granular cells in the blood of rabbits poisoned with phenylhydrazin on the fourteenth day of the intoxication. Sabrazes, Bourret, and Legér refer to a case of fatal copper poisoning in which numerous granules were found, but they failed to produce the degeneration in guinea-pigs by intraperitoneal injections of various toxic and non-toxic substances, such as distilled water, thallium acetate, carbonate of lithium, and sulphate of atropine, or by inhalations of nitrite of amyl, pyrodine, and phenyl hydrazin. Injections of lead acetate, however, caused the degeneration very rapidly in guinea-pigs as well as in rabbits. In our own experiments we have found the degenerations after poisoning a dog with potassium chlorate; the granules did not appear until shortly before death, but became very numerous. Another

dog slowly intoxicated with corrosive sublimate showed only a few granules shortly before death. They were also present in large numbers in the blood of dogs kept under the influence of toxic doses of pyrodine for many days. It is evident, therefore, that toxic causes of various sorts are capable of producing the granular degeneration we are studying, but it is equally evident that no poison thus far studied is as regular in its production of the degeneration or as prompt in its action as is lead.

The discussion of the nature of the granular condition of the red corpuscles has divided observers into two groups: those holding that the granules originate from the nucleus, and those who regard them as the product of a specific degeneration of the protoplasm.

The former view was the one first put forward after the granular cells were recognized, and has been lately defended by Askanazy, Lazarus, Engel, Litten and Strauss. These authors look upon the granules as the result of nuclear fragmentation—*karyorrhexis*—and they believe that it is the means by which the nucleated cell normally loses its nucleus before becoming an adult cell and entering in the peripheral circulation. The attractiveness of this theory is largely dependent upon its adaptability as an explanation of the disappearance of the nucleus, a matter which has remained unsolved in spite of painstaking examinations of histologists; but more definite facts are required to establish the truthfulness of the theory. It would seem entirely reasonable to expect blood pictures showing a gradual transition from the state of normal nucleated cells to that of erythrocytes containing scattered granules, but without a nucleus. As a matter of fact, Litten claims that he has seen direct transitions from the normal nucleus to fully granular cells, both in the circulating blood and in the bone marrow, and he states that the nucleus gradually becomes paler as the granules increase in number and size. Somewhat similar statements are made by Ewing. Litten's observations were made in only one case, a rapidly fatal one of pernicious anæmia. Our own observations have included a large number of cases of intense anæmia and of various intoxications, both clinical and experimental, and we have found in these abundant granular cells in the circulating blood as well as in the bone marrow and other organs, but we have never in a single instance among the thousands of granular cells that we have carefully examined found the slightest suggestion of a nuclear fragmentation productive of granulations. We have repeatedly found and figured, from paintings by an artist and from photographs, associations of nuclei and granules, that is, we have recorded the occurrence of granules in nucleated cells; but in every instance the periphery of the nucleus has either been clear-cut as in the normal nucleated cell, and the chromatin of the nucleus has presented normal appearances, or when degeneration has been

present there has been nothing suggesting even remotely an origin of granules in degeneration of the nucleus. The conditions in the blood of leukæmia seems to us to be particularly important in this connection. Nucleated erythrocytes with and without granules were very abundant in some of the cases studied, and in the same cases we often found evidences of nuclear degenerations, both in the white and in the red corpuscles. In no instance, however, could we see evidence of a transition of nucleated cells to granular cells.

If the theory of the gradual fragmentation of the nucleus were established, we should expect the evidences of such fragmentation to be most abundant in the bone marrow, where nucleated cells become non-nucleated; but our experience has been that the nucleated cells and the granular cells in bone marrow have been exactly the same as those of the peripheral circulation.

An observation made by us in several instances of still greater significance to disprove the nuclear origin of the granules was the discovery of karyomitosis in granular erythrocytes. One observation of this sort seems to us more valuable than any amount of negative evidence or of theorizing. It is unlikely that a nucleus could at the same time be in a state of active mitosis and of karyorrhexis.

The second theory regarding the nature of the degeneration, and the one to which we incline, is that which has been defended by Grawitz, Moritz, Hamel, and Bloch, and which attributes the granular condition to a peculiar protoplasmic degeneration. In the consideration of this theory we would first of all direct attention to the fact that all of the protoplasmic degenerations of erythrocytes that have been thus far described are characterized by the development of a basophilic tendency. Thus, in the studies of Maragliano and Castellino particular attention was directed to this peculiarity of the degenerated areas, and the behavior of polychromatophilic cells, so generally considered degenerative, is very similar. In no case is there the distinct basic affinity of the degenerated parts of the cell that is seen in the granular degeneration under consideration, but this is a point of too little importance to disprove our view.

Grawitz did not consider the granules the results of nuclear fragmentation, because in his earlier work he had not seen these granules in nucleated red corpuscles. He had expected to find them if these granulated cells were the unfinished red cells prematurely placed in the peripheral circulation. Under such circumstances granular nucleated red corpuscles should be found, or at least transitional forms should occur. Later he found nucleated erythrocytes with these granules in the protoplasm, but the nucleus was to all appearances normal. This last observation induced him to study the blood-forming organs, but here he was unable to see any evidence of these granules. From these

observations he came to the conclusion that the granules are the result of degenerative processes in the hæmoglobin, caused by poisons acting directly upon the erythrocytes, and suggests that this takes place outside of the blood-forming organs.

Our own studies have shown granular nucleated erythrocytes in the peripheral blood, and also granular erythrocytes, nucleated and non-nucleated, in the bone marrow; but, like Grawitz, we have failed to find any transitions from the non-granular nucleated erythrocytes to the granular non-nucleated cell. While the proof of the protoplasmic origin of the granules is therefore more or less negative or indirect, we believe the evidence against the nuclear origin of the granules is direct and positive. This evidence may be summarized as follows:

1. Karolytic and karyorrhexic changes may be observed in nucleated red cells without showing any granular change in the protoplasm of the cells; on the other hand, granular degeneration may accompany these nuclear changes without association of the nuclear and granular processes.

2. The granulated red cells (coarse or fine granules) never show the remains or a suggestion of a former nucleus.

3. The granules are observed in karyokinetic red cells, and we have seen them associated with the several stages of the dividing nucleus. We cannot believe that such a progressive and retrogressive change can be present in the nucleus at the same time, without internal evidence of degeneration.

4. The very early appearance of these granules in the blood taken from the peripheral circulation (twenty-five hours after a dose of 7½ grains of the acetate of lead has been taken by one of us) to a certain extent indicates a probable beginning of the destructive changes in the erythrocytes (non-nucleated) of the peripheral blood, rather than in the erythrocytes (nucleated) at the moment in process of formation in blood-making organs.

5. The granules observed in the bone marrow were absolutely the same as those seen in the peripheral blood. Those in the nucleated cells, chiefly normoblasts, showed no evidence of derivation from the nucleus, the nuclei being in each case normal in size, shape, and staining qualities and like those of the neighboring nucleated cells which did not contain granules.

6. Finally it seems to us that in certain cases of leukæmia in which great numbers of nucleated red cells are always present, if these granules were nuclear derivatives, distinct steps or transitions could be demonstrated. Such is not the case. On the contrary, distinct degenerative changes, karyorrhexis, karyolysis, pyknosis, atrophy of the nucleus, etc., are present sometimes with and sometimes without granular protoplasm; but there are never in our experience any

transitional stages to indicate gradual destruction of nuclei with liberation of substance that has gone to form granules. In addition very many of the nucleated cells showing nuclear degeneration contained no granules.

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THE LATERAL CHAIN THEORY OF EHRLICH AS EXPLANATORY OF TOXINS, ANTITOXINS, BACTERIOLYSINS, AND HÆMOLYSINS.

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THE last three years of Continental activity have added so much to our knowledge of the blood in both normal and immunized animals that a retrospect and conclusions by the one who has been most closely identified with progress along these lines is particularly grateful. Ehrlich in several recent communications has reiterated his "lateral or side chain" theory as explanatory of toxins and antitoxins, and found new data in his and others' works on bacteriolysins and hæmolysins from which he has enlarged and made more conclusive this admirable theory of cell mechanism.

TOXINS AND ANTITOXINS. Toxins are labile, poisonous substances, elaborated by plant and animal cells, of which we have no exact chemical knowledge, but whose characteristics are recognizable by their physiological effect on animals. Toxins differ from all known chemical poisons in these important particulars: 1. They are subject to cell assimilation, that is, they enter into direct chemical combination with protoplasm, whereas chemical substances either destroy the cell or are present in it in solution. 2. Toxins can call forth the production of antitoxins in the animal body, while chemical poisons cannot. 3. The action of toxins is delayed by a considerable incubation period.

Toxins which have undergone change by time or certain chemical substances form substances known as toxoids, which possess an equal power of stimulating the formation of antitoxins, but have relatively little toxic effect.

Antitoxins are substances formed in the animal body by the inoculation of toxins or toxoids; they are recognized by their action in neutralizing the effect of toxins. It was at first supposed by some that these bodies were direct reaction products of toxins, but Knorr soon showed that one unit of toxin gave rise to an amount of antitoxin sufficient to neutralize 100,000 such units—a disparity inconceivable in a process analogous to a chemical reaction. The variation in duration of active and of passive immunity, the presence of normal antitoxic bodies, and the increased production of antitoxin under the action of pilocarpine are all observations which make it certain that antitoxins must be regarded as products of cell activity. Roux, Metchnikoff, and others maintained that the neutralization of toxin by antitoxin is due to a stimulation of somatic cells, more particularly the phagocytes of the circulating blood, which enables them to counteract the poisonous effect of the toxins. But it has been shown by Madsen, Ehrlich, and C. J. Martin that the neutralization will take place *in vitro* irrespective of the presence of animal cells. This chemical form of neutralization of the toxins, together with the whole explanation of the formation of antitoxins, is reasonably and adequately accounted for by Ehrlich's "lateral chain" theory, as will be seen.

Virchow pointed out in his *Cellular Pathology* that chemical substances were picked out by certain organs. The almost absolute absorption of the toxins of tetanus and diphtheria by nerve cells are further instances of the elective action. The obvious example of the variation of leucocytes in their reaction to stains is but a further step in cell differentiation. It is on this basis of finer cell structure, as suggested to us, that Ehrlich has conceived each cell as possessing, in addition to its nucleus, multitudes of atomic "lateral chains" or "receptors," each with its affinity for a certain assimilable substance. The normal function of the receptors lies principally in this choice of suitable food-stuffs, but toxins as assimilable substances are also taken up by receptors fitted for the purpose.

The toxin unit must now be considered as more than a simple body; it possesses, in fact, at least two parts, a "haptophore" group, which unites it to the cell receptor, and a "toxophore" complex, which is the poisonous element. Toxoids as altered toxins possess only the haptophore group, which accounts for their absence of toxic effect. The body cell is more or less injured if the united unit be a toxin,

owing to the toxophore group; but even if there be no toxophore group present, as in a toxoid, the receptors united are thrown out of function. Loss of the cell constituents in either case leads to regeneration of the parts affected, and regeneration of tissues, as Weigert has shown, takes place always in excess. This excess of toxin elective "receptors" cumbers the cells, and they are eventually thrown off into the circulating blood, where they form the antitoxins. A toxin cannot harm a cell unless its haptophore group unites with a cell "receptor;" when "circulating receptors" or antitoxins are present in the blood the haptophore groups become anchored and never reach the cell, which accounts for the neutralization of the action of toxin by antitoxin. Absence of "receptors" suitable to unite with toxins explains the natural immunity of certain animals to certain toxins. Predominance of suitable receptors accounts for the election of certain toxins by certain tissues. A multiplicity of toxic bodies such as may occur in a single bacterial toxin simply calls for a corresponding multiplicity of receptors.

The period of incubation is the time during which the "receptors" are regenerating in the cells and before they are thrown off into the circulation. That the origin of antitoxins is primarily in the fixed cells is further shown by the fact that organs acquire antitoxic properties before the blood itself.

"Receptors" which unite with toxins are called "receptors of the first order," being relatively simple and having only one receptive process. Similarly toxins are "uniceptors," that is, they have only one uniting arm, the haptophore group. This conception is better understood on comparison with the structure of the hæmolysins and their "receptors."

BACTERIOLYSINS AND HÆMOLYSINS. In 1894 Pfeiffer noted when the living vibrios of cholera were mixed with the serum of animals immunized against cholera, and the mixture injected into the peritoneal cavity of a normal guinea-pig, that within an hour the bacteria were broken up and destroyed, that is, bacteriolysis had taken place. Metchnikoff soon showed that this could be produced *in vitro* with immune serum which had been kept for some time, provided that the fresh peritoneal exudate of a guinea-pig were added. Bordet then showed that agglutination and bacteriolysis would take place with fresh immune serum alone. With serum which had stood for some time or which had been heated to 55° C., for twenty minutes, however, the power of bacteriolysis was lost, while agglutination was retained; and then, curiously enough, he found that the addition of fresh serum of a normal animal would restore the bacteriolytic power. From these data it was easy to conclude that bacteriolysis depends on two elements: first, a

specific body acquired by immunization which is not destroyed at 55° C. ; and, second, a substance present in all normal sera, destroyed by heat or by standing. A similar bacteriolytic power which frequently occurs in normal sera only shows that a natural immunity exists to a certain extent.

It has long been known that the serum of certain animals has the power of breaking up or hæmolizing the red blood-corpuscles of other animals, causing the opaque solution of normal defibrinated blood to become laky and transparent by the dissolving out of the hæmoglobin from the discoplasm. Such occurrences give rise clinically to the accidents of transfusion. It was shown first by Belfanti and Carbone, and later, in 1898, by Bordet, that this hæmolytic power can be artificially produced in practically any animal. If, for example, a guinea-pig is inoculated with the red blood-corpuscles of a rabbit, its serum soon acquires a marked power to agglutinate and hæmolyze the rabbit's red blood-corpuscles. In parallelism with the power of bacteriolysis, guinea-pig's serum hæmolytic for rabbit's red blood-corpuscles loses its power when heated to 55° C. for twenty minutes, and regains it when normal, fresh, guinea-pig serum is added. To the analogous immune bodies in bacteriolytic and hæmolytic sera Bordet gave the name of "*substance sensibilisatrice*," from his conception of it as acting merely as a fixative agent which renders it possible for the thermolabile substance, the "*alexine*," to attack and destroy the cell. This use of the word "*alexine*" is confusing, as the same name has been used by Buchner to indicate the substance causing bacteriolysis before this causative element was shown to be composed of the two substances mentioned above.

Ehrlich was able immediately to confirm Bordet's experiments, but instead of accepting the latter's conclusions, showed at once the relation of hæmolysis to—and its confirmation of—his already elaborated lateral chain theory. Both from his own data and conclusions, and from the experiments of Bordet and others, Ehrlich in the past few years has developed a concept fascinating in detail and increasingly more convincing.

As already shown, a hæmolytic serum contains two substances, to which Ehrlich has given the names of "*immune body*" and "*complement*." The immune body has two affinities, a stronger one for the red blood cell, and a weaker one for the complement. At 0° C. the immune body will unite with the red blood cell, causing no hæmolysis, and leaving the complement free in the solution. The union can be proved by repeatedly washing in normal saline the centrifuged red blood-corpuscles to get rid of all complement, and then adding at room-temperature fresh normal serum containing complement alone; hæmo-

lysis takes place, showing that the red corpuscles contain a united immune body.¹

The immune body having two uniting processes is an "amboceptor." The complement resembles in its constitution and action a toxin; it has a haptophore group, which unites with the immune body, and a "zymo-toxic complex," which acts on the red blood cells saturated with immune body in a manner partially toxic and partially fermentative. The complement can unite with red blood-corpuscles only in the presence of the immune or intermediary body. Just as the inoculation of toxins gives rise to antitoxins, so inoculations with "hæmolysins" give rise to antihæmolysins, which are more complex, however, being composed of "anticomplements" and "anti-immune bodies." There is reason to suppose that although the heating of hæmolysins destroys the action of the complement, that in reality it is not wholly destroyed but forms a "complementoid," which, as in the case of toxoids, can form anti-complements.

We have seen that receptors of the first order have but one haptophore group, which in the case cited united with a toxin. Red blood cells, in common with other cells, contain these receptors of simpler structure, and, in addition, "receptors of the second order" with which we have to deal in the phenomena of hæmolysis.² Receptors of the second order have two haptophore groups, one of which unites with a nutritional substance (immune body), and the other with a fermentative substance (complement). That these receptors occur in excess, at least in the case of hæmolysis, is shown by the fact that the red blood cells in the absence of complement will fix many times the amount of immune body necessary for complete hæmolysis. The reproduction and throwing off of these receptors gives rise to amboceptors in the blood, in the instance which we are following, to antihæmolysins. And in general it may be said that any substance which has one or more haptophore groups can give rise to antibodies, which are either uni-ceptors or amboceptors, depending on the number of haptophore groups in the causative substance.³

The formation of antitoxins by means of toxins, bacteriolysins by means of bacteria, hæmolysins by means of red blood-corpuscles, is

¹ The immune body, on account of its acting position between the complement and the red blood cell, is also referred to as "intermediary body."

² Ehrlich, in the "*Schlussbetrachtungen*," adopts a slightly different classification. In this communication only he has referred to the receptors mentioned above as "receptors of the third order," and interpolates another class. The function of these other receptors is that of breaking up the more complex nutritional molecules, for which purpose they possess two arms, one, a haptophore group to unite with the molecule, and the other a "zymophoric complex," which exerts a fermentative action on the attached substance.

³ Ehrlich has of late used the term "haptines" to denote in general any thrown-off receptors.

but the overproduction of normally existing cell elements. Natural immunity is but an expression of a relatively large number of such preformed elements. Furthermore, each complement, each immune body, and so forth, is individual and gives rise to a specific antibody. That a certain interaction of relatively specific substances is possible is easily explainable. For example, isohæmolysins are producible, that is, hæmolysins acting against the blood of other animals of the same species; but they will hæmolize the red blood-corpuscles of only certain of these animals, namely, of those having red blood-corpuscle receptors very similar to or identical with the receptors of the blood giving rise to the hæmolysins. Incomplete hæmolysis is, perhaps, due either to merely similar receptors or to an insufficient quantity of identical receptors. Bordet has steadfastly maintained the unity of the complement, but neither his experiments nor his conclusions seems confirmable.

CYTOLYSINS. As might be expected from the effect of red blood cells, many animal cells can be shown to produce in the blood of inoculated animals substances which will destroy these cells, or cytolytins. In such a way spermotoxin (or spermolysin) was produced by Metchnikoff, epitheliolysin by von Dungern, nephrolysin by Lindeman, and leucocytolysin by Metchnikoff. And in analogy with the hæmolysins the various cytolytins can produce antibodies such as anti-spermotoxin, etc. The exact parallelism of construction and formation of such bodies with the hæmolysins, according to Ehrlich's theory, need only be indicated. Whether isocytolytins can be formed is as yet problematical.

AGGLUTININS AND PRECIPITINS. The phenomena of hæmolysis and bacteriolysis are usually accompanied by the presence of a marked agglutination of the bacteria or the red blood-corpuscles as the case may be. But such coagulative phenomena are entirely independent of any destructive property in the serum, as is shown by the fact that perfect bacteriolysis or hæmolysis may occur without any agglutination, and the well-known fact that red blood-corpuscles are agglutinated by practically any normal sera which, however, may possess no hæmolytic power. Agglutination, then, is simply a parallel phenomena in the course of immunization, and, according to Walker, in the case of bacteria, parallel with the antimicrobial rather than with the actually protective power of the serum. That agglutination will assist bacteriolysis and hæmolysis is quite probable. The exact mechanism of the process is unknown; Grüber has regarded it as due to some deleterious effect on the membrane of the bacteria which makes them sticky, while others have explained it on a chemical basis, inasmuch as similar effects may be produced by very dilute chemical substances, such as chrysoidin.

In 1897 Krause showed that antitoxins frequently gave a precipitate when mixed with their own toxins, but never with other toxins. Bordet then showed that absolutely specific precipitating bodies could be produced by inoculating rabbits with the milk of various animals. Meyers and also Uhlenhuth have found precipitins for peptones, and also for various kinds of egg albumin, the latter, however, not being absolutely specific, owing, doubtless, to the similarity of constituents in certain of the albumins employed. Bordet in his first observations on hæmolysis noted also the concurrence of a precipitate. Wasserman and Schütze, in 1900, found that this precipitate was specific, for rabbits when inoculated with various bloods gave sera which precipitated only the blood used. In the case of human blood the observations of these authors, together with those of Nuttall, make it probable that we have here a sure method of differentiating human blood and blood stains from the blood of other animals, excepting that of the monkey.

According to the Ehrlich hypothesis agglutinins and precipitins are uniceptors, composed of a haptophore group and a "coagulative complex."

Apart from its theoretical interest, this concept of Ehrlich as we have outlined it is of twofold value: it gives us a rational basis for serum therapy as we have it, and suggests by analogy the problems of the serum therapy that may arise.

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REVIEWS.

UTERINE TUMORS, THEIR PATHOLOGY AND TREATMENT. By W. ROGER WILLIAMS, Fellow of the Royal College of Surgeons. London, Paris and Madrid: Baillière, Tindall & Cox, 1901.

THIS book, which is quite up to the high standard of the author's previous works, is perhaps the best monograph on the subject which has yet appeared. It is truly a philosophical treatise, and the object has been the generalization and reduction into order of the masses of chaotic facts which may be found scattered through medical literature on the subjects of the pathology and treatment of uterine tumors.

The origin of uterine tumors is shown to be more closely associated with embryology and the aberrant elements, or so-called "rests," than with bacteriology; at the same time, the complications which arise as a result of bacterial influences are admitted. Uterine fibroids are properly termed "myomata," and there are several excellent chapters on their pathogenesis, morphology, biology, and complications.

The question as to whether benign tumors are peculiarly apt to become malignant is settled in the negative, and while it is admitted that myomata and malignant disease are sometimes concomitant in the uterus, it is pointed out that in such cases the two neoplasms are generally quite separate and independent, and that myomata are far less liable to originate cancer than are the glandular elements and embryonic remnants or "rests" in the uterus itself.

The chapters on the operative treatment of myomata and cancer are clear and reflect the best teaching of the day. Preference is given to the abdominal route, whenever practicable, in both classes of cases. The extra-peritoneal treatment of the cervical stump in the abdominal wound after supravaginal amputation is admitted to be obsolescent if not yet obsolete in conservative England.

Hysteromyomectomy with intra- or rather subperitoneal treatment of the stump, which should always be associated with the name of Bear, is credited to Schroeder, and in the chapter on the operative treatment of cancer no mention is made of combined vaginal and abdominal hysterectomy, an operation which is of distinct advantage in many cases.

The chapters on morphology and the general pathology of uterine cancer are most interesting and instructive. The author in 160 personal consecutive cases of cancer of the uterus found only 4 of the corpus, or 1 in 40; but he points out that these cases were not specially selected for their operative suitability. He believes that 90 per cent. of all uterine cancers are of the cylinder-celled (mucosal or glandular) variety, which he says should be regarded as the typical form for this organ. He points out that in this conclusion he differs from Cullen, who out of 141 cancers of the lower part of the uterus found that 123 were of the squamous and only 18 of the glandular type, and he says

that such a result could only be arrived at by interpreting the histological appearances differently to what he has done, and he believes that Cullen's interpretation is erroneous. The book contains 58 engravings which fairly well illustrate the text.

J. B. S.

THE PRINCIPLES OF PATHOLOGICAL HISTOLOGY. By HARVEY R. GAYLORD, M.D., Professor of Surgical Pathology in the University of Buffalo, etc., and LUDWIG ASCHOFF, M.D., Professor and First Assistant in the Pathological Institute of the University of Göttingen. With an Introductory Note, by WILLIAM H. WELCH, M.D., Professor of Pathology in the Johns Hopkins University. Illustrated with 81 engravings in the text and 40 full-page plates. Philadelphia and New York: Lea Brothers & Co.

The plan of this work is substantially that of a laboratory guide. In the first chapter we find directions for the preparation and examination of tissue, both fresh and hardened. The methods given are well selected, and include practically all of the important special stains now in use, as well as the standard stains generally used in laboratories. The directions are clear and full. The second and largest part is devoted to special pathological anatomy, the classification being based on organs, including the mucous membranes and the serous membranes—with tumors and blood. The laboratory idea is still further suggested by summaries of normal histology preceding the descriptions of the alterations of each organ. The work does not, therefore, aim at a complete exposition of morbid histology, yet the authors have included a wide range, and the reader can usually find information on any point, while the descriptions are admirably clear and detailed. The plan followed involves the further risk of not impressing the student sufficiently with the principles of general pathological anatomy, a risk that has not been so successfully avoided, as in the case of tubercle. In this part of the book we find its chief novelty, perhaps even its *raison d'être*, in the numerous heliotype reproductions of photomicrographs. With very rare exceptions these are admirably chosen and perfectly reproduced, and form a noteworthy addition to medical illustration. They compare favorably with the more costly reproductions that have been used in special works, and show the practical possibility of something far better than the half-tones now so much used. Very few of the illustrations are actually poor, but the one showing typhoid bacilli with flagella cannot be otherwise described, and the other photographs of bacilli in the same plate do not exceed mediocrity. As a rule, the amplifications are well adapted, but the important picture of miliary tuberculosis of the lung (Fig. 4, Plate XIII.) might well have been more highly magnified. It would have been more satisfactory if the amplifications had been expressed in diameters rather than in general terms, as high, low, etc. Although such works as this have a very important place, they do not give one a belief in the absolute superiority of photographs over good drawings as aids to the study of histology. We think we have noticed a greater tendency to neglect the art of focusing among students who use text-books with photomicrographic illustrations. A single plane

or even a foggy preparation often seems satisfactory to them. For special purposes, however, and with proper instruction, the newer method has a most important place. The illustrations in the text in the present work are, as a rule, good. Fig. 18, however, does not show much. The tricolor photographs will be examined with great interest, and those who are not aware of the progress of this art will be surprised at their excellence. But a comparison with the lithographs will show, as might be expected, that the former are not yet able to replace drawings, and are far from sufficing to settle doubt as to the nature or even the existence of newly discovered histological elements. The short section on Blood, by Dr. Irving Phillips Lyon, is very satisfactory as far as it goes. The addition of a chapter on the Principles of Optics and Photomicrography, with a brief account of reproduction processes, is a most happy one, and makes still more desirable this valuable and timely work. The only serious fault we have found is the very incomplete index, but this, and some typographical errors, can easily be remedied. Mechanically, the execution of the book is most satisfactory, and the cost is relatively so moderate as to ensure the wide use the work deserves.

G. D.

HANDBOOK ON SANITATION: A Manual of Theoretical and Practical Sanitation for Health, Sanitary, Tenement-house, Plumbing, Factory, Food, and other Inspectors, as well as for Candidates for all Municipal Sanitary Positions. By GEORGE M. PRICE, M.D., Medical Sanitary Inspector, etc. New York: John Wiley & Sons, 1901.

THE author states in his preface that he has been induced to undertake the work by reason of the "very few sources where the necessary knowledge of sanitation can be gained by those who propose undertaking sanitary inspection as a livelihood," and expresses the hope that he has presented the subject in such form that the student and candidate may make a creditable showing in the civil-service competitive examinations and obtain the desired position. He says truly: "The first step in the study of sanitation is to understand the principles of the science," and then proceeds to demonstrate with unusual clearness that his posing as a teacher is an admirable example of the blind leading the blind. Perusal of the book by one having even a superficial knowledge of hygiene can hardly fail to excite wonder that so much misinformation and nonsense can have been compressed into so small a space without entangling a larger measure of fact. Though doubtless well intentioned, the work is undeserving of serious notice except in so far as it is important to warn against all such short cuts to scientific knowledge, which are capable of doing much injury. A critical review of the very numerous chapters—most of them, fortunately, very brief—would require more space than can ordinarily be accorded a work of the highest excellence, and a few examples will, therefore, have to serve as an index of the general worthlessness of the entire work.

We are informed that "pathogenic bacteria are either those formed during the progress of organic decay . . . or those which become lodged in the soil through the contamination of the latter by groundwater and air, and which find in the soil a favorable culture medium

until forced out of the soil by the movements of the ground-water and air." We learn that "the peculiar disease called cretinism, as well as goitre, has been traced directly to a certain chemical composition of the soil," and must regret that he who holds this secret should fail to reveal it in more explicit terms. Contrary to what is now generally accepted, it appears that malaria and other paroxysmal fevers, tuberculosis, rheumatism, neuralgia, etc., are directly due to high-water level and marshy ground. Among the diseases due to soil influences are cholera, plague, yellow fever, and septicæmia. Under "Air," we learn that the greatest amount of carbonic acid "in confined spaces after the air has been breathed by people" is 6 parts in 10,000, and that "the carbonic acid content of air is taken as the standard of impurity," when we have supposed it to be an index of the amount of contamination. Bacteria appears in one place to constitute a kingdom by themselves and in another to be a part of the animal kingdom. Thus, "the impurities in the air according to their substance and character are as follows: mineral, vegetable, animal, bacteria, and gases," and among the animal particles found in air the author includes "the various micro-organisms." Carbonic acid in large amounts is said to be a "violent poison."

Although it may be true that "we may truthfully say that there is hardly a disease which is not directly or indirectly caused by the impurities found in the air," we have as yet no very direct evidence that such diseases as chronic interstitial nephritis, locomotor ataxia, cirrhosis of the liver, gonorrhœa, and syphilis are air-borne, nor can we reconcile the statement with the author's assertion that goitre, rheumatism, malaria, tuberculosis, neuralgia, cholera, plague, yellow fever, and septicæmia are due to soil influences.

Under "Ventilation," we learn that, "as a rule, the height of a room ought to be about one-third of the cubic space," and since the air space needed is said to be 1000 cubic feet, we are led to infer that the height of the room should be "about one-third," or 333 $\frac{1}{3}$ cubic feet. Perhaps he means the cube root. The three forces mentioned as the active agents of ventilation are "diffusion, motion, and gravity."

Under "Water Supply," it appears that artesian wells are those over fifty feet in depth; that boric acid is one of the chemical agents used in the purification of water; and that Ancus Martius began the first Roman aqueduct two years after the generally accepted date of his death.

Nobody will question the statement that "the amount of refuse and garbage in cities is quite considerable," but not all will admit that the chief constituents of sewage include potash and phosphoric acid. If "sewer gas" is present in a room a piece of unglazed paper, saturated with a solution of acetate of lead and hung therein, "will be immediately blackened," but, we may add, not unless there is also present in full operation some form of sulphuretted hydrogen generator.

It appears that milk contains 12.83 per cent. of solids, divided as follows: fat, 3.69; carbohydrates, 4.88; salts, 6.71; total, 15.28—and no proteids. The salts have been supposed hitherto to amount to about 0.70, and milk has been noted as a valuable proteid food. Chalk is mentioned as an adulterant of milk, although never found. The lactometer is mentioned as a valuable assistant in milk inspection, and the instrument is shown in an illustration. Whatever the instrument from

which the drawing was made may be, it certainly is not a lactometer, and cannot be used as such. It appears to be a portion of a complicated piece of chemical apparatus, whereas a lactometer is exceedingly simple in construction.

As a whole, while the book may not cause the unskilful to laugh, these not being able to note its grotesqueness, it certainly is calculated to make the judicious grieve.

C. H.

ATLAS AND PRINCIPLES OF BACTERIOLOGY. By PROFESSOR DR. K. B. LEHMANN and R. O. NEUMANN, Drs. of Philosophy and Medicine. Edited by GEORGE H. WEAVER, M.D. In two Parts. Philadelphia and London: W. B. Saunders & Co., 1901.

LEHMANN and NEUMANN's work is now well known and its value established. It is an excellent work ; with certain limitations it covers the field satisfactorily, and the illustrations in the Atlas leave little to be desired. We believe that accurate drawings for the purpose of studying bacteria are more valuable than photographs. This is particularly true in the representation of cultures, a fact to which Lehmann calls especial attention in his preface. There is some question whether the plan adopted here of representing all cultures upon a black background is entirely satisfactory. The greatest virtue of any drawing is the accurate representation of nature, and any form of diagram is a poor substitute unless the actual picture is so complex that its essential details cannot be appreciated by the uneducated eye. It is possible that the mechanical difficulties in the reproduction of the cloudy amber-color of the agar culture media, or the transparency of the gelatin culture media was so great that the present method was on the whole better. As far as it goes it is exceedingly satisfactory, and perhaps, for purposes of diagnosis, it is all that could be demanded. Practically all the important organisms are represented, and in such a variety of forms and cultures that any other Atlas would rarely be needed in the ordinary hospital laboratory.

The most interesting part of such a work is of course the general section ; the most useful, the special section. Lehmann's article on the general aspects of bacteriology is one of the most satisfactory that we have ever read. It is thorough, approximates completion sufficiently ; it is clear, and devotes itself rather to facts than to theories. The discussion of immunity is perhaps somewhat briefer and less thorough than the other parts of the book. The subject of agglutination receives such scant consideration that it might well have been omitted. Widal, who has certainly done more to promote the utility of this reaction in typhoid fever than any other man, is barely mentioned, and then only in connection with one of his subsidiary suggestions. In the article upon the typhoid bacillus the statements on this subject, in view of its extreme importance, are even less adequate than in the general section. In the discussion of the classification Lehmann is at his best. He places it upon a clear scientific basis, recognizes the difficulties, suggests methods for overcoming them, and, as far as possible, urges the great importance of a thorough study of the individual species of bacteria.

While not advocating names etymologically incorrect, he does not believe in changing those already suggested, because, after all, the important thing is to designate the genera and species and not to avoid hybrids of Greek and Latin. The classification which he adopts is based upon rational principles, and, as a matter of fact, is one of the most satisfactory at present in use. The subdivision of the family *bacteriaceæ* into the bacterium without endogenous spores and bacillus with endogenous spores will be difficult to carry against the habit of the world of calling all rod-shaped organisms bacilli. We agree with Lehmann that any practical division into the pathogenic and non-pathogenic bacteria is absurd. There is no scientific distinction between the pathogenic and non-pathogenic excepting in regard to their action upon certain living organisms, and it is not impossible that there are few species that are not pathogenic for some form of life. On the other hand, there is accumulating evidence that the so-called pathogenic and non-pathogenic bacteria may pass one into the other. The descriptions of the individual species are concise, clear, and thorough; the translation is fair. There is a distinct flavor of the original German, such as complicated adjectival phrases and a persistent use of the inverted sentence. We think that in another edition the book might be improved in this respect, and yet it must be admitted that the language is quite comprehensible, although we doubt whether it would be so to a reader not familiar with the German language.

J. S.

CARE AND TREATMENT OF EPILEPTICS. By WILLIAM PRYOR LETCHWORTH, LL.D. New York and London: G. P. Putnam's Sons, 1900.

THIS work is not medical in the narrow sense of the word. It does not tell what physic to give epileptics. It is a careful study of the question of what the State and charitable persons owe to the epileptic as an unfortunate who, not being able to care for himself, must be guarded and cared for. It is a very careful study of a serious social problem. The author gives an account of what measures have been taken in the several States and in foreign countries to aid epileptics by institutional treatment. It is to the discredit of America that the problem has been more successfully treated in several European countries than by us. Indeed, the first attempts began abroad, and have only been slowly followed here. No class appeals more worthily for aid, and yet, as compared with the insane and feeble-minded, they have been sadly neglected. Every State should have an institution in the country—a large farm, not a mere house of detention—where patients could live a hygienic life, work out-of-doors, and have rational and healthy amusements. Apart from the moral duty we owe to unfortunates as a business question, it probably would be cheaper to properly care for epileptics than to neglect them. Prisons and houses of correction, institutions which cost money to support, contain not a few epileptics who pass in and out repeatedly, or, finally, get themselves hanged, which also costs money. It might be cheaper to prevent crime by proper care. How much has been done and how much more remains to be done this excellent book well shows.

C. W. B.

DIFFERENTIAL DIAGNOSIS OF THE SEPARATE FORMS OF GALLSTONE DISEASE. By PROF. HANS KEHR (Halberstadt). Authorized translation by WILLIAM WOTKYNs SEYMOUR, A.B. Yale, M.D. Harvard. With an introduction by Prof. Kehr. 12mo. Pp. 370. Philadelphia: P. Blakiston's Son & Co., 1901.

PROF. KEHR divides this very excellent book into two parts, Part I. consisting of four lectures on the pathology, the examination, the special diagnosis, and the treatment of cholelithiasis, while Part II. gives the clinical and operative histories of one hundred cases, illustrating the various conditions and difficulties which the disease may present. In addition there is an appendix containing the histories of eighteen more operative cases, with remarks on inflammatory jaundice, and insidious infection of the biliary system.

In the introduction Kehr states that he has operated 547 times for gallstones, and his remarks and conclusion on the diagnosis and treatment of cholelithiasis are based on this enormous and unprecedented experience. It would, therefore, be idle for us to criticise his opinions on diagnosis or operative technique, for the experience of surgeons in this country is limited to dozens of cases, where he deals in the hundreds. In his opening lecture he takes the ground that few clinicians have had the experience to make an exact diagnosis in cholelithiasis, for the reasons that the experience of the majority is limited to cases subjected to medical treatment or those that come to the post-mortem table. The book, therefore, is intended primarily to assist the general practitioner in making an exact diagnosis, and detailing to him the brilliant possibilities which surgical treatment holds out.

Kehr states that the symptoms of gallstone disease arise almost invariably as the result of inflammation and infection; in other words, that gallstones may remain an indefinite time in the gall-bladder without occasioning a symptom until a trauma or infection occurs. The pain experienced, he explains, is due either to the swelling of the gall-bladder and ducts or to adhesions binding these tracts to neighboring organs. That pain is excited by the mere presence of the stone, he considers most unlikely and very exceptional, even when the stone is lodged in the common duct. He also states that the majority of the pains which are called cramps of the stomach, occurring in adults, are of gallstone origin.

Under the heading of special diagnosis, the subject-matter is extremely good and rich in ideas, but the arrangement of it is possibly not as systematic as it might be. A concise table in this chapter gives twelve different forms of the disease with their prominent differential symptoms, and their appropriate treatment.

The chapter on treatment represents the best modern views. He does not advocate operation in every case where gallstone is diagnosed, believing that many mild cases can be relieved under appropriate medical treatment. At the same time he gives a clear picture of the results of prolonged medical treatment in cases where the knife should have been used. He, therefore, takes the middle ground between von Winniwater and Kraus, the former believing that the diagnosis of gallstone should indicate operation, and the latter that surgical intervention should be sought only upon a vital indication. He sums up the action of the Carlsbad treatment with Sprüdel water in a few words: It

allays the inflammation of the biliary passages and renders the disease latent, freeing the patient from pain, but has no power to dissolve or remove the stone. In other words, its action is like a hot poultice or opium.

His operative statistics are interesting. When the gallstones are only found in the gall-bladder his mortality has not yet exceeded 1 per cent. Where it is necessary to excise the gall-bladder the mortality reaches 3 per cent., but when the common duct has to be incised it rises to 10 per cent. The troubles which sometimes develop after successful operation he ascribes usually to adhesions, the fixation of the gall-bladder to the abdominal wall, etc., and not to stones left behind or again developing. Many points in his operative technique will appeal to the surgeon. His strong objections to exploratory puncture of the gall-bladder or massage of that organ when distended are well taken, and he places such procedures under the heading of technical sins.

Dr. Seymour has accomplished a good work for the profession in reproducing in English the experiences of so eminent an authority, and he is to be congratulated upon the literalness of the translation.

R. G. LE C.

THE PRINCIPLES OF HYGIENE: A Practical Manual for Students, Physicians, and Health Officers. By D. H. BERGEY, A.M., M.D., First Assistant, Laboratory of Hygiene, University of Pennsylvania. Octavo volume of 495 pages, illustrated. Philadelphia and London: W. B. Saunders & Co., 1901.

THIS very excellent and valuable book presents in compact form a very clear exposition of the general principles of hygiene. The author disclaims in his preface any attempt to treat the subject exhaustively, his object being "to give the general principles upon which the health officer and the physician work in their respective capacities in dealing with conditions which are detrimental to health or which tend to improve it."

The subjects considered include air, ventilation, heating, water and water-supply, removal and disposal of sewage, garbage disposal, food and dieting, soil, habitations, vital causes of disease, disinfection, quarantine, vital statistics, exercise, clothing, and military, naval, personal, industrial, and school hygiene, all of which are presented clearly, concisely, and attractively. Especially to be commended is the chapter on vital causes of disease, the consideration of immunity and susceptibility being handled with signal ability and the whole subject presented in better form than, in the opinion of the reviewer, ever before in a work of similar character.

The book is faulty in some minor respects, such, for instance, as including with implied approval the arbitrary standards of purity of drinking-waters, which have long since been abandoned as unscientific and untrustworthy; but, as a whole, it commends itself for simplicity and accuracy. The appendix, containing rules for interchange of different expressions of results obtained in analyses, for conversion of thermometric degrees, etc., is, however, singularly unfortunate, since wrong

factors are given for converting nitrogen to nitrous acid and nitric anhydride, the rules for converting thermometric degrees of one scale into those of another are wholly wrong, and the United States gallon has been confused with the imperial gallon, which measure is not used in this country. C. H.

HAND ATLAS OF HUMAN ANATOMY. By WERNER SPALTEHOLZ, with the advice of WILHELM HIS. Translated from the third German edition by LEWELLYS F. BARKER, with a preface by FRANKLIN P. MALL. Vol. I., Bones, Joints, Ligaments. Leipzig: S. Hirzel. New York: G. E. Stechert, 1900.

THE German edition of this most excellent text-book has long been familiar to students of anatomy, and it is the experience of its practical value that has led the well-known teachers of anatomy, whose names figure upon the title-page, to further its publication in English. The special feature of the book is to be found in the illustrating. As in most German text-books of anatomy, the text plays a subordinate part, and the student is taught the appearance of the anatomical structures by his sense of sight and not by his ability to memorize anatomical terms. The plates are most accurate and have been executed with the greatest care, the illustrations of the present volume being reproduced from the original plates. It is doubtful whether any book on anatomy can ever altogether supplant his Gray in the heart of the American medical student of the present generation, but Spalteholz, without supplanting the other book, will make a useful companion to it. J. H. G.

THE MEDICAL NEWS POCKET FORMULARY. By E. QUIN THORNTON, M.D., Demonstrator of Therapeutics, Pharmacy, and Materia Medica in the Jefferson Medical College. Philadelphia: Lea Brothers & Co.

IN the preface to his first edition the author says that "no man of independent thought will be hindered in his development by having the best information placed conveniently at hand."

That the medical profession agrees with the above statement of the author is shown by the publication of the fourth edition of the book.

The little volume contains besides the large collection of formulæ a comparative table of the metric system, a list of the important incompatibles, a synopsis of the treatment of poisoning by the various drugs, and a complete table of the doses of the drugs of the Pharmacopœia expressed in the metric system as well as in the usual apothecaries' method. Due space is also given in the collection of formulæ to the more recent preparations. One of the most important points to the physician after he has made up his mind as to the proper drug to order in any case is how best to exhibit it, and the author has expended great care as to the palatability and pharmaceutical elegance of the preparations without, however, sacrificing therapeutic efficacy. W. R. N.

INTERNATIONAL CLINICS. Edited by HENRY W. CATTELL, A.M., M.D., Philadelphia, with the Collaboration of Various Authors. Eleventh series. Vol. III., pp. 303; Vol. IV., pp. 302. Philadelphia: J. B. Lippincott Co., 1901-1902.

IN the six hundred pages of these two volumes we find many articles of great practical, and some of permanent, value. The subjects are ample in range and in the main well presented. Clinical Aspects of Spa Treatment, by Robinson; Localization of Nervous Diseases, by Wiener; Clinical Laboratory in Private Practice and in the Physician's Office, by Camac; and Some Results of Microbic Infection in Urinary Disease, by Heming, are of marked interest. Winged Insects and their Larvæ as Parasites of Man, by Walsh, is both readable and instructive, as is his other paper on Prophylaxis and Early Diagnosis in Heart Disease; Palpitation and Organic Disease; Tobacco and Heart Lesions; Cure of Heart Lesions. Broca's plea for Small, Repeated Doses of Solutions of Mercurial Salts in treating Syphilis, Guinard's Modified Technique in the Spinal Injections of Cocaine, and Jonnesco's Splenectomy for Malarial Cachexia, are important contributions, the last two suggestive. Burnett points out the dangers of too strong solution of adrenalin chloride, and Taylor illustrates Deformities in Children from the stand-point of the General Practitioner. This article is doubly interesting from its graphic descriptions and because it not only demonstrates that the general practitioner exists, but, as well, that he is observant and practical. We wish that Newton had treated pulmonary osteo-arthritis more fully, although his carefully reported case is a real addition to the somewhat meagre literature. We have on other occasions spoken favorably of this series, and we have criticised when on one occasion a volume did not meet our expectations. Of those under consideration we believe that a full measure of value is given, and the various authors have ably seconded the editor in his efforts to make *International Clinics* represent the present status of the healing art.

R. W. W.

GYNECOLOGICAL PATHOLOGY: A MANUAL OF MICROSCOPIC TECHNIQUE AND DIAGNOSIS IN GYNECOLOGICAL PRACTICE, FOR STUDENTS AND PHYSICIANS. By Dr. CARL ABEL, Privat-Dozent, Berlin; translated and edited by SAMUEL WYLLIS BANDLER, M.D., Adjunct Gynecologist to Beth-Israel Hospital, New York, with a chapter on the Embryology of the Female Genitalia and the Pathological Growths Developing from Embryonal Structures. Illustrated by one hundred engravings. New York: William Wood & Co., 1901.

THIS translation brings within the reach of the English student a book which was perhaps the first on the subject in any language, and which immediately took its place as a standard work in the comparatively new field of gynecological pathology. The translator has added an excellent chapter on embryology and on the origin of growths from embryonal cells and organs.

Part I. contains 21 pages, and deals with the technique of obtaining and handling the material for examination. The directions are concise and are the result of a large experience. The few illustrations in this part are unnecessary.

Part II., of 143 pages, is on diagnosis, and deals with the macroscopical and microscopical appearances of the various pathological conditions of the vulva, the vagina, the cervix uteri, the uterus, the tubes, and the ovaries. Special attention is directed to microscopical illusions, and the student is warned to be on his guard, especially in making the diagnosis of carcinoma of the cervix. Before considering the pathology the normal anatomy of each structure is given. There is an excellent chapter on the normal anatomy of the uterus during menstruation, during the first months of intra-uterine pregnancy, and in extra-uterine pregnancy.

Part III., 59 pages, has been added by the translator, and deals with the embryology of the female genitalia and the pathological growths developing from the embryonal structures. The subject is made unusually clear, and the illustrations are selected from such well-known authorities as Waldeyer, Keibel, Kollman, His, Lemon, Schultze, and Nagel.

The book is intended to serve as an introduction to practical experience in gynecology, and is the result of a large clinical experience. The value of the book would be greatly increased by more liberal illustrations of the various microscopical appearances described.

J. B. S.

TEXT-BOOK OF PHYSIOLOGY. Edited by E. A. SCHÄFER, Professor of Physiology, University of Edinburgh. Vol. II. Edinburgh and London: Young J. Pentland. New York: The Macmillan Co., 1900.

THE first volume of Schäfer's *Physiology* appeared several years ago, and it is pleasing to observe that the second volume of this encyclopedic work is fully as interesting and valuable as the first. The authors of the various articles in this volume are: Leonard Hill, W. H. Gaskell, J. B. Haycraft, E. H. Starling, J. Burdon Sanderson, Francis Gotch, E. A. Schäfer, J. N. Langley, C. S. Sherrington, W. H. R. Rivers, John G. McKendrick, and Albert A. Gray, all of whom may be regarded as authorities on the various matters of which they treat. The contents of the volume include many interesting articles on the various portions of the circulation and the nervous system, and the sections on the organs of special sense; the latter are particularly to be commended as possessing great value to the general practitioner or specialist who desires to look up some physiological data in regard to the senses of vision, hearing, or smell.

The book may be regarded as probably the most complete exposition in English of modern physiological research. It does not wholly answer to our idea of a text-book, as it more corresponds to a system than to a book for use by students. It makes a bulky volume, but when the value of its contents is considered one is glad that no attempt was made to condense the subject-matter.

G. M. C.

MEDICO-SURGICAL ASPECTS OF THE SPANISH-AMERICAN WAR. By LIEUTENANT-COLONEL DR. NICHOLAS SENN, Chief Surgeon U. S. V.; Chief of Operating Staff with the Army in the Field; Professorial Lecturer on Military Surgery, Chicago University. Chicago: American Medical Association Press, 1900.

THIS book is largely a reproduction of a number of articles which have appeared from the pen of its eminent author. Where subjects of practical surgical importance are under consideration, its contents are most interesting. The descriptions of the organization of various branches of the hospital service in a large army should be read by all who are interested in the subject, containing as they do much practical advice drawn from the author's personal experiences. Camp hygiene is fully entered into. The proper location and construction of sinks, the various sources proper to be utilized for water-supply, and the practical care of the men is gone into in detail. Particularly interesting are Dr. Senn's descriptions of various cases which came into his hands for operation and treatment. The Spanish-American war offered an excellent opportunity for the study of wounds produced by modern small arm projectiles, and Dr. Senn utilized that opportunity to its fullest extent.

The book, however, has certain very obvious defects. It contains many irrelevant and poorly executed illustrations, such as portraits of President McKinley and General Shafter; of Miss Wheeler attired as a hospital nurse, and of groups of hospital stewards and medical officers of the Illinois National Guard, and a portrait of the wife of the Governor of the State. It bears evidence also of hasty composition. As over two years have elapsed since the termination of the war, it certainly seems a pity that the distinguished author did not see fit to revise and edit his manuscript and eliminate much useless material for the sake of that which was of value.

The typography of the book is lamentably deficient. A number of pages could be picked out in which lines are entirely separated from their context, thus, on page 12, there stands as a lonely sentence, "There was probably never a day when their duty." No explanation can be found anywhere throughout the book as to what this sentence refers to.

F. R. P.

THE PATHOLOGY AND SURGICAL TREATMENT OF TUMORS. By N. SENN, M.D., Ph.D., LL.D. Second edition; revised. Philadelphia and London: W. B. Saunders & Co.

RECENT work done in Rome upon the parasitic origin of malignant tumors has been added to this volume. A new section has been added upon sarcoma of the decidua.

This work of Professor Senn is most comprehensive and exhaustive in its scope. After considering general questions of the origin, morphology, anatomy, and pathology of tumors, tumors of plants and animals are described. Then the clinical appearances in general, the diagnosis, prognosis, and treatment in general are considered.

The succeeding chapters describe each variety of new growth sepa-

ately as it occurs in the different tissues and organs of the body of the human individual. For example, under adenoma is the heading mammary gland, and under this heading will be found all that is of value concerning adenoma of the mammary gland, so that the book is a splendidly arranged reference-book upon tumors.

The importance of careful teaching in the diagnosis of tumors can hardly be overestimated. In few medical schools is the diagnosis of tumors taught systematically and with proper clinical material. The chapter upon the diagnosis of tumors, p. 88, is full of suggestions to the teacher of clinical surgery. The sense of sight and touch should be exercised to their full extent before additional assistance is sought in aspirators and harpoons, etc. The physical conditions of the growth are very important in determining its nature. Operative procedures requiring especial manipulations are described in connection with the organ involved. That the teaching of the diagnosis of tumors should be taught as a special branch in our medical schools is coming gradually to be recognized. This book of Dr. Senn's very admirably emphasizes the importance of this branch of surgical pathology. The number of original and new illustrations is commendable.

C. L. S.

THE AMERICAN YEAR-BOOK OF MEDICINE AND SURGERY FOR 1902. A yearly Digest of Scientific Progress and Authoritative Opinion in all branches of Medicine and Surgery, drawn from journals, monographs, and text-books of the leading American and foreign authors and investigators. Arranged, with critical editorial comments, by eminent American specialists, under the editorial charge of GEORGE M. GOULD, A.M., M.D. In two volumes: Vol. I., including General Medicine, octavo, 700 pages, illustrated; Vol. II., General Surgery, octavo, 684 pages, illustrated. Philadelphia and London: W. B. Saunders & Co., 1902.

UP to the present date no better substitute for the much to be lamented *Index Medicus* than the form of publication known as the year-book has presented itself. There are many varieties of publication which aim to present in compact form the gist of the most valuable contributions to medical literature; thus most of the medical journals have an abstract department, aiming thereby to present to their readers an epitome of current literature as it appears; but this can never be so satisfactory a method as that which combines in book form and under classified headings the same kind of material.

The *American Year-Book* is issued in two volumes: one entitled "General Medicine," the other "General Surgery." These volumes are sold separately, and thereby enable the purchaser to procure the one which contains those subjects in which he is especially interested. It is impossible to attempt an analytical review of such a *mélange* of riches as each volume presents. The contributions to them are not merely abstracts from literature, but they constitute critical essays by competent authorities on the different subjects considered.

The work is a standard, and its possessor may congratulate himself upon having on his book-shelves the sum and substance of all that is really worthy of preservation out of the vast mass of medical literature which has appeared during the past twelve months.

F. R. P.

DISEASES OF THE NOSE AND THROAT. By D. BRADEN KYLE, M.D., Clinical Professor of Laryngology and Rhinology, Jefferson Medical College, Philadelphia; Consulting Laryngologist, Rhinologist, and Otologist to St. Agnes' Hospital. Second edition, revised. Octavo, 646 pages; over 150 illustrations and 6 lithographic plates. Philadelphia and London: W. B. Saunders & Co., 1901.

THE first edition of Dr. Kyle's book well merited the success which has brought about the publication of the second edition. The second edition has been carefully revised and several minor errors which appeared in the former have been corrected.

The grouping and classification of the various pathological conditions met with in the nose and throat is particularly to be commended. Some exception may be taken to the author's system of nomenclature, but Dr. Kyle is a trained pathologist and his definitions and studies are based upon thorough pathological research.

There is a notable absence of reference to foreign literature in rhinology, but it is well compensated by the large number of American authorities whose work in this specialty is acknowledged and referred to.

It may be regarded as one of the standard American text-books on the subject of rhinology. F. R. P.

THE RÖNTGEN RAYS IN MEDICINE AND SURGERY. By FRANCIS H. WILLIAMS, M.D. 8vo., pp. 639 and index. Fully illustrated. New York: The Macmillan Co., 1901.

IN discussing the application of this new method of diagnosis, Dr. Williams has followed the trend of his previous writings, and shows a decided preference for the development of technique in fluoroscopic examinations. He undoubtedly ranks among the first in the acquirement of an ability to see and interpret what he sees upon the fluorescent screen. He devotes the major part of this work to the description and discussion of his apparatus and methods and their result. This work is particularly valuable to those desirous of following his methods and of acquiring such technique and the ability to see and interpret the transient images seen with the fluoroscope.

The description of the static machine which he favors is given in detail, but too little space is devoted to the description of other forms of apparatus and other tubes than those he prefers to make the book "A Guide to Practitioners and Students" in this method of diagnosis. It should have dealt with the apparatus and technique of other investigators to a fuller extent if he desired it to be universal in its application. As it is, it is a guide to the technique and practice of the author, and as such is very valuable.

While discussing the application of this method in surgical diagnosis, it fails to give sufficient assistance to the student in detailing the technique and methods of making the skiagraphs which afford the basis of accurate and reliable diagnosis. C. L. L.

VENEREAL DISEASES: A MANUAL FOR STUDENTS AND PRACTITIONERS.

By JAMES R. HAYDEN, M.D., Chief of Clinic and Instructor in Venereal and Genito-urinary Diseases at the College of Physicians and Surgeons, New York, etc. Third and revised edition. Philadelphia and New York: Lea Brothers & Co.

It affords us considerable satisfaction to review a third edition of this little treatise, which we have for some time considered to be one of the best of all the manuals published upon the subject. It is a work eminently adapted to the needs of the medical student in particular, giving, as it does, in the words of the author, "in a clear and compact form, a practical working knowledge of gonorrhœa, stricture, chancroid, and syphilis." Several new sections have been added, and the whole volume has been sufficiently revised to bring it thoroughly up to date.

The fad known as the "Janet method" for the treatment of gonorrhœa receives unqualified condemnation at the hands of the author, who advises a well-known and rather old-time method of treatment, which he styles a "rational" treatment. The chapters devoted to stricture and syphilis give in a concise manner most of that which students and general practitioners need to know upon these subjects. Indeed, this little volume will prove of great value to all those members of the profession too busy or disinclined to study more pretentious works on venereal diseases.

H. M. C.

MANUAL OF PHYSICAL DIAGNOSIS. FOR THE USE OF STUDENTS AND

PHYSICIANS. By JAMES TYSON, M.D., Professor of Medicine in the University of Pennsylvania and Physician to the University Hospital; Physician to the Philadelphia Hospital; Fellow of the College of Physicians of Philadelphia; Member of the Association of American Physicians, etc. Fourth edition, revised and enlarged. With colored and other illustrations. 18mo., 298 pages. Philadelphia: P. Blakiston's Son & Co., 1901.

With each succeeding edition the subject-matter of this well-known manual has been carefully scrutinized, revised, amplified, and amended as seemed necessary to secure completeness and that detail possible in a book of its size. In the fourth edition the statements are accurate and the entire subject of physical diagnosis is presented in a manner attractive to the student.

The value of the book is increased by short chapters containing brief directions for examining the blood and the gastric contents and for making a necropsy. As a whole the book is unsurpassed by any of its size.

A. O. J. K.

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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AND

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ASSOCIATE PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

On the Presence of the Bacillus of Eberth in the Blood and in the Rose-spots during Typhoid Fever.—COURMONT (*Journal de physiologie et de pathologie générale*, 1902, vol. iv., p. 155) has made careful studies of the blood in all cases of typhoid fever entering his wards during the year 1901, nine in number. The typhoid bacillus was found in the circulation in every instance, and in many cases it was found at several different times in the course of the disease, leading the author to the conclusion that it is present in the blood as a rule in typhoid fever. In no instance did he obtain other than pure cultures. The bacillus may appear at an early period, inasmuch as it was found in one case on the fifth day. In order to obtain these results it is necessary to use a considerable quantity of blood in a large quantity of bouillon. In fourteen instances in which several drops of blood were inoculated in about 20 c.c. of bouillon only three positive results were obtained. He is inclined to believe that one should employ 300 c.c. to 500 c.c. of bouillon, which should be inoculated straightway from the syringe and introduced immediately into the thermostat. The results are by no means as good when the blood has been allowed to clot before taking the culture.

In some instances at the end of twenty-four hours the bouillon may seem sterile, but on shaking it up thoroughly a growth usually follows. From 2 c.c. to 4 c.c. of blood should be used.

The virulence of the bacilli obtained was rather marked. Their agglutinability was almost invariably markedly inferior to that of bacilli which had been maintained for a long time in artificial cultures. They were, for instance, agglutinated commonly at a dilution of 1:50 by a serum which agglutinated laboratory bacilli at 1:200. These characteristics were retained by the second or third generations. After many generations, however, they became less agglutinable. The typhoid serum acts apparently in the same

manner upon its own bacilli as upon others. There is no special sensibility of the bacilli in any given case to the serum of that particular patient.

There appears to be no relation between the presence of the typhoid bacilli in the blood and the agglutinating power of the serum. In four of the nine cases the bacilli were obtained while the agglutinating power of the serum was absolutely *nil*. The author concludes: "A search for the bacillus of Eberth in the blood by the above-mentioned method should be undertaken to make an early diagnosis of typhoid fever in every instance in which the Widal sero-diagnosis is negative."

Yet more remarkable are the results reported by POLACCO and GEMELLI (*Centralbl. f. innere Medizin*, 1902, vol. xxiii., p. 121). These observers have made a series of cultures from the rose-spots in fifty typhoid patients during an epidemic lasting from July until November, 1901. They used the method of Neufeld with certain modifications. The most important points they believe to be the necessity of taking a little of the tissue as well as the blood from the rose-spot and to use fluid culture media. In obtaining the substance they used the "Vaccinostyle Maréchal." They took their cultures always from at least two spots. The method is so simple that it is practicable with even small children. Positive results were obtained in every case, in many instances before the Widal reaction had appeared.

[It will be interesting to see whether these surprising results will be confirmed by other observers. Cole, in Professor Osler's clinic, has been able to demonstrate the presence of typhoid bacilli in the circulating blood by methods very similar to those of Courmont in about 80 per cent. of the cases, not infrequently before the development of the Widal reaction or before the appearance of roseola. A simple and practical method of demonstrating the presence of typhoid bacilli in the circulation in the first week of the fever would be of inestimable clinical value.—W. S. T.]

Organo-therapy in the Treatment of the Fatty Stools of Pancreatic Disease.—SALOMON (*Berlin. klin. Wochenschrift*, 1902, vol. xxxix., p. 45). A few observers have already pointed out that in cases with fatty stools, in the absence of jaundice, due inferentially to pancreatic disease, the administration by the mouth of fresh pancreas has resulted in marked improvement. The more careful observations of von Noorden indicated that commercial preparations of pancreatin are without essential value, that only the fresh pancreas is of use. Weintraud also observed that while preparations of pancreatin were of some value in furthering the absorption of albuminoids, yet they helped but little toward the absorption of fat.

Salomon reports two extremely interesting cases. The first was that of a woman, aged fifty years, who consulted him in June, 1900, having suffered for over a year from occasional attacks of diarrhoea and sensations of unrest in the abdomen, with a loss of twenty pounds. The stools contained large quantities of fat. A slight degree of glycosuria was present, which disappeared on diminishing the carbohydrates. The patient was given a careful diet in which the exact amount of fat and nitrogen was estimated. After a period of preliminary observation fresh pancreas was administered. It was clearly demonstrated that this treatment greatly increased the absorption of both fats and albuminoids. The character of the stools entirely changed,

and the distressing symptoms disappeared. Later on pancreatin prepared by Hausfeld in St. Gallen was administered in Sahli's glutoid capsules without essential effect upon the absorption of fats, although, as in the case of Weintraud, the nitrogen absorption was appreciably helped. On the other hand, the administration of 0.25 pancreatin of the Rhenania-Aachen firm, five times a day, resulted in marked benefit, though somewhat less than in the case of treatment with the fresh gland. A new preparation by the Rhenania firm, a compound of pancreatin and 10 per cent. tannin, to which the name pankreon has been given, appears also to have good effect, while in that it is unattacked by the gastric juice, it need not be given in glutoid capsules. In a second case similar results were obtained.

In connection with these cases the author points out another very interesting observation. Recent works have shown that the acetone body in diabetes probably arises from fat. If this observation be true one might expect that in cases like the above the formation of acetone should go hand-in-hand with the greater absorption of fat. This proved to be true in Case II., where the quantity of acetone was materially increased by the administration of pankreon, which, as has been mentioned, increased greatly the absorption of fat.

Intestinal Perforation by *Ascaris Lumbricoides*.—SOLIERI (*La Riforma Medica*, 1902, vol. xviii., p. 280) reports a case of a man, aged sixty-seven years, who, in perfect health, was seized with sudden abdominal pain, followed by evidence of an acute peritonitis. Laparotomy was performed twenty-seven hours after the onset. The peritoneum was found to be full of pus, feces, and ascarides. In a loop of the small intestine not far from the cæcum and opposite the mesenteric insertion was a round perforation from which feces and ascarides escaped. This was excised and an artificial anus made. The patient died nine hours later.

On microscopical examination it was found that the perforation was not in a Peyer's patch. The mucosa was normal throughout its extent and everted at the point of the ulceration, so that at some points it almost came into contact with the serous membrane. The submucosa and the muscular coat are cleanly cut through, normal histologically, and buried under the mucosa. The subserous and the serous coats showed changes dependent upon the peritonitis. Cocci and bacilli were found in scanty numbers on the surface of the mucosa and the serous membrane. Micro-organisms were not abundant in the tract of the perforation, not accumulated in colonies or deep in the tissues. The absence of small-celled infiltration of the internal and middle coats, the absence of necrotic or hemorrhagic areas, of tubercles, of marked changes along the course of the tract of the perforation, and the normal condition of the vessels were sufficient to exclude typhoid, tuberculosis, or inflammatory perforation. The author, therefore, considers himself justified in assuming that the perforation did not occur through a diseased area, but was the result of an "act of violence," which was exercised during a period of time so short as not to give time for manifestations of reactive inflammatory processes on the part of the tissues. In addition to this, in a number of the sections there were found under the serous coat distinct sections of an ascaris. A similar observation of an ascaris in the tissues of the

intestine has been made by Stork. The few cases in literature in which perforations by ascarides have been reported are referred to. Inasmuch as many of these have been doubtful in nature, this instance, which would seem to be unquestionable, is of considerable interest.

The Value of Subcutaneous Gelatin Injections in Melæna Neonatorum.—HOLTSCHMIDT (*Münchener med. Wochenschrift*, 1902, vol. xlix., p. 13) reports five cases of melæna neonatorum occurring in the gynecological clinic at Dresden during the year 1901, in which subcutaneous injections of a 2 per cent. solution of gelatin were used. During the seven years from 1894 to 1900 seven of the fourteen cases of melæna occurring in this clinic resulted fatally. All of the five cases occurring in 1901 recovered. As a rule, 15 c.c. of gelatin solution were used, and in two instances only was this repeated. While four of the cases positively recovered, one child left the hospital on the ninth day in a very serious condition, although there had been no hemorrhage for five days.

Early Syphilitic Hydrarthrosis.—DE GRANDMAISON and BODIN (*Archives Générales de Médecine*, 1902, p. 58) report an interesting case of acute hydrarthrosis of the left knee-joint in connection with secondary syphilis. This appeared simultaneously with the roseola, headache, dysphagia, and loss of hair, four weeks after the appearance of the initial lesion, and two months later, with a relapse after insufficient treatment, it returned a second time, entirely disappearing under satisfactory treatment on each occasion. The fluid from the knee-joint, which was of a lemon-yellow color, showed in the centrifugalized sediment, leucocytes and red corpuscles in the relative proportion of 70 and 80 per cent. These leucocytes showed lymphocytes, 73 per cent.; mononuclears, 11 per cent.; polymorphonuclears, 16 per cent. Ten cm. of the fluid were injected into a guinea-pig without result.

Literature contains so few cases of this nature that the authors deem it worthy of publication, and conclude that when one meets with joint swelling during secondary syphilis he should consider it in all probability luetic in nature, and that by the adoption of this treatment:

"1. He will cure his patient as far as the manifestations of syphilis may be cured.

"2. He will have occasion to control the exactness of his diagnosis in virtue of the old adage, *naturam morborum ostendunt curationes*."

On the Pathogenic Importance of Balantidium Coli.—HENSCHEN (*Arch. f. Verdauungs Krankheiten*, 1901, vol. vii., p. 501) reports two new instances of chronic diarrhœa apparently dependent upon the presence of balantidium coli. He has observed altogether eight similar instances. In five of these cases a rapid recovery followed the disappearance of the parasites. In one case with severe symptoms and a great number of balantidia in the stools there was an early improvement, but the patient failed to continue the treatment. In another instance recovery began about a month after the disappearance of the parasites, and in a third the diarrhœa continued despite the disappearance of balantidia. In this case, however, there had been a chronic colitis of two and a half years' duration before the beginning of the

treatment. The author believes that during this time the intestine had suffered changes sufficient to account for the duration of the symptoms. He concludes: "One is fully justified in assuming that in these cases balantidia were the cause of the diarrhoea, and that the main indication in the way of treatment is their destruction."

In all of these cases the treatment advised by Waldenstroem and Henschen, namely, large enemata (2500 c.c.), to which 50 to 75 c.c. vinegar and 5 to 7.5 tannin were added, resulted in a rapid disappearance of the parasites.

Recent observations appear to have settled beyond a doubt the question as to the pathogenicity of this parasite.

STRONG and MUSGRAVE (*Bulletin of Johns Hopkins Hospital*, 1901, vol. xii., p. 31) report from the Philippines a case of diarrhoea resulting fatally in four months, in which the stools contained great numbers of balantidium coli. The blood showed an increase in the number of eosinophilic cells. At necropsy the lower part of the jejunum and ileum were reddened and contained considerable mucus. In the large intestine the mucosa throughout was reddened and covered with bloody mucus; there were also a number of shallow ulcers. Balantidia were demonstrated throughout the mucosa and passing through the submucosa and muscularis.

A most elaborate study of this question is that of SOLOWJEW (*Centralblatt f. Bakt.*, 1901, vol. xxix., pp. 821, 849), who has also demonstrated that balantidium coli may enter into the healthy mucosa between the glands. "Making their way further into the submucosa they increase markedly and bring about there also extensive changes. Hence, they penetrate the muscularis mucosae between the muscle bundles. The most marked changes being observed in the submucosa, the necrosis begins here. This spreads in all directions, reaching on the one side the muscular coat, on the other the mucosa. The glands deprived in this spot of their proper nourishment become necrotic if they have not already been destroyed by the parasites.

"Thus, the general opinion that balantidium coli is met with only on the surface of the mucosa, where it feeds upon the mucus, cannot be considered correct. The presence of the parasites in the depths of the tissues explains the malady produced by them, as well as the tendency to frequent relapses after their apparent complete disappearance."

This article is accompanied by a full table of references.

[There would appear to be little specific in the clinical manifestations of these cases which, in some instances, present the symptoms of a chronic diarrhoea, in others of an acute dysentery, with mucus and bloody dejecta. The reviewer has seen some of Strong's specimens which are convincing as to the pathogenicity of the parasite.—W. S. T.]

The Etiology of Acute Dysentery in the United States.—Under the direction of FLEXNER, VEDDER and DUVAL (*Journal of Experimental Medicine*, 1902, vol. vi., No. 2, p. 181) have undertaken to solve two problems: 1. To determine by comparative study whether the organisms described by Shiga in Japan, by Flexner and Strong in the Philippines, and by Kruse in Germany are of the same species, and 2, to discover the cause of acute dysentery in the United States, and if possible to identify it with the organisms of the observers mentioned. In other words, to determine

whether acute dysentery is the same the world over, and whether it be due to a specific organism, bacillus dysenteriae Shiga.

After briefly describing their technique they state that before the organism under consideration can be considered to be bacillus dysenteriae, it must have fulfilled the following requirements :

- a. It must give the proper cultural characteristics, as shown by standard cultures of Shiga, Flexner, and Kruse.
- b. It must possess the right morphology, as shown by the same.
- c. It must give a positive agglutinative reaction with the same of the known dysenteric sera.

Their material was drawn from various sources. The bacillus dysenteriae was grown from the stools of five different cases studied in various Philadelphia hospitals. It was also obtained in three cases of dysentery which occurred in the Lancaster County Almshouse and Insane Asylum, where several deaths from the disease had occurred. They also had the opportunity of studying a severe epidemic of dysentery which broke out in the Spring-side Home, New Haven, Conn. In this epidemic more than fifty cases occurred within three weeks among the 350 inmates, all being of a very acute form, with fatalities. They obtained the bacillus in fourteen of these cases which they studied. Three of these were insane patients.

They describe in detail the cultural characteristics, morphology, and agglutination reactions of the organism. The agglutination tests were made with the organisms obtained from Shiga, Flexner, Kruse, and Strong and with the organisms which they isolated from their own patients. The tests were made with the blood of the patients they observed, and also with a sample of antidyenteric serum sent by Shiga to Flexner. Briefly stated, the agglutination reaction was practically identical with all the organisms.

As a result of their study of the cases in this country, they firmly believe that there is a specific bacillus, and that it is identical with the organism described by the observers mentioned. They have no doubt that the organism is pathogenic. It is interesting to note, however, that in no instance have characteristic intestinal lesions followed inoculation of animals with the organism. Flexner and Strong have both produced typical dysentery in man following the ingestion of bacillus dysenteriae.

It is important to note that the organism in this country has been found both in sporadic and epidemic cases ; also that it is apparently the cause of acute dysentery in the insane. It is also present in the cases of " terminal " dysentery in association with chronic Bright's disease.

They draw the following conclusions from their study :

1. The several standard cultures used in this study are indistinguishable—a conclusion previously reached and stated by Flexner.
2. The acute dysentery of the United States is due to a bacillus indistinguishable from that obtained from the epidemics of dysentery in other parts of the world.
3. The sporadic and the institutional outbreaks of acute dysentery are caused by some micro-organism, and this organism is identical with that causing epidemic acute dysentery.
4. The cause of acute dysentery, whether sporadic, institutional, or epidemic, is bacillus dysenteriae Shiga.

A Case of Uræmic Ulceration of the Stomach and Small Intestines.—

MATRIEU and ROUX (*Archives Générales de Médecine*, January, 1902, p. 14) remark on the infrequency of ulceration of the gastro-intestinal tract in uræmia. They report a most interesting case which came under their personal observation and which terminated fatally. The patient was a young woman, aged twenty-two years, who was admitted to the Andral Hospital, Paris, suffering from symptoms which, on admission, were thought to be due to advanced pulmonary tuberculosis. Later it was learned that she had been in various hospitals during the previous three years, suffering from chronic nephritis, and the urine on this admission was found to be loaded with albumin. The patient's main complaint was severe abdominal pain. This was most marked in the right iliac fossa. On palpation of the abdomen it was found that there was marked muscular rigidity. There was no distention of the abdomen, nor was there any nausea, vomiting, or hiccoughing. The patient failed rapidly, and died a few hours after admission. The autopsy showed marked chronic interstitial nephritis, with hypertrophy of the heart. The most interesting findings were in connection with the gastro-intestinal tract. Along the lesser curvature of the stomach there were six ulcerations of the mucous membrane. One was large, measuring 3 by 4 cm., and presented all the features of an ordinary round ulcer, and was much deeper than the others. The remaining five were much smaller and more superficial.

There was quite an extensive ulceration of the smaller intestine, reaching from the point 4 to 5 cm. from the ileo-cæcal valve for a distance of 75 cm. The ulceration was situated along the free border of the intestine. Over this area there was no marked loss of substance, but its surface was covered with a yellowish diphtheric-like exudate. This long ulcerated area seemed to have been formed from a series of separate ulcers, measuring from 15 to 20 mm. by 2 to 6 cm. There were no ulcers found in the large intestine.

The authors in a search of the literature have collected 22 cases in which there have been ulcerations in some part of the gastro-intestinal tract in connection with advanced cases of Bright's disease. The majority of the cases have been in patients from eighteen to twenty-five years of age. In 20 of the 22 observations definite reference to the age is made. In 13 of these the ages range between fourteen and twenty-four years. In 10 of these 13 cases the ulceration was in the ileum, near the cæcum; in 1 the stomach was involved; and in 2 the large intestine showed more or less extensive ulceration.

The ages of the 7 other patients ranged between thirty-two and forty-seven years. Of these 3 had hemorrhagic erosions of the stomach, and in 4 the large intestine showed less numerous ulcerations. In none of these 7 cases was the small intestine involved. The ulceration of the small intestine appears then to be more frequent in young individuals.

In practically all the cases the ulceration was not suspected during life. All the abdominal symptoms which have been present in these cases have also occurred in nephritic cases without intestinal ulceration. In the majority of cases there has been severe diarrhœa, the stools being at times hemorrhagic. In a few instances constipation has occurred. Abdominal pain is not a constant symptom. It is worthy of note that in 2 cases, those

of Méry and Barbe, perforation had occurred, with resulting peritonitis. The writers consider the complication a most grave one from a prognostic stand-point, stating that nearly all the cases terminate fatally within from a few days to a few months. They believe, however, that recovery may occur after ulceration has once occurred, owing to the fact that in some of the cases reported many healed ulcers have been found. They were not able to establish definitely that the ulcerations were due to infarction of the bloodvessels supplying the areas involved. They seem inclined to the view that they were due to the result of toxic substances circulating in the blood. They state that the large gastric ulcer was practically indistinguishable, either by gross or microscopical examination, from the ordinary round ulcer of the stomach, and they consider it of interest to find that a typical round ulcer may occur in Bright's disease and be apparently due either directly or indirectly to this condition.

SURGERY.

UNDER THE CHARGE OF

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A Case of Excision of the Vesiculæ Seminales for Primary Tuberculous Disease.—HODGSON (*British Medical Journal*, November 16, 1901) reports the case of a man, aged thirty-two years, married, who presented himself for treatment for frequent and painful micturition of three-years' duration. On an average he micturated at half-hour intervals day and night, but just previous to his admission to the hospital this interval was increased to five minutes. The pain, which was cutting in character, always shot from his perineum to the end of the penis, always preceded micturition and increased in intensity as the desire to micturate increased. No family history of tuberculosis could be obtained, and the previous venereal history was also negative. Examination showed the prostate to be normal in size; but there was an irregular, nodular mass, firm to the touch and hardly the size of the little finger, which could be detected on the right side of the base of the bladder. This swelling commenced immediately above the prostate, and extended upward, backward, and slightly outward, but the prostate itself was normal in size. The urine was negative except for tubercle bacilli. Operation being decided upon, the patient was chloroformed and placed in the lithotomy position. A sound was introduced into the bladder, the sphincter ani stretched, and then a semilunar incision three inches long was made across the perineum one inch in front of the anus. The dissection was continued until the seminal vesicles

were exposed, ligatures were suitably placed and both vesicles were removed in their capsule. The wound was closed except for a gauze drain. The patient made an uninterrupted recovery.

The Surgical Treatment of Sciatica.—HALLEY (*The Scottish Medical and Surgical Journal*, February, 1902) reports five cases of sciatica treated by stretching, which were relieved of pain almost immediately after the operation. Relief from pain is known to have continued for four months in two cases and thirteen months in one. In one of these it had existed for eight years before the operation. In one case, where no subsequent history has been obtained, relief followed the operation, although sciatica had existed for the previous five years. The sheath should be opened only if there is much effusion, otherwise simple stretching is sufficient. The author believes that from the relief after nerve-stretching, which results in even long-standing cases, this treatment ought to be adopted sooner. Many of these chronic cases treated with numerous drugs and external applications would benefit more from prolonged rest, but many cannot carry this out. Provided the cases are limited and well defined, having no other pains and no other organic condition in pelvis or elsewhere, nerve-stretching is beneficial, and should be recommended early. Should the pain recur, after operation, in the limb below the knee the author recommends further nerve-stretching lower down.

Intubation.—KUHNS (*Centralblatt f. Chir.*, December 28, 1901) states that the instrument used by him for this purpose is most simple, being a metal cylindrical pipe or tube. In order to introduce this tube into the larynx and trachea a guiding tube is first introduced. This should be bent in a half circular form, like a metal catheter. In order to secure the tube outside of the mouth after its introduction, the author recommends the application of a diagonal piece of metal like the shield of a tracheotomy canula, which can be maintained in position at the corner of the mouth by a rubber band which encircles the head. To this band should be attached a thick piece of rubber which should be placed between the teeth, and so prevent the teeth from coming in contact with the tube under all circumstances. The indications for intubation are many—in the removal of tumors of the throat, or during any operation under anæsthesia there may arise a necessity demanding its use. The technique of intubation is so simple that every surgeon may attempt it without fear. In intubation of a grown person it is sufficient to draw up the epiglottis quickly, then to grasp or press the underside of the epiglottis with the left forefinger, and then allow the tube to slide along the under side into the larynx. Gentle pressure or a slight rocking motion of the tube from side to side will materially help in the introduction. As the tube is introduced the guiding tube should be gently removed. Unless the patient be fully anæsthetized some cough is caused by the irritation of the foreign body, or sometimes a sense of suffocation as the result of an excess of mucus lodging in the trachea. Cocaine or antipyrine will relieve the irritation, and the excessive mucus may be left to take care of itself. The largest tube that can be successfully introduced is the best. An important point that should not be neglected is to keep the tube thoroughly free from mucus by wiping it out with a small swab or brush. The chief indications for intubation are:

1. Compression of the trachea from any cause; and it is to be preferred to tracheotomy in operations on goitres, for the reason that the anæsthesia may go on in its natural way, the tube may be removed immediately after the operation; and, most important, there is no danger of infection of the wound as there is when tracheotomy is performed. 2. There is no danger of blood flowing into the air-passages, as may happen in tracheotomy. 3. In case of asphyxia the air may be forced through the tube by a bellows, and, as a rule, this will quickly be followed by relief.

Practical Observations on Appendicitis.—LLOYD, (*Birmingham Medical Review*, December, 1901) states that he should like to see a closer and more constant association of the words "peritonitis" and "cellulitis" with that of "appendicitis," so that the public might in time learn that it is the peritonitis or the cellulitis which constitutes the serious elements in this disease, and not appendicitis itself, and that the one is likely to follow on the other at any time and with every degree of severity; and they would then better interpret the attitude of the profession toward the disorder, certainly as regards the employment of medicines on the one hand and of operations on the other. Inflammation within the appendix is not necessarily attended by symptoms of any kind so long as the products of the inflammatory process are able to get out of it, pain being induced only when more or less obstruction exists to the easy escape of these products into the lumen of the bowel, and in this factor of obstruction chiefly lies the key to the whole subject of appendicitis. What the appendiceal obstructions are, how they originate, and how they give rise to trouble, may be classified under the heads of strictures, kinks, occlusions, and foreign bodies—under which term is included fecal concretions. Strictures may occur in any part of the appendix, of every degree of tightness, and may be either congenital or acquired. The congenital are by far the more common. Acquired strictures may be either simple or specific—the former the result of chronic inflammation, and the latter of ulceration of the mucous lining and subsequent cicatrization.

The etiology of the simple form of acquired stricture here is precisely that of the better known but less common stricture of the urethra, except that it is not due to gonorrheal inflammation. It results from a chronic inflammatory process affecting the mucous membrane, extending gradually into the submucosa, and generating there a fibrosis of the connective tissues; it is contractile and constant. This form of inflammation within the appendix occurs usually by extension from the lining of the great intestine. The strictures due to specific causes follow on ulceration, and the types of ulcer met with in the appendix are similar to those which affect the intestine in its neighborhood, viz.: typhoid, dysenteric, tubercular, syphilitic, and carcinomatous.

Kinks or twists of the appendix are usually congenital and obstruct by the "chokage" which results in every soft-walled tube when it is actually bent or twisted upon itself in any direction.

Occlusions of the appendix are where the canal of the appendix is organically obliterated in a part only of its length, so as to leave on the distal side of the obliteration a closed cavity lined by mucous membrane, more or less normal. Inflammatory effusions within such cavities are practically abscesses

ab initio, as they cannot empty themselves along the appendix into the cæcum; their contents can only escape by bursting into the peritoneal cavity the intestines, or the adjacent cellular tissue.

Foreign bodies, the majority of which are fecal concretions, may both irritate and obstruct. They become obstructing agents when they endeavor to escape along the proximal part of the canal toward the cæcum. They may block up the outlet of the organ just as the gallstone does the cystic duct, or a renal calculus the ureter, and the cavity of the appendix on their distal side then becomes distended by its own products. The consequences of this increasing distention constitute the real dangers of "appendicitis," and vary in their kind according to the distensibility and position of the appendix and the character of the inflammation by which it is affected. The signs and symptoms of appendicitis are many and varied, but the author lays particular stress upon pain and inflammatory swelling. The pains of appendicitis are due to two distinct conditions—distention of the appendix and inflammation of the peritoneum or cellular tissue. The first always precedes the others, and differs from them in character and in locality. They are, however, quickly merged together in the ordinary type of case, and it requires, therefore, careful and early investigation to discriminate between them. Appendicular pain is not always severe at its first onset, and is not often referred to the appendix region. It is frequently felt on waking in the morning, and is commonly epigastric in position. Usually within twelve hours, however, it extends to the iliac region. Continuous severe aching pain during the early days of the attack is of grave moment, and is typically seen in the acute gangrenous form of the disorder. It is due to obstruction of the appendix, is temporarily diminished when the organ gives way, and is shortly followed by the more paroxysmal pains of peritonitis.

Heat, tenderness, hardness, and swelling are essential features of every inflammation, and must be present, therefore, in all cases of appendicitis. They may not always be appreciable by physical examination, but they are often overlooked when they are quite discoverable; and inasmuch as the "presence of a tumor" is the chief diagnostic sign of the affection we are discussing, it is necessary that we should give great attention to this feature in our examination. Although the base of the appendix is fairly constant in its anatomical position, the tip of the organ may swing in any direction, and the tumor, therefore, which attends or results from its inflammation may develop at any part within reach of the appendix; for working purposes we may describe them as iliac, pelvic, lumbar, abdominal, and retrocecal.

Fortunately for our patients, iliac swellings are the most common and are easily palpable in the right iliac fossa, and their diagnosis is simple enough; but when the iliac tumor is absent diagnosis is a less easy matter; careful examination, however, will nearly always reveal the presence of swelling in one or other of the positions already named.

The pelvic variety lies in Douglas' pouch and can be felt by examination from below, either per rectum or vaginam, when absolutely nothing is to be discovered by palpation through the abdominal wall. Rectal or vaginal examination should be a routine procedure in every case of inflammatory abdominal disease.

The lumbar variety lies deep in the lumbar region, sometimes extraperi-

toneally in the connective tissue behind the bowel and below the kidney, and sometimes intraperitoneally outside the ascending colon. It is easily overlooked, particularly in well-developed or stout people; a distinct tumor cannot always be made out, but a comparative examination with the region of the opposite side readily reveals the presence of "fulness."

The abdominal variety may be situated in the epigastric, hypogastric, or right hypochondriac regions, often in the neighborhood of the umbilicus, either above, below, or to either side of that structure. Careful comparative palpation cannot fail to disclose such swellings.

The retrocaecal variety is next in frequency to the iliac, and lies at the back of the cæcum, at the level of the pelvic brim and opposite to the sacro-iliac joint. It can be felt only on deep backward palpation rather as a fulness than a distinctly localized tumor. It is usually resonant on percussion for the reason that the head of the cæcum lies in front of it, and is often mistaken for feces in the large bowel. A finger passed high up in the rectum and to the right sometimes appreciates the lower limit of the inflammatory area.

There is one type of appendicitis in which "tumor" is conspicuously absent, viz.: the acute gangrenous or perforative variety, with widespread peritonitis, where everything is lost in the general abdominal distention; although in these cases a tumor formed by the distended appendix can usually be felt before rupture has taken place.

On the question of treatment there is still something to be learned. At the commencement of an attack it is necessary to clear out the bowel; calomel and castor oil are the best remedies, followed by salines and belladonna. The question of operation should be considered from the very outset of the attack, because if it is to be of service in some of the varieties of the affection it must be performed within the first thirty-six hours. In those cases in which the indications for operation are in doubt operation is the safest alternative. Operation having been decided upon, the incision should be made over the tumor except in the pelvic variety, where no tumor is palpable, but there is fluctuation in Douglas' pouch, when the incision should be made through the posterior vaginal fornix in women, and through the rectum in children and men.

For the drainage of appendiceal suppurations there is nothing better than the rubber tube surrounded by a packing of iodoform gauze. The tube may be withdrawn early, and the gauze comes away usually within a week, and may be renewed if necessary. A narrow single strip carried lightly into the depths of the wound is all that is required—there is no need for the painful stuffing so often seen.

Great care should be given to the suture of the parietal wound, even where drainage is arranged for. This can be best done by interrupted fishgut sutures passed carefully through all the layers of the abdominal wall. This thoroughness can only be secured by seeing that each separate layer in the sides of the wound is systematically transfixed by the needle.

The appendix should always be removed if it comes readily to hand, but it is safer to leave it if there is likely to be much separation of adhesions in getting it away. When it is left behind, a few days after operation it occasionally happens that feces appear in the discharge from the wound, and anxiety as to the soundness of the cæcal wall is often felt, but usually the

feces come through the open stump of the appendix, and no harm follows, the majority healing soundly in the end.

Operation especially done for the removal of a diseased appendix during an interval between attacks has become a well-established procedure, and its results are extremely satisfactory; as a rule, it is best done through the oblique iliac incision. The after-treatment of appendiceal operations is that common to the general run of abdominal sections.

A Table for Operations upon the Stomach and Liver.—KELLING (*Centralblatt f. Chir.*, October 19, 1901) states that every surgeon has often experienced difficulty in operations upon the upper portion of the stomach and liver for the reason that this region is so hidden by the arch of the ribs as to be almost inaccessible. Lannelongue has proposed his rib-resection method for cases of this character, but this method is inadvisable, as it means an added risk to the patient, and does not always produce the desired result. For this reason it is of material assistance to devise a posture which would render the diaphragmatic region accessible to the eye as well as to the hand. The posture devised by the author imitates that which anatomists use when they wish to prepare the muscles of the diaphragm. They lay the body with the trunk horizontal, but bent to nearly a right angle in the lumbar region, so that the pelvis and legs hang downward and the feet touch the floor. The portion of the table on which the trunk rests is movable. On the upper surface of this portion two buckles are placed through which a strap is fastened to hold the body in place. In order to obtain better access to the subphrenic region the usual median incision in the linea alba should be supplemented by an oblique incision starting at the apex of the twelfth rib and extending diagonally toward the median line. In order to keep the intestines in the abdomen a towel is placed over them below the stomach and its margins fixed with clamp-forceps. The central portion should be pressed down on the viscera by an assistant's hand, while with the other hand he pulls the ribs upward. When working in the subphrenic space the operation may often be facilitated by dividing the suspensory ligament of the liver.

Quinine as a Styptic and Antiseptic.—MARX (*Centralblatt f. Chir.*, 1901, No. 45) states that as the result of his experiments he recommends quinine as a styptic and antiseptic. Everybody knows quinine as a specific protoplasm poison. The author states that space does not permit him to describe the experiments which proved the antiseptic powers of this drug. The styptic powers of quinine arise from the ability of its salts to agglutinate the red blood-corpuscles. For practical use, the best preparation is a 1 or 2 per cent. solution of quinine hydrochloricum, which should be applied to the bleeding surface with a gauze compress or tampon. It is needless to state that its use should be confined to cases of parenchymatous bleeding. Careful observation has failed to show any injury to the tissues; in fact, no reaction of any kind was observed. The disinfecting power of quinine on infected wounds is but slight, but in aseptic wounds it absolutely stops parenchymatous bleeding and has the additional power of rendering innocuous any germs that may have been conveyed into the wound by the operator's hands.

THERAPEUTICS.

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Contribution to the Knowledge of Phloridzin Diabetes.—DR. O. LOEWI has been experimenting to establish the fact, already determined by von Mering, that subcutaneous injections of phloridzin are followed by a greater excretion of sugar than when the drug is taken by the mouth. In the repeated experiments he found, as the cause of this peculiarity, (1) that the absorption of the phloridzin in the intestine is not a complete one, but that a decomposition takes place there, whereby a product is formed, difficult of absorption, yet capable of inducing diabetes if brought directly into the blood. The proof of this lies in the fact that if an extract of the feces of a dog fed with phloridzin be injected into another animal, glycosuria will follow; (2) in order to insure the highest degree of glycosuria, a certain amount of phloridzin is required, and, no matter how much this is increased under like conditions, no more sugar will be voided. If, however, the amount of food capable of producing sugar be increased, the amount of phloridzin must be larger to insure a maximum excretion; and (3) changes in the temperature of the animal are of no appreciable effect upon the glycosuria.—*Arch. für exp. Path. u. Pharmac.*, 1902, Band xlvii., p. 48.

Action of Digitalis and Its Allies upon the Vessels.—Drs. R. GOTTLIEB and R. MAGNUS have been repeating the experiments of F. Pick, which had been considered to prove conclusively a direct constricting action of digitalis and similar drugs upon the bloodvessels. This investigator did not give any evidence of the part played by the different vessels in this process, nor upon the time the vasomotor effect takes place and its duration. The new experiments were conducted on dogs. Two entirely different methods were employed: in one the amount of blood flowing out of the veins of different regions was registered after a sufficient amount of atropine had been given to overcome the slowing of the pulse; in the other the plethysmograph was used. The experiments showed that the increased blood pressure was due to increased heart action and contraction of the vessels, and that the latter is due to peripheral action which, in case of digitoxin, is general. In the case of the other examined glycosides (digitalin, convallamarin, strophanthin), the action is restricted to the splanchnic area. There is, however, some active constriction going on here in the peripheral vessels, yet this is overcome by a passive dilatation owing to reflux of blood from the intestines and an active reflex dilatation set up by the splanchnic

contraction. The general narrowing of the pathway of the blood seen with digitoxin gives a high resistance, which must be overcome by the heart; strophanthin, etc., open the vessels of the periphery, and this materially relieves this organ.—*Archiv für exp. Pathologie u. Pharmacologie*, 1902, Band xlvii., p. 135.

The Influence of Carbon Dioxide Inhalation upon the Body Temperature.—DR. N. VON WESTENREYK, to determine what effect the inhalation of small amounts of carbon dioxide would have upon the body temperature, employed rabbits with normal temperature and such in which an artificial fever had been induced by punctures of the heat centres. The animals were placed in an air-tight chamber in which some of the air was substituted for oxygen, and the experiments were conducted with 3 per cent. of volume of carbon dioxide gas. General symptoms did not appear except a slight acceleration and deepening of the respiratory movements at first, and a remarkable quietness of the animals. Within the first thirty to forty minutes the temperature dropped 0.4° C. to 1° C. and then again slowly reached the normal, even if they were brought back into the box. In the animals with fever the same observations were made, except that the reduction was 1° C. to 2° C.—*Archiv für exp. Pathologie u. Pharmacologie*, 1902, Band xlvii., p. 82.

Active Principle of Guaiacum.—DR. E. SCHAEER has found that the antisiphilitic, diaphoretic, antiarthritic, and emetic properties of guaiac depend solely upon the saponin, which is most abundant in the bark, less so in the outer part of the wood, still less in the medulla, and least of all, yet in appreciable amounts, in the resin. The fact that the decoction of the wood contains the most saponin of all galenical preparations, and has been found to be the most active preparation for centuries, seems to support this theory, as do also the recent observations that other ingredients, such as guaiaconic acid, have no effect in syphilis. The author recommends that the saponin of the bark and wood be isolated in pure form, that they be compared with each other and other known forms of saponin, and that they be tried pure in the various affections for which guaiac is known to be valuable.—*Archiv für exp. Pathologie u. Pharmacologie*, 1902, Band xlvii., p. 127.

Arsenic and Beriberi.—DR. RONALD ROSS reports on the analysis of twenty samples of hair taken from Chinese patients afflicted by beriberi. Since it has been suggested that this type of neuritis might have something in common with arsenical neuritis, the results were suggestive at least. These analyses gave indefinite results as far as a causal nexus being established. In six positive evidence was given; yet this is of importance, especially since the recent cases alone were those that gave positive results; the results from patients with long-standing disease were negative. The exact site from which the hair was obtained was not recorded. As this is a matter of importance, the suggestion is offered for future study that samples should be taken from near the scalp.—*British Medical Journal*, 1902, No. 2145, p. 329.

The Effects and After-effects of Ethyl and Ethylene Bromide.—DR. D. SCHERBATSCHOFF, after conducting a number of experiments, finds that there seems to be considerable difference between the physiological action of the ethyl bromide and the corresponding ethylene salt; for while the former seems to be relatively safe as an anæsthetic, the latter manifests many dangerous properties if used by inhalation. Indeed, many of the accidents reported are to be ascribed to the use of the wrong drug, owing to the great similarity of the names. To decide this point definitely, however, a number of experiments with frogs, white mice, and dogs were made. The frogs and mice were placed under a bell-jar, the dogs in an air-tight glass case, and a definite amount of ethylene bromide was allowed to evaporate. A real narcosis was induced only in frogs; and in all animals evil after-effects resulting in death were noted. The respirations were much increased and rendered shallow; there was a continuous sinking of the blood-pressure, and the cornea became opaque. Periods of excitation, convulsions, and vomiting also developed. The cause of death must be looked for in specific changes in respiration and circulation; the possibility that hydrogen bromide was formed and the blood thus acidified must be excluded, since in all cases it reacted alkaline. Dissection showed irritation of the bronchi, hyperæmia, and sometimes hepatization of the lungs and of the liver. Similar experiments conducted with ethyl bromide gave a pure narcosis which ended in death if continued long enough, but never did any ill-effects accompany the anæsthesia.—*Archiv für exp. Pathologie u. Pharmacologie*, 1902, Band xlvii., p. 1.

Upon the Toxicity of Dimethyl Sulphate.—DR. S. WEBER has thoroughly investigated the toxicology of dimethyl sulphate on animals. The use of dimethyl sulphate, a colorless, oily fluid of a specific gravity of 1.33 and a boiling-point of 370.4° F., has recently become an extended one in chemical factories. These studies were taken up because of the fact that three cases of poisoning have been reported. Two of these ended fatally under marked local and pulmonary symptoms. A twofold action was found: one local, strongly caustic, and one general, upon absorption of the poison. The caustic effects are seen most intensely upon the respiratory organs and conjunctivæ, especially if the substance is heated so that free vaporization takes place; while if applied to the skin, or if given subcutaneously, hyperæmia and necrosis, and if given *per os*, general gastrointestinal disturbance and perforation result. The general symptoms consisted of nystagmus and convulsions, increasing in frequency and intensity, then again diminishing, and ending in coma and paralysis, with accelerated respiration and unaltered heart action. The lethal dose was 5 centigrammes per kilo body weight in rabbits if injected, and death generally set in after several hours. The action of dimethyl sulphate does not depend upon its decomposition products, sulphuric acid and methyl alcohol, but upon a specific action inherent to the body itself. The caustic properties especially are peculiar to this compound, and are not found in the other ethers and esters of the fatty series, which are also incapable of setting up convulsions.—*Archiv für exp. Pathologie u. Pharmacologie*, 1902, Band xlvii. p. 113.

GYNECOLOGY.

UNDER THE CHARGE OF

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Pigmentation of the Abdomen.—LEHMAN (*Revue prat. d'Obstétrique et de Pédiatrie*, September, 1901) calls attention to the common error that the pigmentation in the median line of the abdomen is supposed to be found only in pregnant women. He shows that it may be observed in both sexes and at any age. Even in young children it may appear in connection with inflammation of the intestines, typhoid fever, and tuberculosis, while it is often marked in healthy girls at the time of puberty. The discoloration in the latter is probably due to the general congestion of the pelvic organs, and may be regarded as the forerunner of the first menstrual flow. If menstruation is regular and normal, the dark line disappears; if irregular and painful, it is apt to persist.

Among 122 young girls examined by the writer the line was present in 66. Of these 26 suffered from amenorrhœa, 20 from dysmenorrhœa, and 14 from menorrhagia; 6 had obstinate constipation and abdominal pains. The other 56 patients menstruated regularly without pain, and had no intestinal disturbances.

After the climacteric the line of pigmentation is barely visible.

Genital Atresia.—LABUSQUIÈRE (*Annales de Gynécologie et d'Obstétrique*, No. 8, 1901) believes that the majority of atresis are acquired, and hence that it may be possible to prevent them. He advises that not only should a routine examination of the external genitals of the newborn be made, especially where the mother has a gonorrhœal discharge, but that prophylactic treatment should be adopted in the shape of antiseptic lotions. The same solution of nitrate of silver which is instilled into the eyes may be used with advantage.

So-called precocious menstruation is always to be regarded with suspicion as a possible evidence of gonorrhœal infection, and an examination should be made to ascertain if the latter is present. Cases of persistent leucorrhœa in little girls may result in atresia, so that it is advisable to prove its absence by the careful introduction of a sound.

Prolonged absence of the catamenial flow in girls at the age of puberty may justify an examination, especially if there is a history of a previous infection. The presence of menstrual molimina, or even vicarious hemorrhage, does not contraindicate such an investigation.

Referring to the development of hæmatosalpinx in connection with genital atresia, the writer affirms that when the enlargement of the tube is due to

simple retention of blood and is not of inflammatory origin, as shown by the absence of any history of acute infection, of constitutional disturbance, or of persistent abdominal pains, primary laparotomy is not justifiable. It is better to first evacuate the retained blood per vaginam if possible, abdominal section being performed later if necessary.

Infection of Abdominal Wound during Operation.—PEISER (*Monatschrift für Geb. u. Gyn.*, Band xvi., Heft 2) states that even in cases of benign adenocystoma of the ovary cells from the fluid may be transplanted to the abdominal wound or to the site of a trocar puncture, from which growths develop similar in character to the parent tumor. These may grow internally into the intestine and externally through the skin, and these secondary growths may become cancerous. The writer infers from this fact not only that explorative puncture or tapping of ovarian cysts is a dangerous procedure, but that during operation the edges of the wound should be carefully protected before the contents of the cyst are evacuated, especially if thick and gelatinous.

SCHAEFFER (*Zeitschrift für Geb. u. Gyn.*, Band xlv., Heft 3) reports a case of ovariectomy in which the tumor was found to be an adenocarcinoma. A recurrence occurred which was confined entirely to the cicatrix, the pelvic and abdominal cavities being healthy. He infers that the wound was inoculated at the time of operation.

Fixation of the Prolapsed Uterus.—BUCURA (*Zeitschrift für Geb. u. Gyn.*, Band xlv., Heft 3) reports sixteen cases from Wertheim's clinic with only one failure. Briefly, the operation practised by the latter consists in denuding an oval surface on the anterior vaginal wall, extending as low as the meatus urinarius. The fundus uteri is drawn down through an opening in the anterior vaginal fornix and is sutured to the surface in question; then the pelvic floor is repaired.

The advantages claimed are prevention of a recurrence of the cystocele, which is the cause of failure in most operations for prolapsus. Menstruation persists and there is a free discharge of the uterine secretions. Marital relations are not interfered with. However, it is important that conception should not occur, which in younger women can be secured by resecting the tubes. In general this plastic operation is preferable to hysterectomy in intractable cases.

Conservative Treatment of Fibromyomata.—OLSHAUSEN (*Zeitschrift für Geb. und Gynäkologie*, Bl. lxiii., Heft 1) pleads for the more general adoption of myomectomy instead of supravaginal amputation of the fibroid uterus, especially in women under forty. He does not hesitate to leave small nodules, which may never give any future trouble. While the operation of enucleation in former years was a dangerous one, now it is quite safe, especially if the bed of the tumor is carefully sutured with catgut.

In 207 myoma operations he had 29 enucleations without a death. As regards the number of cases of fibroid in which interference is called for, the writer notes that in 200 cases in private practice he found it necessary to operate in only 33. During the three years from 1897 to 1900 320 private cases were seen, in only 53 of which was an operation indicated. He believes

that probably 18 per cent. really require operation, which is not yet entirely free from unavoidable dangers, especially embolus and intestinal obstruction.

Ultimate Results of Operations for Uterine Fibroids.—BURCKHARD (*Zeitschrift für Geb. und Gynäkologie*, Bd. xliii., Heft 1) finds, from an analysis of the statistics of a number of operators, that with few exceptions the results of myoma operations are quite satisfactory. After castration hemorrhage usually ceases if all the ovarian tissue has been removed; the tumor ceases to grow and usually diminishes in size. Menstrual molimina rarely persist after the removal of both ovaries, but may do so if one ovary is left, even if the uterus is removed. When the entire uterus is removed, with or without the adnexa, disturbances are less frequent than after castration and supravaginal amputation; if the ovaries are left they are still more rare. The writer has never observed profound psychical disturbances after the removal of both ovaries.

Since no injury to the health results from the presence of the ovaries after hysterectomy, and climacteric phenomena are less marked, they should always be preserved when they are healthy.

The Early Diagnosis of Uterine Cancer.—ABEL (*Archiv für Gynäkologie*, Bd. lxiv., Heft 2) calls attention to a peculiar arrangement of the elastic fibres in and around malignant foci which, he believes, constitutes an important diagnostic point in doubtful cases. Under a low power of the microscope these fibres appear to surround the cancerous alveoli, but under higher powers they are seen crossing in all directions between the cells, where they present a peculiar broken appearance, as if they were actually destroyed by the disease, instead of being merely pushed aside. These broken fibres are never seen in any other neoplasm of the uterus, such as condyloma or tuberculosis, nor in hyperplasia of the connective tissue, hence they serve to distinguish simple atypical epithelial growths from true carcinoma.

Ligation of the Ureters.—FRAENKEL (*Archiv für Gynäkologie*, Bd. lxiv., Heft 2), from experiments on rabbits, arrives at the conclusion that ligation of the upper end of a ureter after section of the duct does not always prevent the escape of urine from the kidney, as the ligature is apt to slip. Since in exceptional cases it may not be possible or justifiable to perform anastomosis of the ureter, it is important to be able to ligate it in such a way as to prevent slipping of the ligature. The writer advises that after ligation the upper end be sutured in the abdominal wound. If the urine escapes, the fistula can be cured by a subsequent operation; if the ligature holds and hydronephrosis of the corresponding kidney occurs, the patient still has a good chance of recovery, as shown by several reported cases. If the opposite kidney is diseased or uræmic symptoms appear the ligature can at once be divided.

Hæmatocœle Due to Hemorrhage from the Ovary.—GABRIEL (*Archiv für Gynäkologie*, Bd. lxiv., Heft 2), in reporting a case of hæmatocœle due to follicular hemorrhage in which the diagnosis of ruptured extra-uterine pregnancy had been made, reviews the literature of the subject. He finds

no satisfactory statistics with regard to the etiology of hæmatocele, ectopic gestation being regarded as furnishing about 28 per cent. of the cases. Authentic cases of ovarian hemorrhage in which the diagnosis was confirmed by operation are comparatively few in number, but none the less the condition is well recognized.

Hysterectomy for Cancer of the Uterus.—JORDAN (*Zeitschrift für Geb. und Gynäkologie*, Bd. xlv., Heft 21), after reviewing carefully the statistics of abdominal hysterectomy, arrives at the following conclusions: Vaginal hysterectomy is the natural method of removing cancer of the uterus when the disease is confined to that organ. Since the mortality by the abdominal route is three to four times that by the vaginal, and the operation is much more difficult, the indications for vaginal hysterectomy should be extended. The abdominal operation should be performed only when it is impossible to remove the uterus per vaginam, as in cases in which carcinoma is complicated by fibroid or ovarian tumors. If the pelvic connective tissue is so infiltrated that radical vaginal extirpation cannot be performed, there is no object in resorting to abdominal.

Vesical and Ureteral Fistulæ following Vaginal Hysterectomy.—HENKEL (*Zeitschrift für Geb. und Gynäkologie*, Bd. xlv., Heft 2) finds that the majority of injuries to the urinary tract occur during operations for cancer of the cervix uteri, rather than of the portio. As shown by Mackenrodt's statistics, the bladder and ureter are especially liable to injury during igni-extirpation, but the clamps have been responsible for many cases.

In 263 hysterectomies at the Berlin clinic previous to 1897 the mortality was 5.13 per cent., while the bladder was injured seven times, and one ureter was injured or tied in five cases. After the latter year, when the indications for the operation were extended to include cases in which the broad ligaments were infiltrated, in 375 cases (with a mortality of 5.06 per cent.) the bladder was injured fourteen times and the ureter nine times.

The writer finds that these accidents (especially to the ureter) have increased in number, especially when clamps are used. The prognosis in wounds of the bladder is good, about 68 per cent. healing when sutured at the time.

Hot Air in the Treatment of Pelvic Exudates.—KEHREER (*Centralblatt für Gynäkologie*, 1901, No. 52), in view of the fact that at least 95 per cent. of all pelvic exudates tend to become absorbed, emphasizes the importance of assisting natural processes by the use of such agents as reduce local hyperæmia. He uses hot vaginal douches several times daily, allowing at least three gallons of water to flow through a double-current glass tube, as well as hot sitz-baths and compresses applied to the lower abdomen. He speaks highly of the apparatus devised by Polano, in which continuous heating is effected by electricity.

THOMSON (*Ibid.*) commends the method of treatment introduced by Polano, with which he has obtained striking results in cases of chronic perimetritis. Not only is pain promptly relieved, but exudates are rapidly absorbed. The only contra-indications are advanced cardiac lesions or disease of the arteries.

OBSTETRICS.

UNDER THE CHARGE OF

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Hydroa During Pregnancy Caused by Staphylococcus Albus.—In the *British Medical Journal*, January 11, 1902, HOLMES and BULLOCK report the case of a patient in her third confinement who, on the day following the birth of the child, complained of severe itching in the palms of the hands and the soles of the feet. The tissues were red, and a thick confluent rash, the size of peas, was present. A similar eruption was seen upon the thighs, chest, and back. The patient had no fever.

On inquiry it was learned that in her other confinements the patient had suffered in a similar way. An examination of the fluid from one of the recent spots of eruption was made, and a pure culture of the staphylococcus albus was obtained.

The patient improved daily, and the child, which continued to nurse, was entirely free from the eruption. The treatment consisted in giving saline mixture, with small doses of arsenic, internally. Later tablets of sulphur were used.

Pregnancy and Parturition in a Case Complicated by Advanced Tabes Dorsalis.—At the meeting of the Gynecological Society of Munich, MIRA-BEAU (*Centralblatt für Gynäkologie*, 1902, No. 5) reported the case of a patient in advanced tabes dorsalis, who passed through pregnancy and labor.

In books upon diseases of the nervous system various statements are made concerning the influence of this disease upon the pelvic organs. The statement is made by Leyden and Goldscheider in Nothnagel's *Handbook of Internal Medicine*, that menstruation proceeds regularly in these cases and that pregnancy and labor may also go on normally. As the disease advances the statement is made that the functions of the pelvic organs are also disturbed. Books upon obstetrics contain little information upon the subject. Litschkus (*Wratsh*, 1885) reports the case of a patient, aged twenty-six years, who was confined nine years previously, and who for seven years had manifested symptoms of tabes dorsalis. Labor lasted five days, but terminated spontaneously and the patient made a good recovery. Mirabeau's case was aged thirty-four years, who had been under observation for three years. The patient's history showed that after the seventeenth year her menstruation became regular, and that soon after she was treated in a hospital for syphilis. After thorough treatment by inunction she made a good recovery. Five years afterward she married, but had no children. Nine years after her infection and four years after her marriage she was found to have tabes

dorsalis. Mirabeau was called to the case because the patient had severe hemorrhage from the genital tract. On examination the patient seemed well nourished. She had ceased to menstruate for two periods and had been bleeding for five days. An abortion seemed threatened. This, however, ceased under rest in bed, warm douches and an ice-bag over the abdomen. The patient was found to have well-established tabes dorsalis. Both lower extremities were paralyzed, the patellar reflex destroyed, ataxia was well pronounced, and sensibility much diminished. The pupils were narrow, with absence of reflex. There was great difficulty with the functions of the bladder and rectum. The patient was immediately put upon a vigorous anti-syphilitic treatment and made considerable improvement as a result. As pregnancy drew to its close the probability of spontaneous labor did not seem great.

Contrary to expectation, labor proceeded without the slightest evidence of pain. The amniotic liquid escaped at the beginning of labor. The cervix was considerably dilated at this time. The expulsion of the child occurred sixty-five minutes after the rupture of the membranes, and but seven pains actually occurred, five of great length and two of short duration. The patient had not the slightest feeling from the uterine contractions. At the last the abdominal muscles contracted once, and the patient said that she felt as if the rectum was emptying itself easily of its contents. She had been without sensation in the rectum for several years. With the sixth pain the child, a well-developed girl, was born, and with the seventh pain the after-birth. The mother was not aware that she had given birth to a child until she heard its cry and saw the infant. The child did well and had a syphilitic eruption three weeks after birth, which subsided under the use of calomel. The patient made a good recovery from labor.

This extraordinary case illustrates remarkably the physiology of labor. It is a striking demonstration of the fact that the contractions of the uterus do not depend upon the ganglia of the spinal cord so much as upon the smaller ganglia upon the nerve trunks in the uterine structure. Evidently the transmission of sensation through the cord to the brain is a frequent source of delay in complicated labor. It is a familiar experience in dealing with patients that labor ceases through the reflex inhibition of suffering. When this is removed by disease or by an anæsthetic labor goes on regularly and independently.

Harelip.—In the *Archives of Pediatrics*, February, 1902, RACHFORD reports the following interesting and extraordinary case:

The parents in question have a family history of tuberculosis. Their first child, a girl, was born with a cleft of the upper lip and nostril and hard and soft palate. The second child, also a girl, had a similar deformity. The third child, a boy, was without deformity, but died subsequently of tuberculosis. The fourth child, a girl, was so terribly deformed that the child lived but a few hours. The fifth child was a boy, perfect in development, dying of tubercular meningitis shortly after he was two years old. The sixth pregnancy resulted in the birth of a girl with double harelip, afterward remedied by operation, the child dying of tuberculosis at eighteen months. The mother then became tuberculous, but improved on removing to another climate. Her

seventh child was a boy, perfect in development, and at the time of writing was thriving well.

A review of the case shows the fact that there is no family history of hare-lip or cleft palate. The mother has a highly arched palate. There is a very bad family history of tuberculosis. The four girls had cleft palate and hare-lip, and the three boys were entirely free from this deformity. The first and second boys died of tuberculosis, and the third is too young to have had much opportunity to acquire the disease.

Repeated Rupture of the Uterus.—In the *Monatschrift für Geburtshülfe und Gynäkologie*, 1902, Band xv., Heft 1, KRZYSKI reports the case of a patient, aged thirty-three, who had been pregnant five times, having borne three living children, one abortion, and on one occasion a deformed child. The pelvis was justo-minor with considerable flattening. The patient considered herself at full term. The membranes ruptured at the beginning of pains. Upon examination the heart sounds were not heard, the uterus admitted three fingers, and the child was presenting in the usual manner. The head had entered the pelvis, the pulse of the mother was somewhat rapid, and she seemed impatient and suffering considerably. There seemed to be no essential reason for terminating the parturition at that time. Some twelve hours afterward the abdomen was considerably enlarged and very sensitive. The patient was apathetic and had a sighing respiration. Contractions of the uterus were weak, but could be plainly discerned. The mouth of the womb was smaller than formerly, and the head considerably higher and above the promontory of the sacrum. The internal conjugate was between 10 and 11 cm. The patient was anesthetized and version performed. It was necessary to bring down both feet and to perforate the head to deliver the patient. Upon examining the uterus after delivery, it was found that a laceration of the tissues into the parametrium was present on the right side. The uterus contracted well, and there was no hemorrhage. The pulse was good; the patient roused speedily from the anæsthetic, and complained of pain in the abdomen, which was distending. The placenta came away soon after the birth of the child. The vagina was tamponed with sterile gauze, and the uterus was compressed by a pad and bandage. The gauze was removed after some time, when a rupture was found in the upper portion of the uterus which was well closed by uterine contractions. An exudate developed at the site of the tear, which gradually passed away. Twenty-four days after confinement the patient was discharged in good condition. She afterward reported for examination, when a hard, painful tumor was found three fingers above the pubic bone, while the uterus was hard, large, and somewhat sensitive. On the left side the uterine appendages were enlarged, and there was a remnant of parametritis, and on the right side evident traces of perimetritis and parametritis.

This patient returned fourteen months after her confinement, again in the pregnant condition. She was admitted to the hospital, and premature labor was brought on. During the labor elastic bags were employed to dilate the uterus, and strong labor pains were excited. It was determined to deliver the patient by Cæsarean section because of the danger of uterine rupture and the fact that labor did not proceed spontaneously. While she was being prepared for operation the heart sounds grew slower, and finally ceased, and

the patient's pains became much less and practically ceased. On opening the abdomen the breech and limbs of the child were found to have escaped from the uterus, while the head still remained within the cervix. The uterus had ruptured in its anterior wall near the site of the former rupture. The entire anterior wall was thin and dark in color through extravasated blood. The posterior wall was uninjured and of normal contour. The muscular tissue of the anterior portion of the cervix and lower uterine segment felt like ruptured elastic connective tissue. The uterus was so badly torn that it was impossible to recognize accurately the site of the former rupture. Hysterectomy was performed, and the stump drained by a strand of iodoform gauze passed into the vagina. The patient made a good recovery.

The opportunity was taken to examine the muscular tissue of the uterus microscopically to observe whether a degenerative process in the muscle was responsible for the rupture of the uterus. At the site of rupture the connective tissue of the uterus was infiltrated with blood, the capillaries much distended, the elastic fibres of the walls of the vessels distinctly demonstrable by staining, and individual muscle cells were found in a degenerative condition. There was no evidence of inflammation either recent or chronic, nor could any adequate explanation be found by microscopical examination for the repeated rupture and also for the location of the rupture.

The Formation of Decidua in the Fallopian Tube in Tubal and Intra-uterine Pregnancy.—**LANGÉ** (*Monatschrift für Geburtshilfe und Gynäkologie*, Band xv., Heft 1, 1902) considers this question at length and gives the results which he has obtained in the examination of specimens from twenty cases. He has observed that just as in the gravid uterus there is the forming of a decidual membrane, so in the gravid tube to a less extent and with variable formation a decidua can exist. It is usual for decidua to form in the womb in most cases of tubal pregnancy. He finds that cases are reported showing that in tubal pregnancy decidua is formed in the non-pregnant tube and also in that portion of the uterus which is most adjacent to the tube. He examined a second series of five cases, and concludes from this investigation that the mucous membrane of the Fallopian tube, like that which lines the uterus, has the property of forming a decidual membrane. For this to happen the ovum must not only be in contact with the mucous membrane, but the impregnated ovum must form its attachment to the tube or to the uterus. The capacity of the mucous membrane of the tube for forming decidual membrane is much less than that of the lining membrane of the uterus. In the early months of pregnancy the tube forms scarcely any decidual lining membrane. Except in cases where the attachment of the ovum excites irritation in the tube during the early months of pregnancy, it is not uncommon to find the merest trace of decidual formation.

Three Cases of Cæsarean Section for Eclampsia.—In the *Centralblatt für Gynäkologie*, No. 5, 1902, **LOWENSTEIN** reports three Cæsarean sections for eclampsia.

The first was a primipara, twenty-eight years old, who had suffered during pregnancy from swollen legs and cough. She had a slow labor terminating in five eclamptic convulsions. The head was above the pelvic brim, the pelvis

was normal, the external os not dilated, and the membranes unruptured. Cæsarean section was at once performed, followed by the birth of a living child. A wide-spread bronchitis and nephritis supervened. Pneumonia followed, and the patient perished from respiratory failure. Autopsy showed purulent necrosis of the uterine cavity, with beginning peritonitis around the uterine stitches. There was stitch abscess in the abdominal wall.

Case second was also a primipara at full term, the fœtus in second position. She had had violent convulsions. The external os permitted the entrance of one finger, and the cervix was not shortened. She had no labor pains, and the child was living. An effort had been made to control the convulsions by morphia and chloroform without success. Cæsarean section was practised by transverse incision. The incision came down upon one corner of the placenta at the fundus. The uterus contracted well without much hemorrhage. The child was asphyxiated, but revived. The mother had a few slight convulsions after the operation. Immediately after the operation the patient seemed to improve, but later convulsions returned, and the patient perished from œdema of the lungs.

Case third was seven months pregnant and a primipara. She was unconscious, without pupillary reaction, when admitted to the hospital. Convulsions supervened, five occurring in two hours. The urine showed an excessive quantity of albumin. As hot baths and other remedies failed to control the convulsions, the child was delivered by Cæsarean section. The patient perished within the next twenty-four hours from rapid œdema of the lungs.

Embolus following Labor.—VOIGT (*Centralblatt für Gynäkologie*, No. 2, 1902) reports a case of central placenta prævia in which the placenta was perforated by the fingers, combined version made, and a foot brought down. A partially asphyxiated child was delivered shortly afterward. The uterus contracted well.

Three-quarters of an hour later the patient had a severe embolic attack with marked dyspnoea and rapid, feeble pulse. She gradually recovered during the nineteen hours following, but suffered from dyspnoea and weak action of the heart. Four days after labor a second attack occurred, with the formation of an infarct in the lung and bloody sputum. On the tenth day after labor a third attack happened and the patient expectorated blood with elastic lung fibre. The patient finally made a recovery in more than a month's time. He also reported two other cases, one after craniotomy, the second after spontaneous labor, in a weak and debilitated patient. Both of these patients recovered.

Death from Pressure of a Pregnant Uterus upon a Horseshoe Kidney.—WALSH (*British Medical Journal*, January 18, 1902) describes an interesting case recently reported by the coroner of the city of London. A young married woman died suddenly upon the street. Upon autopsy she was found several months advanced in pregnancy, and the enlarged uterus pressing upon a horseshoe kidney situated low in the abdominal cavity. Pressure had practically disorganized the kidney, and the sudden death was due to a resulting uræmia.

OPHTHALMOLOGY.

 UNDER THE CHARGE OF

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Diagnosis and Treatment of Glaucoma.—TERRIEN (*Le Prog. Méd.*, No. 34, 1901) observes that the etiology and pathogenesis of glaucoma are not yet understood. A single symptom is dominant from the clinical stand-point—hypertonus. Cases occur of excavation of the papilla without increased tension; it is doubtful whether these should be regarded as glaucomatous. According as the increase in the tension is sudden in an eye previously healthy, or takes place slowly and progressively, permitting the organ to accustom itself to the change, the symptoms will be those of acute and chronic glaucoma respectively. Chronic glaucoma is simple, if the increase in tension is slight and unaccompanied by appreciable changes, or irritative in the opposite case. Each of these three varieties requires special treatment.

(a) *Acute glaucoma*—sudden steps with intervening remissions: It is preceded usually by prodromic attacks. Suddenly after fatigue, or a heavy meal, or without appreciable cause, the vision is disturbed; objects appear smoky, lights are surrounded by rainbows. If the eye is examined at the time of the attack it will present the following diagnostic marks: 1. The pupil is slightly larger than that of the other eye, and reacts but slowly to light. 2. The cornea is somewhat hazy at its centre. This is the main cause of the disturbance of the vision. 3. The tension is increased. The prodromic attack lasts but a short time, several minutes or hours. After a variable period—days, weeks, or months—a second and third attack recurs; finally the intervals between the attacks become of short duration until an outbreak of acute glaucoma proper. The latter may occur at once without prodromes; if carefully sought for, however, the latter are rarely absent. It is important not to confound a prodromic attack of glaucoma with ophthalmic migraine. The symptoms of acute glaucoma are those of the prodromes very greatly accentuated. There is ciliary injection, extremely violent pains are present in the eye and the periorbital region radiating to the occiput. The pupil is dilated, a point which differentiates it from iritis. The anterior chamber is shallow. The small black pupillary margin of ciliary pigment reflected upon the anterior face of the iris, hardly visible in a normal condition, is more apparent. The tension is usually very high. Hybrid cases of iritis complicated with hypertonus (glaucomatous iritis) occur, and in these the diagnosis is different and has an element of danger in the possible use of atropine—a true specific in iritis, but which may entail the loss of the eye in glaucoma. The ophthalmoscopic examination is negative. The fundus cannot be illuminated, and the vision is *nil* or nearly so.

After a variable time, one to two weeks, all the symptoms diminish or disappear; the vision returns, though somewhat affected, and a slight degree of dilatation of the pupil persists. Attacks recur more frequently with incomplete remissions. The pupil remains dilated and the tension raised. The disease tends to become chronic, to terminate finally in blindness.

(b) *Chronic Irritative Glaucoma.* The beginning of the affection resembles the prodromic attacks of acute glaucoma; it is marked by transient obscurations with slight hypertonus. But the attacks and the remissions are less frank, and in the intervals the tension remains somewhat above the normal; thus a certain degree of tolerance of the organ is established. Apart from slight ciliary congestion and slight dilatation of the pupil, the hypertonus manifests itself only by changes in the fundus (dilated veins, arterial pulse, excavation of the papilla). It leads quite rapidly to blindness.

(c) *Simple Chronic Glaucoma.* Insidiousness is the essential characteristic. The tension is but slightly increased, and does not manifest itself by any external symptom. It betrays itself solely by changes in the fundus which may cause total excavation of the papilla and complete blindness without the patient being aware thereof, if the second eye remains healthy.

The importance of recognizing these three forms of glaucoma depends upon the fact that the treatment is very different for each form. They are not always sharply marked. Intermediate varieties occur following fluctuations in the degree of tension.

Treatment. This is both medical and surgical. The medical treatment consists entirely in the use of myotics combined or not with antiphlogistic measures (leeches to the temples, warm compresses, purgatives, etc.). Eserine, first employed by Laqueur in 1877, may be used in aqueous, or better, oleaginous solutions. The latter is more active, and the alka'oid is not transformed into rubreserine as in the aqueous solution. The strength should never exceed 1 per cent., whatever be the vehicle employed. Pilocarpine nitrate or salicylate is less energetic. It does not cause neuralgic pains. It is suitable for slight cases. The myotics in narrowing the pupil diminish tension, and this effect, the mechanism of which is little known, is very real. It is only palliative, never curative, and may be *nil* in certain cases. Under these circumstances it is necessary then to have recourse to surgical means, paracentesis, sclerotomy, iridectomy, etc. All these operations, requiring special skill and training, cannot be performed by every physician.

Acute Glaucoma. 1. At the beginning of the prodromic attacks myotics, combined with antiphlogistic treatment, may suffice to cause the attack to rapidly subside; the myotics should be employed in strong solutions—eserine in oil, or a mixture of eserine and pilocarpine. A recurrence is to be carefully watched for, and the patient or his family warned of the gravity of the disease and of the absolute necessity of preventing recurrence. During the attack instillations of eserine are to be employed morning, noon, and night. The effect of the myotics lasts but seven or eight hours. After the attack is over an instillation, morning and night, suffices, and the strength of the alkaloid is to be diminished. The myotic is to be kept up for a long time; indeed, it should never be entirely abandoned. An eye which has once suffered a single attack is predisposed to others, and it is rare that such do not occur. Myotics are, therefore, employed to put off such occurrence as

long as possible, and if a new prodromic attack does supervene it is to be treated as before, provided that the pupil becomes well contracted under the myotic and the attack rapidly disappears. Medical treatment is only to be employed in the beginning in the prodromic period, and on condition that the attack quickly yields to the myotics. The latter are to be continued in the interval between the attacks. But if the attacks are frequent and of long duration, the question of surgical intervention is to be considered. The operation may definitely arrest the progress of the affection. An iridectomy is the more efficacious the sooner it is done. But even then medical treatment is not to be neglected at the time of an attack; it will relieve pain, gain time until the arrival of the surgeon, and place the patient in better condition if an iridectomy is necessary. It cannot be sufficiently insisted upon that no mydriatic is ever to be employed in any form of glaucoma.

2. *Treatment of an Attack.* Iridectomy at once. No other choice is offered to the surgeon. Unquestionably the operation is not successful in all cases (about one-third), but it is the only measure which is successful in acute and subacute glaucoma. In chronic glaucoma the treatment is medical (see below). Iridectomy in glaucoma to be effective must be done early. We are still ignorant of the exact way by which the operation regulates the tension, but we can determine the anatomical conditions which must be present if iridectomy is to be successful in lowering tension and those in which the operation will be powerless. These conditions are to be looked for in the angle of the chamber. If it is obliterated the operation will be without effect. The iridectomy should be very wide, of keyhole shape, and before doing it myotics should be instilled into the eye. If the tension is very high and the anterior chamber obliterated a small sclerotomy should be done first, or, better still, scleral puncture with a Graefe knife, and the operation should be postponed until the next day or day after that when the anterior chamber is re-formed. It is only in this way that disasters can be avoided which may follow an iridectomy done upon an eye with excessive tension, luxation of the crystalline lens, grave intraocular hemorrhage, etc. Thus in absolute glaucoma the operation which may give good results, even under these circumstances, is only to be done with extreme prudence. Careful preliminary examinations should determine whether the glaucoma is of the hemorrhagic variety, in which case iridectomy must be rejected; or whether the absolute glaucoma is due to a tumor. In the first case arteriosclerosis is usually present. In the second the tension fluctuates greatly.

Done under these conditions iridectomy is capable of giving excellent results, definitely relieving the attack and preventing recurrences. After the operation the tension gradually becomes normal, the inflammatory symptoms yield, the sight returns to what it was before the attack, and the tension is little or not at all raised. But such an ideal result is not always obtained. In some cases the good effect is but slight. In others the disease progresses, or the iridectomy may even hasten the fatal process. This result is sometimes observed in chronic glaucoma, but very rarely in acute.

It is impossible to foresee, certainly, before operation, the effect of iridectomy, but there are certain points to be taken into account. The length of time the attack has lasted is of capital importance for the prognosis. If the iridectomy is done early, the first or second day after the beginning of the

attack, when the retina and papilla have not been exposed to the increased pressure long enough to be organically altered, diminution of tension, relief of pain, and re-establishment of vision may be hoped for. In the opposite case, especially where light perception and projection are faulty or absent, the prognosis must be very guarded. The operation may diminish tension and relieve pain but the vision remains compromised. The age of the patient is also of importance. Glaucoma is more serious in proportion to the age of the subject.

Iridectomy will not always prevent the return of new attacks. It is then necessary to open the old cicatrix or to make an incision in the sclera further back; these may give excellent results. If they fail, a second iridectomy is to be done opposite the first. In all cases myotics should be continued a long time (several months and even years). They should only be abandoned when it is certain that the disease has been definitely cured and shows no tendency to relapse. The effect of the myotic may at first sight appear doubtful, the sphincter of the iris being destroyed. It is, however, very real and capable of causing those slight and transient attacks of increased tension to disappear, which may occur after iridectomy—proof that the myotics do not act solely by contraction of the pupil. Iridectomy is the operation of selection in acute glaucoma. It should never be done, on the contrary, in hemorrhagic glaucoma and infantile glaucoma (buphthalmia), for which recourse must be had to less radical procedures (small sclerotomies, very narrow paracenteses, etc.); iridectomy in these cases may end in disaster.

3. *Chronic Irritative Glaucoma.* This form rarely constitutes a mean between acute and simple chronic glaucoma; iridectomy may be done at the time of the attack or in the intervals, especially if they recur frequently, the results are less certain. The operation is never so urgent; it may be replaced temporarily by sclerotomy or repeated paracenteses. According to the effect from these, it is possible to judge of what is to be expected from the iridectomy. Myotics are to be employed with or without surgical treatment.

4. *Simple Chronic Glaucoma.* The treatment is by myotics alone. Operation is without effect and may even be injurious. The myotics must be used for a long time and very regularly, three times daily. Pilocarpine nitrate, 1 to 50, suffices in mild cases; severe forms require eserine. Very narrow paracenteses and small sclerotomies are, as a rule, the only operative procedures permissible. The degree of tension furnishes an indication; if this is at all pronounced the prognosis is more favorable; surgical intervention may effect real improvement. M. Dianoux proposes sclerotomy with the massage. The evening of the operation gentle pressure is made upon the globe by the index fingers, alternating as in testing the tension, in order to separate the lips of the wound and determine partial evacuation of the aqueous humor; the manipulation is repeated morning and evening for five or six days, and the patient is taught to do it himself. Myotics are instilled into the eye for a long time; quinine and iodide of potassium are given internally. Finally, if the disease continues to progress, section of the sympathetic is justifiable. This operation is of too recent date to be judged definitely. The results seem to be but little encouraging. Improvement, even if it occur, is never permanent.

Etiology and Treatment of Convergent Squint.—WORTH (*Lancet*, May 11, 1901) presents conclusions based on 1278 personal cases. Squint is not simply a deviation of the visual axis of one eye as defined in the textbooks. In addition to the deformity there are four other elements. 1. Deficient development of the fusion faculty. 2. Suppression of the image of the deviating eye. 3. Amblyopia usually. 4. Refractive error. Squint is not a muscular defect. In over 83 per cent., outward rotation of each eye was normal. In 9 per cent. abduction was defective in each eye, and in only 7.7 per cent. the defect was confined to the deviating eye. In constant squint the zero of convergence is some cross-eyed position, instead of parallelism. Of constant squint, 89 per cent. are monolateral, and 11 per cent. alternating. In 809 of the 1278 cases the age at which the deviation appeared is as follows: Before one year of age 102 cases, about 12 per cent.; between one and two years, 142 cases, about 17.5 per cent.; between two and three years, 195 cases, about 24.5 per cent.; between three and four years, 159 cases, about 19.5 per cent.; between four and five years, 91 cases, about 11 per cent.; between five and six years, 58 cases, about 7 per cent.; after six years of age 62 cases, 7.5 per cent. Thus, in 74 per cent. the deviation appeared before the end of the fourth year; and in only 7.5 per cent. was it delayed until after the end of the sixth year. At the first appearance of a monolateral squint there is often a considerable degree of congenital amblyopia in the deviating eye, and occasionally in the other eye also; but the deviating eye always first has the power of fixation when the other is covered. If the case is not properly treated the deviating eye becomes blinder from disuse. After a time in a considerable proportion of cases the deviating eye loses the power of central fixation. This loss is especially likely to occur in young squinters; so that there is a wide-spread belief that an eye which deviates during the first twelve months of life is very blind; yet this is not the case. Thirty per cent. treated by glasses alone eventually become straight—the spurious squint of infants. Ocular co-ordination is very rudimentary during the first few months of life. It often happens that the eyes momentarily deviate. This is of no importance. If, however, true squint be present, treatment is demanded without delay.

Etiology. The essential cause of squint is a defective fusion faculty. All other causes are but predisposing—hyperopia, anisometropia, congenital amblyopia. Heredity is a very strong predisposing cause. In 1028 cases there was a history of squint in father, mother, brother, or sister in 51 per cent.

Treatment. The indications are four: First, to prevent the loss of central fixation; second, to prevent deterioration of vision; third, to train the fusion faculty; fourth, to restore the visual axes to their normal directions.

Full correction of the refractive error is to be prescribed as early as possible, even under twelve months (?). To exercise the deviating eye atropine is dropped into the straight eye. The common practice of dropping atropine into both eyes is wrong. Neglected cases with acquired amblyopia or loss of central fixation are usually quite hopeless. The author's amblyoscope is adapted for squint of any degree. Suppression of the image is overcome by unequal illumination of the object slides. The variety of the pictures amuse and interest the child.

Operation. If binocular vision cannot be obtained, operation is to be deferred to the age of eight or nine years. If the amblyoscope has produced a strong desire for fusion, operation may be performed at any age. Operation is indicated when periodic measurements of the angle of squint shows that this is either not decreasing or decreasing very slowly. The dynamic convergence should always be examined. If the fixing eye suddenly fixes a nearer object, a dynamic convergence is superadded to the abnormal static convergence. If the dynamic convergence is very deficient, simple tenotomy is strongly contraindicated. If the fusion faculty has been developed and binocular vision is expected after operation, a tenotomy is never done except in combination with an advancement. The author maintains that the great cause of failure in advancement is the difficulty of getting the sutures which pass through the muscles to hold. The tough fibrous tissue at the margin of the cornea furnishes a suitable anchorage.

Ophthalmic Complications of Plague.—MAYNARD (*British Medical Journal*, September 14, 1901) reports twelve cases during the recent epidemic in Patna, India. Six recovered with one eye sound. In the eighteen eyes the following lesions were noted: Cornea, hazy in four, opaque in two, and sloughed in four; iris, iritis in twelve cases from the mildest to occlusion pupillæ; sclera, staphyloma in two; lens, hazy in seven and quite opaque in five. The media were hazy in six; the fundi showed hemorrhages in one. The tension was diminished in twelve cases. Vision *nil* in five, light perception in eight, and fairly good in five.

DERMATOLOGY.

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Multiple Nodular Melanocarcinoma of the Skin from a Nævus.—A. RAVOGLI (*Journal of Cutaneous and Genito-Urinary Diseases*, June, 1901) describes the case of a married woman, aged thirty-three years, the mother of two children, who from birth had a pigmented nævus verrucosus, the size of a silver quarter-dollar, in the middle of the sternal region. At the age of thirty-one years, while pregnant with her second child, it first began to grow. It was excised. Two months later numerous disseminated, dark-brown nodules appeared around the scar and for a considerable distance away from it, accompanied by painful itching sensations, preventing sleeping.

Microscopical examinations of excised nodules were made, and a question of interest was whether the disease should be regarded as being sarcoma or carcinoma. Clinically, the growth looked like sarcoma, but the microscope made the diagnosis of carcinoma certain, thus confirming the views and the doctrine of Unna on this important subject. The author thinks that cases of this kind are identical with those designated in olden times as *noli me tangere*.

Eruptions Due to Orthoform.—W. DUBREUILH (*La Presse Médicale*, May 18, 1901) states that while it is generally admitted that this drug is innocuous and is usually well tolerated, the fact that it is sometimes productive of eruptions should be kept in mind in prescribing it. The eruptions in the skin, when they occur, are usually (1) erythematous, complicated or not by vesiculation or pustulation; or (2) ulcerative and gangrenous. Two typical instances of the latter variety are given, both in women, one involving the anal and genital region, the other the hands, the lesions being both ulcerative and gangrenous. The author calls attention in particular to the observation, not heretofore noted, that the analgesic property of the drug persists upon the lesions which it itself has produced upon the skin.

Chaulmoogra Oil Subcutaneously Injected in Leprosy.—DU CASTEL (*La Semaine Médicale*, March 6, 1901) reports the results of treatment according to this method in four cases, with the dose of seventy-five grains daily. In general, the injections benefited the leprosy lesions, especially those of ulcero-tuberculous form; but they always gave rise to severe pain and often to inflamed nodes, and occasionally to grave fatty embolism, so that this method of treatment should only be employed in very rebellious cases, and never in the arms (as advised by Tourtoulis Bey), but only in the subcutaneous cellular tissue of the thighs or abdominal walls.

Crude Petroleum of No Value in Baldness.—G. T. JACKSON (*Journal of Cutaneous and Genito-Urinary Diseases*, June, 1901) is to be commended for the frankness with which he discusses the value of remedies. In the case of crude petroleum he found that no good results followed its local application for a period of from three to seven months, tried in a number of patients affected with baldness. The disagreeable odor of crude petroleum may in part be overcome by using a mixture of one part of cologne water and two parts of the oil.

Action of Concentrated Light on the Skin as a Therapeutic Agent.—A. RAVOGLI (*Journal of Cutaneous and Genito-Urinary Diseases*, December, 1901) gives his experience in the treatment of several cases of lupus vulgaris and lupus erythematosus, in all of which it was beneficial and in some very satisfactory. The apparatus employed was that made in accordance with Finsen's principles as modified by Lortet and Genoud, of Lyons. It consists of an arc light, the rays passing through a small condenser. In this way the force of the heat rays are diminished, which are absorbed by nearly any medium in the condenser. The lenses are of quartz, and water circulates in them and in the shield which contains the light, so that the apparatus is

perfectly cool; thus the heat rays are prevented from going through, without any obstacle to the passage of the chemical rays. The photochemical intensity is so strong that a ten to twenty minutes' exposure is sufficient. The method of treatment is based exclusively on the application of the chemical rays, blue, violet, and ultra-violet. The physiological action of the treatment consists of a local erythema, sometimes followed by the formation of vesicles, ending in six or eight days in epidermic exfoliation; followed by diminished congestion and re-absorption. Hypertrophic lupus nodules become flat, small, and in the middle show a kind of necrotic appearance, and then disappear. The effect on ulcers is admirable. According to the size of the lesion, large or small lenses are employed. Reference is made to Finsen's now well-known work and to his satisfactory results therapeutically.

The Histopathology of Lupus Erythematosus and of the Elastic Fibres.

—P. H. SCHOONHEAD (*Archiv für Dermatologie und Syph.*, Bd. liv. p. 163) excised skin in 11 cases of this disease, and examined the same after preparation in alcohol, formol, Müller's and Flemming's solutions, and staining with various methods. He concludes that in the beginning the inflammatory process attacks the reticular portion of the skin, and that the sebaceous and sweat glands are involved in the earliest stage. Later the connective tissue undergoes hypertrophy, the areas of infiltration, formerly circumscribed, become more diffused, and the epithelial layers assume a distinct tendency toward keratinization. When the progressive changes come to an end marked degenerative changes occur in the elastic fibres, and it is these changes that cause the peculiar scar-like atrophy so characteristic of the latter stages of the disease. This atrophy takes place in the upper strata of the corium, and herein lies the explanation of the scars being so superficial and soft. The author considers that the disease has such distinctive histological features as to entitle it to a place distinct from all other diseases of the skin.

The So-called Angioneuroses of the Skin.—L. TOEROEK (*Archiv für Dermatologie und Syph.*, Bd. liii. p. 248) considers that writers have committed an error in their interpretation and acceptance of the cutaneous angioneuroses. From a review of the various well-known authors (especially German) and his own observations, he concludes that all the symptoms which serve as a basis for considering certain pathological changes in the skin as due to an angioneurosis and to an inflammatory process cannot be regarded as such; and that the cutaneous manifestations of urticaria, erythema multiforme, and erythema nodosum cannot be looked upon as simple "inflammations."

Parakeratosis Variegata.—T. COLCOTT FOX and J. M. H. MACLEOD (*British Journal of Dermatology*, September, 1901) report a new case of this rare dermatosis, the first observed in London. The patient was a healthy adult male with a good family and personal history. At the age of thirty-one a circumscribed red patch, covered with fine scales, appeared in the lumbar region, unattended by subjective symptoms, except cracking on bending. About the same time the palms became thickened and the sense

of touch dulled. In six months the patient was covered all over except the face with a hard, dry skin. The scalp was scurvy, the hair falling out to some extent, but the nails were healthy. This condition of the skin persisted, in spite of various forms of treatment, for five years without change. The efflorescence consisted of a hyperæmic mottling in the form of a network, the threads of which were made up of macules and slightly raised, scaly papules. There was hyperkeratosis of the palms, and, to a less extent, of the soles. The author's conclusions from a study of this case and those reported by other observers are as follows: Parakeratosis variegata is a clinical entity. It attacks chiefly the male sex, usually occurs in adult life, and the patients are generally in robust health when attacked. We know little of its etiology, but its general configuration and histology suggest a vasomotor disturbance associated with œdema and infiltration of cells in the corium and secondary changes in the epidermis. The initial lesion is a macule, or maculo-papule of small size, flat on the surface, and covered with a fine, adherent scale, which may be scratched off without causing bleeding. By the coalescence of the lesions a peculiar retiform arrangement results, in which areas of normal skin are enclosed, and which, combined with differences in the color of the lesions in the more dependent parts of the body, produces the marbled or variegated appearance of the dermatitis. It affects the skin almost universally, except, as a rule, the face, scalp, palms, and soles. It is subject to remissions and exacerbations, but is peculiarly chronic in its course. Marked subjective symptoms are absent; and it is resistant to local treatment. It consists histologically of a superficial inflammation affecting the subepidermal layer, with dilatation of vessels, œdema, and infiltration of cells, and an œdematous condition of the epidermis with more or less defect in the process of cornification. It may be regarded as belonging to a group of superficial inflammations of the corium, with secondary changes in the epidermis, which the authors have provisionally called "Resistant Maculo-Papular Scaly Erythrodermias."

Lepra.—VICTOR BABES (*Journal of Cutaneous and Genito-Urinary Diseases*, December, 1901, p. 576), the well-known pathologist and clinician, contributes a valuable book on this disease, chiefly the result of personal experience. He is a firm believer of the contagiousness of the disease, and substantiates his views with impressive observations obtained in his native land, where lepra is prevalent. He regards external unfavorable conditions such as climate and diet as favoring, but not as producing, the disease. Neither does he regard the disease as hereditary. When the children born of leprous parents are removed from their surroundings they rarely become lepers. The bacillus of lepra up to the present date has not been successfully cultivated. It is allied to the tubercle bacillus—the general and local reaction of leprous individuals when tuberculin is injected into them seems to confirm this view. The chief prophylactic remedy is seclusion and governmental supervision, the establishment of lepra districts and colonies where the disease is prevalent.

Acetanilid-Iodized Starch Powder as a Local Remedy.—T. G. LUSK (*Journal of Cutaneous and Genito-Urinary Diseases*, December, 1901) speaks

highly of the value of the following combination in numerous diseases, applied in the form of a powder or a paste: Acetanilid, 3j; zinc oxide, 3iij; iodized starch, 5 per cent. 3iv. It is necessary that the iodized starch be properly prepared and the acetanilid finely pulverized. Sufficient water is added to make a paint or paste, to be applied with a stiff brush. Liquid albolene, benzoin or olive oil may be used instead of water when the application is intended for dry surfaces or ulcers; a gauze bandage may be used to prevent it from being rubbed off. When dry the powder is of a light drab color, when wet of a slate color, but when in contact with pus it turns white, showing that the iodine has been liberated. This combination of the drugs gives an antiseptic, astringent, soothing, and protective remedy, having remarkable healing properties, useful in eczema, ulcers, dermatitis from all causes, including superficial burns, impetigo sycosis, herpes zoster, and chancroids.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Bacterial Contamination of Milk.—In order to determine the degree to which milk is necessarily contaminated during milking, and the extent of bacterial growth which occurs during transit from the dairy to the city, DR. W. H. PARK, of New York (*Journal of Hygiene*, July, 1901, p. 391), undertook a series of investigations which yielded most striking and important results, which indicate that milk with far less bacterial contamination than is commonly the case can be supplied without appreciable increase of expense to the farmer and shipper. The milk of large cities comes necessarily from a wide extent of country, and part of it at least is, on arrival, already from twenty-four to forty-eight hours old, during which time the original bacterial content has enormously increased. Of the influences which determine the number of bacteria in the milk of healthy cows, cleanliness in procuring, the degree to which it is cooled, and the length of time it is kept can be almost completely controlled. Reference is made to the inexcusable lack of cleanliness in the methods of procuring milk and of care in cooling and keeping it during transportation, and to the unnecessary delay in shipping.

Milk obtained from cows where every reasonable means was taken to insure cleanliness, cooled within an hour to 45° F., and subsequently kept at that temperature, yielded after forty-eight hours an average of less than 11,000 bacteria per cubic centimetre. Taken during winter in well-ventilated, fairly clean but dusty barns, and cooled within two hours to 45° F., the visible dirt having been cleaned off the hair of the udder; the milker's

hands having been wiped off, but not washed; the milk pails and cans being clean, but the straining cloths dusty, it averaged after forty-eight hours 75,000 bacteria per cubic centimetre. Taken from more or less dirty cows stalled in ordinary barns, the teats having been cleaned slightly in the usual way by running the unwashed hands over them once before milking, it averaged after forty-eight hours in winter and in warm weather, respectively, 210,000 and 680,000 bacteria per cubic centimetre. Twenty samples of ordinary city milk taken immediately on arrival, many of them having been transported over 200 miles, yielded from 52,000 to 35,200,000 bacteria per cubic centimetre. Milk as sold in the shops yielded the following average figures: From the poorer tenement districts in midwinter, 1,977,692; in September, 15,163,600. From the more well-to-do districts in midwinter, 327,500; in September, 1,061,400. The influence of temperature was well shown by the results obtained on allowing portions of the same specimen to stand at different temperatures. Kept for twenty-four hours at temperatures below 50° F., no marked change occurred in the number of bacteria (3000) originally present per cubic centimetre, but at higher temperatures most astonishing growths occurred. Thus, at 60° F., 180,000; at 68° F., 450,000; at 86° F., 1,400,000,000, and at 94° F., no less than 25,000,000,000 per cubic centimetre. At the end of forty-eight hours, the specimen kept at 60° F. had increased its bacterial content from 180,000 to 28,000,000, and that at 68° F. from 450,000 to 25,000,000,000. Those kept at the lower temperatures had not materially changed.

Park concludes that the most deleterious changes which occur in milk are not those due to ordinary adulteration, but are those produced by the multiplication of bacteria, and holds that it is the duty of health authorities to prevent the sale of milk rendered unfit for use through the bacteria and their products, and to force gradually the farmers and middlemen to use cleanliness, cold, and despatch in the handling of their milk. As to standards, he believes in beginning with one of 500,000 for milk on entrance and 1,000,000 on delivery. These figures are conceded to be ten times as high as milk should show, but by the enforcement of such a standard the farmer would receive a compulsory education in the necessity of preventing the presence and multiplication of bacteria.

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JUNE, 1902.

OBSERVATIONS ON THE NATURE OF THE BENCE JONES
ALBUMIN.

BY CHARLES E. SIMON, M.D.,
OF BALTIMORE.

THE clinical history of the patient, whose urine I was enabled to study through the courtesy of the attending physician, Dr. N. E. B. Iglehart, of Baltimore, will be reported in greater detail at another place by Dr. L. P. Hamburger. A preliminary report has appeared in the *Johns Hopkins Hospital Bulletin*¹ of February, 1901. From this I have extracted the following brief account of the patient's illness, which is supplemented by a communication from Dr. Iglehart on the subsequent course of the disease.

The patient, a lady, aged forty-nine years, whose previous health had been good, was suddenly seized with sharp pain over the ninth left rib near its cartilaginous attachment (August, 1900). The pain was severe, and increased on deep inspiration. There was tenderness on pressure over the painful point. The condition at the time so nearly resembled a fracture that it was supposed the patient had injured the rib. A history of trauma, however, could not be obtained, and there was neither crepitus nor a friction rub. Within three weeks the pain had disappeared. She was again seen in September, and at that time complained of nausea, without apparent cause. She had lost thirteen pounds in weight, and her general health had manifestly suffered. Early in October she was again seized with pain, but this time in the region of the eighth rib, in the mid-axillary line. The patient then remarked that since the past summer she had taken more water and had voided a larger quantity of urine than usual (on one occasion 3500 c.c. were passed in twenty-four hours).

¹ L. P. Hamburger. Two Examples of Bence Jones Albumosuria Associated with Multiple Myeloma.

Dr. Oiler saw the patient on November 30th, when, aside from a slight pallor of the visible mucous membranes, the physical examination was negative. In February of the following year a slight but definite elevation was palpable over the eighth right rib in front, of the size of a lima bean. The points referred to continued to be tender to pressure throughout the life of the patient. Subsequently she suffered a great deal of pain, the attacks becoming more and more frequent, but separated even then by periods of comparative comfort. Especially in the upper part of the left thigh there was much pain, which began about three months before death. For a while she used a cane; later she was obliged to resort to crutches, and finally she could not walk at all, and had to take to a wheel-chair. There was no deformity of the bone apparent, and palpation revealed no thickening of the femur, but it was very painful. There was marked muscular atrophy. The general loss of flesh increased, and toward the end amounted to about forty pounds. Nausea and vomiting became more and more marked, and were finally uncontrollable. Even small amounts of water could not be retained. The anæmia also increased markedly, but at no time suggested a pernicious type, so far as the general appearance of the patient went. An examination of the blood, however, was not made. The polyuria which was observed earlier in the disease continued to the last three weeks, but was not so marked as at first. During the last ten days the urine was scant, but the amount had not been estimated. Five days before death spontaneous fracture of the left femur occurred in its upper third, and with gradually increasing weakness death occurred on August 16, 1901. A complete autopsy, unfortunately, could not be made. Examination of the sternal end of what was regarded as the eighth right rib, however, very curiously, showed no abnormality.

The diagnosis of multiple myeloma was made at the time of the second attack of pain, on the basis of a urinary examination. Of this Dr. Hamburger writes as follows: A specimen taken from the collected amount of twenty-four hours (3500 c.c.) was very pale, of acid reaction, with a specific gravity of 1004. It gives a white ring when floated over nitric acid. Heated to a temperature of about 55° C. a heavy, milky-white precipitate appeared. On boiling the fluid becomes clearer to become more turbid again on cooling. The addition of acetic acid to the fluid after reaching its maximum turbidity causes it to clear again. A few drops of nitric acid yield a precipitate which dissolves completely on boiling and reappears on cooling. In the Esbach albumimeter the proteid content reached 0.27 per cent. The urine gives a strong biuret reaction. No casts were seen in a centrifugalized specimen. From these reactions Dr. Hamburger concluded that the urine contained the so-called albumose of Bence Jones, which, according to Kahler, is only found in cases of myeloma. This diagnosis was accordingly made. With Dr. Hamburger's consent I then undertook a more detailed study of the substance in question, with which the present paper has to deal. Through the courtesy of Dr. Iglehart about 16,000 c.c. of the urine were placed at my disposal, which was partly examined at once, partly after preservation with thymol or chloroform.

The urine was sent to me in bulk, without any attempt at separate collection, for periods of twenty-four hours, so that I am unable to give accurate figures in this respect from personal knowledge. Dr. Iglehart, however, informs me that during the period of polyuria, which was first

noted about August 10, 1900, and which extended over nearly a year, the average elimination varied between 2000 and 3500 c.c.

General Properties of the Urine. The urine was very pale and markedly cloudy. Microscopical examination showed that the turbidity was referable to numerous epithelial cells, derived from the genitals, and a fairly large number of leucocytes, probably of the same origin. There were no crystalline elements, no red blood-corpuscles, and no casts. Even after standing for five months a crystalline deposit of the albuminous body was not observed, nor did the substance separate out in amorphous form.

As in Magnus-Levy's case, it was noted that the urine underwent ammoniacal decomposition much less readily than is common. The reaction was constantly acid; the specific gravity usually low, never exceeding 1015.

A study of the common constituents of the urine was not made, but it was noted that uric acid and indican were not increased and that sugar was absent. Special attention was directed to the behavior of the peculiar albuminous body, which was present in considerable amount. Quantitative estimation (gravimetrically) on two occasions gave 0.5072 and 0.5124 per cent. These higher figures, however, were only found in the first lots of urine, while later on the amount was much smaller, and an examination of a 5-litre specimen which had been kept over the summer showed the presence of only about 0.025 per cent.

The general reactions which were obtained with the native urine are the following:

1. Upon the application of heat the acid urine remains clear until a temperature of about 52° C. is reached, when the liquid begins to show a milky turbidity. This rapidly increases in intensity, and at 55° C. the entire volume of urine is perfectly opaque, so that the bulb of the thermometer can no longer be seen in the test-tube. When heated still higher the milky turbidity is replaced by the appearance of flocculi, but even as the boiling-point is reached the urine can scarcely be said to have become clearer. On subsequent cooling the flocculent precipitate is seen at the bottom of the tube as a peculiar, granular-looking sediment, and its amount does not suggest an increased precipitation during cooling.

2. Upon the addition of a drop or two of a 25 per cent. solution of nitric acid to a few c.c. of urine a slight cloud forms, which disappears on shaking. When more of the acid is added an abundant precipitate results, which is insoluble in a large excess of acid (ten times the original volume). On heating the acid solution the albuminous substance first coagulates to a dense mass, but dissolves almost entirely on prolonged boiling, with the formation of a pink foam. On subsequent cooling the precipitate reappears, to disappear again (almost entirely) on boiling.

It is noteworthy that *with the cold nitric acid test, applied in a conical glass, a far more abundant precipitation is manifestly obtained than upon the application of heat.*

3. With hydrochloric acid and sulphuric acid similar results are obtained. As a rule, three drops of the concentrated acid (as also of nitric acid) were necessary to bring about a precipitation of the albumin, which did not disappear on shaking.

4. Carbonic acid was without effect, even if the urine had been copiously diluted.

5. Upon the addition of acetic acid, even in large excess, no precipitate results. On subsequent heating the liquid remains clear.

6. Phosphoric acid also does not cause a precipitation of the substance.

7. The addition of picric acid (13 to 14 drops of a saturated solution for 5 c.c. of urine) causes the formation of a flocculent precipitate, which increases in amount on heating, but does not disappear entirely on boiling. With the mineral acids complete clearing was not always observed, but in this case the urine remained much more turbid.

8. Tannic acid causes complete precipitation after the previous addition of acetic acid. The precipitate disappears to a great extent on boiling.

9. With phosphotungstic acid, in the presence of hydrochloric acid a heavy precipitate results, which does not disappear on boiling.

10. With potassium ferrocyanide and acetic acid a precipitate is obtained which is but little soluble at 100° C.; it increases on subsequent cooling.

11. On neutralization with a dilute solution of sodium hydrate or ammonia no precipitation results.

12. On acidifying the urine with acetic acid and subsequent neutralization with sodium hydrate a precipitate results, which dissolves practically entirely on boiling and reappears on cooling.

If an excess of sodium hydrate is added to the neutral solution the neutralization precipitate disappears, and it is noteworthy that no precipitate now appears on heating; but if then the solution is again neutralized with acetic acid a neutralization precipitate appears; but, in contradistinction to the first, this does not disappear on boiling, and is only slightly soluble in the presence of a large excess of the acid.

13. If the urine is first rendered alkaline with sodium hydrate and is then neutralized with acetic acid, a neutralization precipitate results, which, as in the last instance, does not disappear on boiling, and is only slightly soluble in a large excess of the acid.

In order to obtain such neutralization precipitates, however, not too little acid or alkali must be added, as otherwise no precipitate occurs on subsequent neutralization. In one instance I treated 20 c.c. of a solution of the isolated substance (precipitation with ammonium sulphate and dialysis for eight weeks) with a few drops of hydrochloric acid—such that the solution contained 0.36 per cent. On neutralization with sodium hydrate a precipitate was then not obtained, even on heating; but this developed at once on acidifying the hot solution with acetic acid.

14. Similar results as in 11 and 12 were obtained with dilute mineral acids.

15. Alcohol, when added in the proportion of two volumes for one of the urine, causes complete precipitation of the substance; and it is to be noted that after prolonged contact the albumin loses its subsequent solubility in water.

16. On treating the urine with an excess of a saturated solution of common salt (two to three times the original volume) no change occurs, but on heating the substance is precipitated, and on subsequent boiling the solution remains turbid.

17. Upon the addition of a saturated solution of common salt to the acidified urine (acetic acid) complete precipitation occurs, which is hastened by heating to about 50° C. On boiling there was no evidence of clearing.

18. Saturation of the urine with rock salt at ordinary temperatures causes a partial precipitation, which is soluble in water. Saturation at from 35° C. to 40° C., on the other hand, results in the complete precipitation of the albumin, and it is noteworthy that this precipitate is insoluble in water.

19. Upon the addition of two and one-half volume of a saturated solution of magnesium sulphate to the urine no change occurs, even after the addition of acetic acid; but on saturating the acidified solution with the salt in substance the albumin is precipitated. This occurs at once if the urine is first saturated with the salt, and acetic acid is then added. *Saturation of the urine with magnesium sulphate without the addition of acetic acid caused no change. In a dialyzed specimen, however, which presented a neutral reaction complete precipitation could be produced by saturation with the salt in question.* (See below.)

20. Saturation of the acidified urine with sodium sulphate in substance causes the precipitation of the albumin, as in the case of magnesium sulphate. The addition of an equal volume of a saturated solution of the salt causes partial precipitation.

21. A saturated solution of ammonium sulphate, when added in the proportion of two volumes for one volume of urine, causes the complete precipitation of the substance, the reaction being neutral.

22. On dialysis the substance is not precipitated, nor does it pass through the membrane.

23. Millon's reaction: On heating the flocculent precipitate turns red.

24. Biuret reaction: An intense purplish-violet is obtained if a sufficient amount of the copper solution is added; if but little is present the color is red.

25. Reaction of Adamkiewicz: A beautiful purplish-violet results.

26. Hydrochloric acid reaction: A reddish-violet color results, but is not nearly so intense as with Adamkiewicz's test.

27. The sulphur test yields a positive result (brown color on boiling).

28. The xanthoproteic reaction is positive.

29. Molisch's reaction is not well marked, but occurs on prolonged standing.

*Reactions of the Isolated Substance.*¹ To isolate the Bence Jones substance the urine was neutralized and precipitated with twice its volume of a saturated solution of ammonium sulphate. The copious precipitate was filtered off with the aid of a suction pump, and kept as such in a two-thirds saturated solution of the salt. When first precipitated it appeared white; but later, after exposure to the air, it assumed a pinkish tint, which subsequently changed to a light terra-cotta.

To purify the substance the material was repeatedly dissolved in hot water, reprecipitated with ammonium sulphate, and finally dialyzed. It was noted, however, that it was impossible to remove all the sulphate

¹ Through the kindness of Dr. Edward S. Wood, of Harvard, I was enabled to compare the reactions of my isolated substance with a product which he obtained from the urine of a patient of Dr. Fitz (Case 13, see below). The corresponding report follows below.

in this manner, even after exposure to running water for nearly three months. The resulting solution was used for the common tests, while it was found necessary to resort to some other method of purifying the substance in order to obtain material that could be employed for purposes of elementary analysis. To this end the filtered urine was slowly heated and the albumin thus coagulated. The coagulum was washed with water, dissolved by the aid of a little sodium carbonate in boiling water, and repeatedly precipitated with alcohol. The precipitated substance was finally placed in the dialyzer for twenty-four hours and the contents of the dialyzer treated with double their volume of alcohol, so as to recover a portion of the substance that had passed into solution. The material was washed with absolute alcohol and ether, pressed out between filter paper, and dried at 105° C., when it changed to a horny, brittle mass. Purified in this manner the substance was practically free from ash, while the crude material apparently contained both iron and phosphorus. Two nitrogen estimations gave 15.12 and 15.26 per cent.

The dried substance is almost insoluble in water and dilute saline solutions at ordinary temperatures, and dissolves with difficulty even in hot water unless a trace of sodium carbonate is added. In that event, however, solution takes place with comparative readiness on heating, and it is to be noted that the solution remains clear on subsequent neutralization, providing that a sufficient amount of water is present and that too much of the soda has not been added. The *moist* substance, on the other hand, is soluble even in cold distilled water, though not so readily as the references in the literature would lead one to think. It is noteworthy that it is not nearly so easily soluble as the common water-soluble digestive albumoses. On prolonged contact with alcohol the material is rendered insoluble in water and dilute saline solutions; in such an event, however, as has already been pointed out, solution in water can be effected on heating after the addition of a little soda or a few drops of hydrochloric acid. Such a precipitate, moreover, is soluble in dilute ammonia.

The solution of my isolated substance (obtained, as first described, by precipitation with ammonium sulphate and subsequent dialysis) was of neutral reaction, and on heating became but slightly turbid. If a small amount of salt and a drop of acetic acid were added, however, coagulation occurred as in the case of the native urine. Heated *slowly*, immersed in a beaker with water, the first trace of turbidity was noticeable at 54.5° C.; at 55° C. the entire bulk of the solution was cloudy; at 55.5° C. it was decidedly opaque; the maximum intensity was reached between 58° C. and 60° C. At about 70° C. the general opacity was replaced by the appearance of a flocculent precipitate, which gradually assumed a more granular appearance and finally settled to the bottom in the form of microscopical spheruliths, while the supernatant fluid became clear. An actual solution of the substance was not observed.

On heating the solution and then adding concentrated nitric acid a flocculent precipitate occurred with the simultaneous appearance of the xanthoproteic reaction.

Upon the addition of nitric acid to the cold solution a precipitate occurred, which, in contradistinction to the native urine, was but little

soluble on the subsequent application of heat, even at 100° C., and on cooling no appreciable increase in turbidity was noted. The same result was obtained with potassium ferrocyanide and acetic acid.

On treating the solution with an acid or an alkali and subsequent neutralization a precipitate occurred, which was soluble in an excess of the reagent. To obtain this precipitate, however, it was necessary to add a definite amount of acid or alkali; if too little was used no precipitate occurred. This is shown by the following example: 20 c.c. of the clear neutral solution were treated with an amount of hydrochloric acid such that the solution contained 0.36 per cent. On neutralization with sodium hydrate no precipitate occurred, and on boiling the solution remained clear. If, then, a little acetic acid was added, however, while the solution was still hot, precipitation occurred at once.

On saturating the *neutral* solution with magnesium sulphate at the temperature of the room, complete precipitation occurred on standing for less than twenty-four hours.

The limits of precipitation with ammonium sulphate were 2.3 and 3.8, respectively.

On salting with common salt to saturation a partial precipitation of the substance occurred at ordinary temperatures, the reaction being neutral. On adding two to three times the volume of a saturated solution of common salt to a solution of the isolated substance, however, no precipitation occurred so long as the reaction was neutral, but on adding a few drops of acetic acid a turbidity occurred at once.

On salting with rock salt at ordinary temperatures an abundant precipitate also occurred, which, like the one preceding, was soluble in water.

Upon the addition of a dilute solution of copper sulphate precipitation occurred at once. The biuret reaction was positive—an intense purplish-violet.

In its other reactions the isolated substance showed the same general behavior as the native urine (which see).

From these reactions of the urine and the isolated substance it is clear that the albuminous body in question is not one of the common albumins which we meet with in ordinary cases of albuminuria, but clearly resembles the substance which was first observed by Bence Jones and MacIntyre in a case of supposed osteomalacia.

Special Reactions. Behavior of the Substance on the Application of Heat. Of especial interest is the behavior of the urine on the application of heat. Quite constantly the first indication of a turbidity appeared as the 52° C. mark had just been passed and before 53° C. had been reached. At 55° C. the liquid had become quite opaque, so that it was impossible to see the markings of the thermometer where it was immersed, and still later even obscuring the bulb of the instrument. The turbidity was distinctly milky in appearance, and was first noted in the surface layer, from which it gradually extended downward. Later the starting-point of the precipitation appeared to be wherever the urine came in contact with the walls of the test-tube or the bulb of the thermometer. Heated beyond 55° C., viz., to 60° C., the turbidity sometimes increased a little more, providing that the elevation of the temperature was brought about gradually, as by immersing the tube in a large beaker full of water and heating this with a comparatively small flame. On further heating no increase in opacity was noted, but

as 75° C. to 80° C. was reached the milky turbidity gave way to the appearance of a coarsely flocculent precipitate, which, however, did not dissolve even at 100° C. If left to itself the precipitate settles to the bottom, while the supernatant fluid becomes clear, and remains clear on gradual cooling. If, however, the liquid is suddenly cooled, as by immersion in cold water, the supernatant fluid becomes turbid. A similar result is reached on filtering the hot liquid, when the first portion which passes through at once turns cloudy on coming in contact with the cold walls of the tube or beaker.

Providing that the process of heating is carried on very gradually and the urine is then allowed to cool of itself, a solution is obtained on filtration which no longer becomes turbid on subsequent heating at any temperature, but in which an albuminous substance is manifestly still present. Barring its different behavior toward heat, this solution gives the same general reactions as the native urine, viz. :

1. On treating with potassium ferrocyanide and acetic acid the solution becomes turbid, and on heating the turbidity does not disappear, but becomes even more pronounced. At 100° C. it is but slightly soluble.

2. With nitric acid a marked reaction is obtained.

3. After the addition of acetic acid, even in excess, the solution remains clear.

4. A marked biuret reaction is obtained—a purplish-violet, as in the native urine.

5. Treated with an equal volume of a saturated solution of common salt, no change occurs; but upon the addition of a few drops of acetic acid a turbidity results, which increases upon the application of moderate heat, but disappears on boiling, to reappear on cooling, etc.

6. Treated with two volumes of a saturated solution of ammonium sulphate the albuminous substance is completely precipitated.

The peculiar behavior of the urine on heating, on first consideration, suggested the presence of two distinct albuminous bodies in the urine, one of which could be coagulated by heat, while the other was apparently non-coagulable. It was ascertained, however, that such was not the case, and that it is possible by a suitable variation of the reaction, and by increasing or diminishing the amount of salt present, to increase or to diminish at will the relative amount of the coagulable as compared with the apparently non-coagulable portion. With an alkaline specimen it was thus found that after heating a much larger amount of the albumin remained in the filtrate than in the case of the acid urine, and as I have already indicated it is possible to completely precipitate the total amount of the substance by heating, after the previous addition of an equal volume of a saturated solution of sodium chloride to the acidified urine (acetic acid). In such an event the resulting filtrate is free from albumin.

It was also noted that the rapidity of heating is of marked influence upon the extent to which coagulation occurs. The more gradually this is done the more extensive is the coagulation. That the temperature at which this occurs is also dependent, to a certain extent at least, upon the amount of salt present was first shown by Magnus-Levy, and was also noted in the present instance. It was thus ascertained that whereas coagulation occurred in the native urine at a temperature of from 52.5° C. to 54° C., it was necessary to heat to 55° C. or 56° C. if a double volume

of a saturated solution of sodium chloride had been added. The further addition of salt then lowers the temperature of coagulation, and on saturating the urine with salt in substance complete coagulation may occur at 37° C. already if the liquid is kept at this temperature for several hours.

This influence of the relative amount of salt upon the degree of heat at which coagulation occurs is well shown in the following table, which is taken from Magnus-Levy:

| <i>Urine.</i> | <i>20 per ct. salt sol.</i> | <i>Water.</i> | <i>First turbidity at</i> |
|---------------|-----------------------------|---------------|---------------------------|
| 2 c.c. | 0.0 c.c. | 2.0 c.c. | 54.0° |
| 2 " | 0.2 " | 1.8 " | 54.5 |
| 2 " | 0.4 " | 1.6 " | 55.0 |
| 2 " | 0.8 " | 1.2 " | 55.0 |
| 2 " | 1.2 " | 0.8 " | 55.5 |
| 2 " | 1.6 " | 0.4 " | 56.0 |
| 1 " | 3.0 " | 0.0 " | 56.5 |

Ammonium chloride, in my experience, did not influence the temperature of coagulation, while it apparently renders the precipitated substance more readily soluble at 100° C. This, however, is only observed if a definite amount of the salt is present, which should not exceed 1 to 2 per cent. saturation. At times, moreover, no result at all was obtained without any apparent reason. On one occasion two specimens of urine were diluted, the one with 2 c.c. of a saturated solution of ammonium chloride and the other with water. On heating neither became especially cloudy (the heat was rapidly applied). On cooling the slight turbidity persisted and even increased to a slight extent. On subsequent heating the ammonium chloride specimen now became more turbid than the control, and did not clear whatever on boiling. Upon the addition of a little acetic acid the turbidity increased even more.

Of great interest in this connection is the observation of Spiro¹ that the addition of urea to solutions of albumins increases the degree of temperature at which coagulation occurs, and that a point may be reached at which no coagulation whatever takes place. In accordance with Hofmeister's suggestion, that this might possibly account for the varying statements as regards the solubility of the precipitated albumin of Bence Jones at 100° C., Magnus-Levy undertook a special examination of his substance in this direction. His results are quite in accord with those reached by Spiro in the case of egg albumin and serum albumin. I have repeated this work, and was able to show that, as a matter of fact, a point is reached, on the gradual addition of a urea solution in increasing amounts, at which coagulation no longer occurs; but I was not able to demonstrate that the temperature of coagulation was correspondingly raised. So long as a turbidity occurred at all the temperature was the same until a certain percentage was reached, when the solution remained clear altogether. These results are shown in the following tables:

¹ K. Spiro. Ueber die Beeinflussung der Eiweisscoagulation durch stickstoffhaltige Substanzen, *Zeit. f. physiol Chem.*, 1900, vol. xxx. p. 182. See also the recent work of W. Pauli and P. Rona, *Untersuchungen über physikalische Zustandsänderungen d. Kolloide*, *Beiträge z. chem. Phys. u. Pathol.*, 1902, vol. ii. p. 1.

EFFECT OF UREA UPON THE COAGULATION OF BENGE JONES ALBUMIN

| Urine (neutral). | Urea solution (50 per ct.). | Water. | Per cent. of urea added. | On warm- ing. | At 100° C. | On cooling. | Repeated warming and cooling. |
|------------------|-----------------------------|----------|--------------------------|-------------------|------------------|------------------|-------------------------------|
| 2 c.c. | 0.0 c.c. | 2.0 c.c. | | Marked turbidity. | Slight clearing. | Slight increase. | The same. |
| 2 " | 0.2 " | 1.8 " | 2.5 per ct. | Marked turbidity. | Marked clearing. | About the same. | The same. |
| 2 " | 0.4 " | 1.6 " | 5.0 " | Momentary cloud. | Clear. | Clear. | Clear. |
| 2 " | 0.5 " | 1.5 " | 6.25 " | Clear. | " | " | " |
| 2 " | 0.6 " | 1.4 " | 7.5 " | " | " | " | " |
| 2 " | 0.7 " | 1.3 " | 8.7 " | " | " | " | " |
| 2 " | 0.8 " | 1.2 " | 10.0 " | " | " | " | " |
| 2 " | 1.0 " | 1.0 " | 12.5 " | " | " | " | " |

Magnus-Levy states that by diluting the clear solution, containing an excess of urea, with water, the albumin is precipitated even at ordinary temperatures. This observation I was unable to verify in my case. After the addition of two to three times the amount of water, at least, no turbidity was noted, even on the application of heat.

In attempting to ascertain the effect of urea upon the coagulation point it was only possible to note the first appearance of a turbidity, about the maximum turbidity, about the beginning of clearing, and roughly the point of maximum clearing. A flocculent precipitate was not obtained in these experiments.

| Urine (neutral). | Water. | Urea solution (50 per ct.). | First turbidity. | Maximum turbidity. | First clearing. | Maximum clearing. |
|------------------|--------|-----------------------------|------------------|--------------------|-----------------|-------------------|
| 2 c.c. | 2 c.c. | 0.0 c.c. | 58.5° | 60.0° | 79.0° | 92.0° |
| 2 " | 1.8 " | 0.2 " | 58.0 | 60.0 | 75.0 | 92.0 |
| 2 " | 1.6 " | 0.4 " | 58.0 (7) | Clear. | Clear. | Clear. |

Like Magnus-Levy, I observed that the addition of a moderate excess of urea will dissolve the precipitate which has been produced by heat, even at ordinary temperatures, though at times this did not occur, for reasons which were not apparent.

Limits of Precipitation with Ammonium Sulphate. For purposes of comparison with other albuminous substances, the limits of precipitation with ammonium sulphate were determined both in the native urine and in a dialyzed specimen of the isolated substance according to Hofmeister's method, as described by Pick.¹ To this end a series of test-tubes was prepared, such that each contained 2 c.c. of the urine (viz., the dialyzed specimen) and an increasing amount of a saturated solution of ammonium sulphate, the mixture in each case being diluted with distilled water to 10 c.c. The concentration at which the first permanent turbidity occurred was regarded as the lower limit. The mixture in

¹ E. Pick. Untersuchungen über die Proteinstoffe, Zeit. f. phys. Chem., vol. xxiv. p. 246.

every case was thoroughly agitated, usually allowed to stand for one-half hour and then passed through a double filter; but at times it was necessary to wait for several hours (sometimes for twenty-four hours). To the clear filtrate of each tube 0.2 c.c. of the salt solution was then further added, in order to ascertain whether all of the substance had been precipitated. The concentration of the first specimen in which a further turbidity no longer occurred indicated the upper limit of precipitation. The results which were obtained in the case of the native urine were 0.7 and 4.2, and with a solution of the isolated substance 2.3 and 3.8, respectively.

Behavior on Digestion with Pepsin-hydrochloric Acid. To test the behavior of the albumin on peptic digestion a series of experiments was undertaken. The substance was previously isolated either by precipitation with ammonium sulphate or with alcohol, and purified as described. In some cases the solid substance was used, in others a solution. The degree of acidity of the digestive mixture corresponded to from 0.3 to 0.4 per cent. of hydrochloric acid. The pepsin used was a preparation of Merck's. Examination showed that this was suitable for our purposes. It was clearly soluble in water, gave only a faint biuret reaction, and, treated with an equal volume of a saturated solution of ammonium sulphate, a very faint turbidity. The digestive mixture was kept at a temperature of from 35° C. to 40° C. for a varying length of time. In the first experiment digestion was allowed to continue for a week. At the expiration of this time a thin deposit of an amorphous substance had appeared, of the nature of which nothing could be ascertained. The liquid was filtered and the filtrate neutralized. A precipitate did not occur. The solution was then acidified with acetic acid and treated with an equal volume of a saturated solution of sodium chloride and boiled. The clear filtrate was then treated with an equal volume of a saturated solution of ammonium sulphate, but after standing for nearly seventy-two hours a precipitate had not formed. There was merely a slight turbidity, which could not be removed by filtration. The solution was now further treated with the ammonium sulphate solution to the extent of one-half of the total volume, but again no precipitate resulted, even on standing four times twenty-four hours. The liquid had become somewhat more turbid, but could not be cleared on filtration. It was then saturated with the salt in substance. On standing a gummy material separated out, which manifestly represented the deutero-albumose B of Pick. The substance gave an intense purplish-red biuret reaction and a positive sulphur reaction (at first a brown color, later a precipitate of black sulphide of lead). The reaction of Adamkiewicz was positive, while Molisch's reaction was negative. Dilute copper sulphate solution caused no precipitation. On digestion with trypsin much tyrosin and comparatively little leucin were obtained.

After separation of the deutero-albumose B the clear filtrate was treated with one-tenth its volume of a very dilute solution of sulphuric acid saturated with ammonium sulphate—such that the reaction was feebly but distinctly acid. On standing even for several days a precipitate could not be obtained; only a slight turbidity resulted, which could not be removed by filtration. Deutero-albumose C, if present at all, was hence only formed in traces. On treating the filtered solution with Lugol's solution saturated with ammonium sulphate, a precipitate

formed, which on standing appeared as a gummy, black mass that closely adhered to the walls of the vessel. This was soluble in alcohol, and represents Pick's peptone B. A substance corresponding to Pick's peptone A was present, if at all, only in traces.

In the above experiments no evidence of the formation of primary albumoses was obtained; and as Magnus-Levy claims to have isolated a proto-albumose among the products of peptic digestion in his case, I repeated the experiment, allowing the digestion to continue for only twenty-four hours. The preparation used for this purpose was obtained by precipitation with alcohol, and was placed in the digestive mixture in the solid state. After twenty-four hours a small amount of an amorphous sediment was observed, as in the first instance, but it was likewise insufficient in amount for purposes of special examination. On neutralization no precipitate appeared. On the contrary, the liquid, which was distinctly turbid even after filtration, became almost clear as the neutral point was reached. It was then acidified by the addition of a couple of drops of acetic acid, and it was noted that on the addition of each drop a slight turbidity resulted, which disappeared on shaking. The solution was then treated with an equal volume of a saturated solution of sodium chloride. Upon the application of heat a beginning turbidity was noted between 52° C. and 53° C. This reached its height between 58° C. and 60° C., and was replaced between 78° C. and 80° C. by a cloudy, flocculent precipitate which became finely flocculent on boiling, but did not disappear. On cooling, what appeared to be an undigested portion of the Bence Jones albumin was filtered off, the filtrate neutralized, and further treated as described above. In this case, also, deutero-albumose B was obtained in considerable amount, while both deutero-albumose A and C were present only in traces.

As in this case, also, primary albumoses were not obtained, a third series of experiments was undertaken in which digestion was interrupted after from six to ten hours. About 10 grammes of the carefully purified substance were used and placed in the digestive mixture *en masse*. The process of "digestion" was closely followed and interrupted as soon as all the material had passed into solution. My special aim in these experiments was to separate from each other the undigested portion of the Bence Jones substance and any primary albumoses that may have been formed. To this end I first proceeded as described, viz., the solution was neutralized (no precipitation occurred) and then concentrated to a smaller volume. As in my case, the limits of precipitation of the substance were such that a satisfactory separation of the original substance from primary albumoses by fractional precipitation with ammonium sulphate was not possible, as in Magnus-Levy's case, I was forced to resort to some other plan. After a number of trials it was found that this separation could be readily effected by alcohol. It has been found that the Bence Jones substance could be completely precipitated by adding two volumes of alcohol (95 per cent.), and Pick¹ has shown that proto-albumose is soluble in alcohol of less than 80 per cent., while hetero-albumose is thrown down already in the presence of from 25 to 32 per cent. I accordingly precipitated the neutral digestive solution with an equal volume of a saturated solution

¹ E. P. Pick. Zur Kenntniss d. peptischen Spaltungsprodukte d. Fibrins, Pt. I., *Zeitsch. f. physiol. Chem.*, 1899, vol. xxviii. p. 219.

of ammonium sulphate and allowed the mixture to stand for forty-eight hours. The fairly abundant precipitate which had collected by that time was then filtered off, washed with a small amount of iced water, stirred up with water at an ordinary temperature, and the resulting solution treated with twice its volume of 94 to 95 per cent. alcohol. The mixture was then allowed to stand for at least twenty-four hours. Any undigested portion of the Bence Jones substance—and I must confess that I did not find that it was digested so very rapidly as the statements in the literature led me to believe, although it *dissolved* comparatively rapidly—was thus thrown down and could readily be filtered off. The clear alcoholic filtrate was freed from alcohol by distillation in the vacuum. There remained a *clear* solution, which gave all the reactions of proto-albumose, and which could scarcely contain any other substance. A hetero-albumose was certainly not present in this fraction. Its absence in the alcoholic precipitate also was proved in another series of experiments by fractional precipitation with alcohol. On boiling the filtered alcoholic solution, moreover, the occurrence of a turbidity was never observed if care had been taken to allow the solution to stand sufficiently long so that everything that could be precipitated by alcohol in the cold had separated out.

In this manner, then, *the formation of proto-albumose from the Bence Jones substance could be satisfactorily demonstrated.* The amount of proto-albumose which was obtained was never large, but manifestly much more abundant than the amount of fraction I which Umber¹ could isolate among the digestive products of serum-albumin, of which he writes that it was always present in such small amounts, that it only appears as a faint opalescence on treating the digestive mixture with an equal volume of the sulphate solution, no matter whether the attempt was made to isolate it after three or not until after eighteen hours. He adds: "It would thus seem as though the fraction by no means represented a transitory stage for all following albumoses, which are present in abundance, unless, indeed, it is further decomposed already in *status nascendi*." This observation I regard as important, in view of the fact that, as I shall show later on, the Bence Jones substance is in all probability a globulin, or at least closely related to the class.

After precipitation of the proto-albumose, together with the undigested portion of the original substance, by ammonium sulphate and their separation by filtration, the filtrate was then further treated as described, and with the same results. Deutero-albumose B and peptone B were always obtained in a fairly large amount. Deutero-albumose A also could be isolated in amounts sufficient to establish its identity; but of deutero-albumose C and peptone B only traces could be found, if, indeed, peptone B was present at all.

Crystallization. I have made a number of attempts to obtain the substance in crystalline form, but have thus far not been successful. I have noted, however, like others, that the granular-looking sediment which collects at the bottom of the vessel when the substance has been slowly precipitated by heating is composed of typical spheruliths, such as Hofmeister describes.

¹ F. Umber. Die Spaltung d. krystallinischen Eier und Serumalbumins sowie d. Serumglobulins durch Pepsinverdauung, *Zeitsch. f. physiol. Chem.*, 1898, vol. xxv. p. 253.

While I was about to finish my work on the present case, and was surveying the literature on "Bence Jones' albumosuria," Dr. Edward S. Wood, of Harvard University, very kindly presented me with about 50 grammes of the isolated substance, for comparison, which he had obtained from the urine of Dr. Fitz's second case, and which was reported by Wright (see below). In this case, also, the diagnosis of multiple myeloma was made during the life of the patient and confirmed by autopsy. Dr. Wood wrote me that the substance had precipitated at a temperature between 50° C. and 60° C., but dissolved by heating to boiling, and precipitated again on cooling. It contained 0.2 per cent. of phosphorus, and owing to this observation, no doubt, Dr. Wood regarded the substance as a nucleo-albumose. Elementary analysis gave the following results, which, together with those obtained by other observers, I have placed in the accompanying table:

| | C | H | N | O | S | P |
|-----------------------|-------|-------|-------|-------|-------|-------|
| Bence Jones | 52.04 | 7.08 | 15.02 | ----- | 1.09 | 0.19 |
| Kühne | 52.18 | 6.83 | 16.0 | ----- | ----- | ----- |
| Neumelster | 52.87 | 6.91 | 15.55 | ----- | 1.12 | 0.0 |
| Magnus-Levy | ----- | ----- | 15.56 | ----- | ----- | 0.0 |
| Wood | 42.8 | 7.72 | 13.85 | 35.76 | 0.16 | 0.21 |

The product which Dr. Wood used for elementary analysis was prepared by precipitating the urine with 95 per cent. alcohol, filtering, and reprecipitating with alcohol. This process was repeated at least three times, the alcohol being allowed to act upon the precipitate for several days at each time. The final purified product was dried over sulphuric acid in a vacuum to a constant weight. Comparison with the figures obtained by others suggests that even so the product was not quite pure, and I have succeeded, as a matter of fact, in obtaining it free from phosphorus by appropriate treatment.

Dr. Wood further writes me that he obtained the same substance from the diseased portion of the bone tissue, but that he could not obtain it from the bone-marrow in any other portion of the body of the patient.

The material which I received for examination had been isolated by precipitation with alcohol, and appeared as a white, pasty mass, which on prolonged standing dissolved in water, but much more readily in very dilute ammonia (0.5 to 0.25 per cent.). This solution remained clear on subsequent neutralization; on heating a turbidity appeared between 50° C. and 55° C., which cleared on boiling and reappeared on cooling. The behavior toward dilute ammonia showed that the product was not contaminated by the common albumins which may occur in the urine. Like my own substance, this also could be precipitated from its neutral solutions by salting with magnesium sulphate to saturation, and it was noted that the precipitate was soluble in water. With rock salt partial precipitation also occurred, and in this case the precipitate was likewise soluble in water. The limits of precipitation with ammonium sulphate were 1.7 and 2.8, respectively. On digestion with pepsin-hydrochloric acid a proto-albumose was formed which

could be precipitated by one-half saturation with ammonium sulphate, and which was soluble in 66 per cent. alcohol. A hetero-albumose could not be demonstrated; but, as in the first instance, I also obtained the three deutero-albumoses A, B, and C, and the two peptones A and B, though of these the deutero-albumose B and peptone B were the only products that were present in large amounts.

SUMMARY OF REPORTED CASES. It had been my intention to present a more detailed account of the various cases of Bence Jones' "albumosuria" that have been reported in the literature, but it was found that such an analysis would have occupied more space than was available. I therefore abandoned the idea, and have below merely arranged the reported cases in chronological order, together with the necessary references and the anatomical diagnosis whenever a record of an autopsy could be found.

The *first case* occurred in the practice of Drs. Watson and MacIntire, and was reported by Bence Jones,¹ in 1847, before the Royal Society of London. A clinical report of the case by MacIntire followed in 1850. The post-mortem diagnosis was "osteomalacia fragilis rubra."

The *second case* occurred in the practice of M. Doornik, of Amsterdam, in 1869, and was apparently also seen by Stokvis, who examined the urine. The clinical diagnosis in this case also was osteomalacia, but a post-mortem examination was not made. The urine was studied in detail by Kühne,² who published his results in 1883.

The *third case* was described from the clinical stand-point by Kahler in 1889. The clinical diagnosis in his case also had been osteomalacia, but post-mortem multiple myeloma was discovered. Kahler suggested that had a detailed microscopical examination been made in the two other cases it might have been found that they also were in reality cases of multiple myeloma which clinically simulated osteomalacia. He expressed the opinion that the presence of Bence Jones' "albumose" might possibly be of service in the diagnosis of multiple myeloma from osteomalacia. The urine in Kahler's case was examined by Huppert.³

The *fourth case* occurred in the service of Stokvis,⁴ of Amsterdam. A preliminary report appeared in 1891. A more detailed account of the case was given by Ribbink in 1892, and in 1893 Zeehuisen published his report of the autopsy together with the chemical examination

¹ H. Bence Jones. *Philos. Trans. Royal Soc.*, 1848, Pt. I. p. 55. W. MacIntire, *Med. Chir. Trans.*, London, 1850, vol. xxxiii. p. 211.

² W. Kühne and R. H. Chittenden. Ueber die nächsten Spaltungsprodukte d. Eiweisskörper, *Zeit. f. Biol.*, 1883, vol. xix. p. 198. W. Kühne, Ueber Hemialbumose in Harn, *ibid.*, p. 209. W. Kühne and R. H. Chittenden, Ueber Albumosen, *ibid.*, 1884, vol. xx. p. 40.

³ Huppert. Ueber einen Fall von Albumosurie, *Prag. med. Woch.*, 1889, vol. xiv. p. 85.

⁴ B. I. Stokvis. Ueber Hemialbumosurie, *Nederl. Tijdschrift voor Geneesk.*, 1891, vol. ii. p. 26, cit. in *Maly's Jahrb.*, 1892, vol. xxi. p. 412. H. C. G. Ribbink, Een geval v. Albumosurie, *Diss. Amsterdam*, 1892, cit. in *Maly's Jahrb.*, 1893, vol. xxii. p. 525. H. Zeehuisen, *Nederl. Tijdschr. v. Geneesk.*, 1898, Pt. I. p. 829, cit. in *Maly's Jahrb.*, 1894, vol. xxiii. p. 577.

of the diseased tissues. The anatomical diagnosis was osteosarcomatosis (myeloma).

The *fifth case* occurred in Stintzing's clinic in 1895. The clinical history, together with an account of some experiments regarding the general metabolism of the patient, is reported by Seegelman.¹ The study of the urine was made by Matthes in Neumeister's laboratory. Post-mortem examination revealed the existence of multiple myeloma.

The *sixth case* occurred in the clinic of Senator,² who has reported the history of the patient in detail. A general account of the patient's urine is given by Rosin, while a more detailed study appears in Süßmann's thesis of 1897 (Leipzig). Post-mortem myelogenous round-celled sarcomatosis (myeloma) of the ribs was found.

The *seventh case* is reported by Bozzolo³ under the interesting title "Sulla malattia di Kahler." The urine was examined by Belfanti. An autopsy could not be made, but it is quite clear from the detailed clinical account which is given that this patient also was the subject of multiple myeloma.

The *eighth case* is reported by Ewald.⁴ During the life of the patient the diagnosis of myeloma was made upon the basis of a histological examination of a growth of the right clavicle which was removed at an operation.

The *ninth case* occurred in Lichtheim's clinic, and is reported by Ellinger.⁵ The anatomical diagnosis was multiple lymphoma (myeloma) and lymphoid infiltration of the bone-marrow of the ribs, the vertebrae, and sternum.

The *tenth case* was briefly reported by Naunyn,⁶ in 1898, at a meeting of the "Unter-Elsässischer Aerzteverein," in Strassburg (January 29, 1898). He expressed the opinion that in this case, also, multiple myeloma of the bones of the thoracic skeleton existed, but an autopsy was not made. The urine was studied in detail by Magnus-Levy.

¹ Seegelman. Ueber multiples Myelom u. Stoffwechseluntersuchungen b. demselben, Deut. Arch. f. klin. Med., 1897, vol. lviii. p. 276. M. Matthes, Ueber Eiweisskörper im Urine b. Osteomalacie, Verhandl. des XIV. Cong. f. inn. Med., 1896, p. 476. R. Neumeister, Lehrbuch d. physiol. Chem., 1897, 2d ed., p. 806.

² H. Senator. Asthenische Lähmung, Albumosurie u. multiple Myelome, Berl. klin. Woch., 1899, vol. xxxvi. p. 161. H. Rosin, Ueber einen eigenartigen Eiweisskörper im Harn, u. seine diagnostische Bedeutung, Berl. klin. Woch., 1897, vol. xxiv. p. 1044.

³ Bozzolo. Sulla malattia di Kahler, La Clinica medica Italiana, 1898, cit. in Centralbl. f. d. med. Wiss., 1898, vol. xxxvi. p. 572. The case was first reported in 1897 before the Eighth Congress of International Medicine.

⁴ K. Ewald. Ein chirurgisch interessanter Fall v. Myelom., Wien. klin. Woch., 1897, vol. x. p. 169.

⁵ A. Ellinger. Das Vorkommen des Bence Jones'schen Körpers im Harn bei Tumoren d. Knochenmarkes u. seine diagnostische Bedeutung, Diss. Königsberg, 1898, and Deutsch. Arch. f. Klin. Med., 1899, vol. lxi. p. 255.

⁶ Naunyn. Ein Fall v. Albumosurie, Deutsch. med. Woch., 1898; Vereins Beilage, No. 30, p. 217. Magnus-Levy, Ueber d. Bence Jones'schen Eiweisskörper, Zeitschr. f. physiol. Chem., 1900, vol. xxx. p. 200.

The *eleventh case* was briefly reported by Bradshaw¹ before the Royal Medical and Chirurgical Society, April 26, 1898. A further report appeared in 1900. No record of an autopsy is given, but Bradshaw regards the case as an unmistakable instance of bone disease and probably of myelomata.

The *twelfth case* is reported by Fitz.² The clinical diagnosis was myxœdema, and there was no clinical evidence of bone disease. The patient died while under treatment with thyroid extract. Unfortunately, an autopsy could not be held. The urine was examined by Dr. E. S. Wood.

The *thirteenth case* also was under the care of Dr. Fitz,³ and is briefly referred to in his previous paper, where he speaks of the patient as being then under the charge of Dr. F. C. Shattuck. An account of the clinical history of the case and a study of the post-mortem findings is given by Wright. The urine in this case also was examined by Dr. E. S. Wood, and through the latter's courtesy I was enabled to study the isolated substance. (See above.)

The *fourteenth case* is reported by Vladimir de Holstein,⁴ in 1898. In this case the diagnosis "multiple myelomatosis" was made during the life of the patient on the basis of the existing "albumosuria." In a subsequent note it is stated that the clinical diagnosis was confirmed by autopsy. A detailed examination of the urine is wanting.

The *fifteenth case* is reported by Buchstab and Schaposchnikoff.⁵ The original diagnosis had been pseudoleukæmia, but after Bence Jones' "albumose" was found in the urine it was changed to myeloma. This was confirmed by autopsy.

The *sixteenth case* occurred in the clinic of Lichtheim, and was reported by Askanazy⁶ before the Verein f. Wissenschaftliche Heilkunde, in Königsberg (Prussia), at the meeting of January 23, 1899. A more detailed account of the case appeared in 1900. Post-mortem examination showed the existence of lymphatic leukæmia, with the corresponding characteristic changes in the bone-marrow.

¹ T. R. Bradshaw. A Case of Albumosuria, Brit. Med. Journ., 1898, Pt. I. p. 1136. Myelopathic Albumosuria, *ibid.*, November 3, 1900.

² R. H. Fitz. The Significance of Albumosuria in Medical Practice, THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1898, vol. cxvi. p. 80.

³ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, *ibid.* p. 43. J. H. Wright, A Case of Multiple Myeloma, Journ. of the Boston Soc. of Med. Sci., 1900, vol. iv. p. 195, and Contributions to the Science of Medicine, W. H. Welch Memorial Vol., Johns Hopkins Press, 1900, p. 359.

⁴ Vladimir de Holstein. Semaine Médicale, 1898, p. 206, and *ibid.*, 1899, p. 83.

⁵ L. Buchstab and B. Schaposchnikoff. Ein Fall von diffusum Myeloma d. Knochen in Zusammenhange mit einer typischen Albumosurie als einem charakteristischen diagnostischen Merkmale. Russ., Arch. f. Pathol., klin. Med. und Bacteriol., 1899, vol. vii. p. 11, Russian.

⁶ S. Askanazy. Ueber die diagnostische Bedeutung d. Bence Jones'schen Albumosurie, Deutsch. med. Woch., 1899, vol. xxv. V. B. p. 177, and Deutsch. Arch. f. klin. Med., 1900, vol. lxxviii. p. 24.

The *seventeenth case* is briefly described by Sternberg,¹ in 1899. An autopsy was not made. In the urine large amounts of the Bence Jones substance were found (examination by E. Freund). An account of the reactions of the body, however, is not given.

The *eighteenth case* is the one under present consideration. A preliminary report was made by Dr. Hamburger² before the meeting of the Johns Hopkins Medical Society on November 5, 1900. The clinical diagnosis, as I have said, was multiple myeloma, but an autopsy, unfortunately, could not be made.

The *nineteenth case* occurred in the service of Dr. Osler, and is likewise reported by Hamburger.³ Autopsy revealed myelomata in the skull, left scapula, both clavicles, the sternum, the right ilium, and neck of the right femur. A detailed report of the post-mortem findings is given by MacCallum.⁴

The *twentieth case* is reported by Kalischer.⁵ Post-mortem examination revealed lymphoid hyperplasia of the marrow of the ribs (myeloma). (Examination by Dr. Franz Müller.)

The *twenty-first case* is briefly referred to by Rostoksi.⁶ An account of the clinical history or of a post-mortem examination is not given.

The *twenty-second case*, finally, is reported by Jochmann and Schumm.⁷ The writers make no mention of any of the cases that have been reported since 1897, with the exception of a supplementary note on the preceding case. They claim that their case is the first true instance of osteomalacia and not of multiple myeloma, in which the Bence Jones substance was found in the urine. Autopsy showed involvement of the vertebrae, the ribs, sternum, pelvic bones, and long bones. In addition, interstitial and parenchymatous nephritis was noted, with slight amyloid degeneration.

The above twenty-two cases are the only ones recorded in the literature in which the presence in the urine of the Bence Jones substance has been definitely established. It will be noted that, barring those in which no autopsy was made, and assuming, with Kahler, that Bence Jones' case was probably myeloma and not osteomalacia, myeloma actually existed in thirteen. The relation between the two conditions in so large a percentage of cases suggested the possibility that the substance may possibly have been present in still other cases of myeloma

¹ M. Sternberg. Vegetationsstörungen und Systemerkrankungen der Knochen, Nothnagel's Pathol. und Therap., 1899, vol. vii., pt. 2, sec. 2, p. 57.

² L. P. Hamburger. Loc. cit.

³ Loc. cit.

⁴ W. G. MacCallum. A Case of Multiple Myeloma, Journ. Exper. Med., 1901, vol. vi. p. 53.

⁵ S. Kalischer. Ein Fall v. Ausscheidung d. Bence Jones'schen Eiweißkörper durch d. Urin bei Rippenmyelomen, Deutsch. med. Woch., 1901, vol. xxvii. p. 54.

⁶ Rostoksi. Albumosurie und Peptonurie, Münch. med. Woch., 1901, vol. xlviii. p. 1115. Proceedings of the Physikalisch-medizinische Gesellschaft v. Würzburg, meeting of June 13, 1901.

⁷ G. Jochmann and O. Schumm. Typische Albumosurie bei Osteomalacie, Münch. Med. Woch., 1901, vol. xlviii. p. 1340.

also, but may not have been recognized as such. Unfortunately, the term myeloma is not applied to a definite clinical picture, nor even to a uniform pathological-histological condition, so that I was much embarrassed in my search of the literature in deciding what cases of apparent malignant disease of the bones belonged to this category. I was greatly aided, however, by the data given in Wieland's¹ excellent paper, although the writer's interpretation of some of our cases in which the Bence Jones substance was found in the urine is at variance with the diagnosis originally made. The cases which Wieland accepts as cases of true myeloma, in addition to the thirteen already referred to, are those of v. Rustitzky,² Zahn,³ and Klebs,⁴ even more probably that of Weber,⁵ possibly the case of Marchand,⁶ and perhaps also those of Winkler⁷ and Funkenstein.⁸ Markwald's⁹ case, which was formerly regarded as an instance of myeloma, is now classified as endothelioma. I have gone over the original report of the case, together with the true myeloma cases, however, notwithstanding, as it was very similar to that of Kahler,¹⁰ in which the Bence Jones substance was actually found. My search, however, has not led to the discovery of a new, well-defined case of Bence Jones' "albumosuria." Zahn, it is true, reports that in his case albumin was always present in variable amounts, and post mortem no definite lesion of the kidneys was found. A more detailed account of the reactions, however, is lacking. v. Rustitzky, Weber, Winkler, and Markwald remark that in their cases no albumin was present in the urine, and v. Jaksch¹¹ further states that in one case of myeloma which he observed the Bence Jones albumin could at no time be demonstrated. Marchand makes no note of a urinary examination whatever. As a result we are forced to the conclusion that while the Bence Jones albumin may be present in association with myeloma, this is not necessarily the case. There is some evidence to show, moreover, that the substance may occur in other diseases affecting the bone-marrow as well. I have thus pointed out that in Jochmann and Schumm's case post-mortem examination revealed the existence of osteomalacia, and in Askanazy's case of lymphatic leukæmia (lymphadenoid degeneration

¹ E. Wieland. Studien über das primär multiple auftretende Lymphosarcom der Knochen. Virchow's Arch., 1901, vol. clix. p. 108.

² v. Rustitzky. Multiples Myelom, Deutsch. Zeitsch. f. Chirurg., 1873, vol. iii.

³ F. W. Zahn. Ueber das multiple Myelom, seine Stellung im onkologischen System u. seine Beziehung zur Anæmia lymphatica, Deutsch. Arch. f. Chirurg., 1885, vol. xxii.

⁴ Klebs. Die allgem. Pathol., 1889, Pt. 2.

⁵ Weber. General Lymphadenomatosis of Bones, One Form of "Multiple Myeloma," Journ. of Pathol. and Bacter., 1898, p. 59.

⁶ Marchand. Ein Fall von seniler Osteomalacie, Berlin. klin. Woch., 1889, No. 29, p. 486.

⁷ Winkler. Das Myelom, in anatom. u. klin. Beziehung, Virchow's Arch., 1900, vol. cxi. p. 252.

⁸ O. Funkenstein. Ein Fall von multiplem Myelom, Inaug. Diss., Strasburg, 1900.

⁹ Markwald. Ein Fall von multiplem, intervaskulærem Endotheliom in d. gesammten Knochen d. Skelets, Virchow's Arch., 1895, vol. cxli. p. 128.

¹⁰ Loc. cit.

¹¹ v. Jaksch. Verhandl. d. 18th Cong. f. inn. Med., 1900, p. 500.

of the bone-marrow). A detailed report of the post-mortem findings in Jochmann's case has not as yet appeared; but if future investigations should prove that their case was actually one of osteomalacia, the observation would gain in interest, as other observers have heretofore not found the substance in the disease in question, although diligent search has been made. Bence Jones himself thus writes to Kühne that he examined the urine of a fairly large number of well-marked cases (from 1845 to 1869), but that he could not find the substance again.

Stokvis, also, has not a single instance to report where Bence Jones' "albumosuria" was associated with osteomalacia, and v. Jaksch¹ states that he never found "albumose" in the urine in numerous cases of the disease. The only other observations which I have been able to find in the literature in which it is claimed that "albumoses" were present in the disease in question are the cases of Langendorff-Mommsen² and of Raschkes.³ In neither case, however, is there sufficient evidence to show that the urine actually contained the Bence Jones substance, although the reactions described by Raschkes suggest it rather more strongly than in the first case.

In association with other multiple new-growths affecting the bones, excepting myeloma, the substance in question has apparently not been found, unless, indeed, some of the cases which have been reported under the title "myeloma" are, as Wieland and others suggest, not cases of true myeloma. Into this question, however, I can scarcely enter at this place. Suffice it to say that an analysis of the reported cases of multiple tumors affecting the bones, which I have made with a view of possibly discovering some overlooked instance of "albumosuria," led to no result. Runeberg,⁴ it is true, reports that in his case the urine contained *much* albumin, and at the autopsy it was noted that in the spongy portion of the sternum, the ribs, the vertebrae, and the sacrum there was a marked hyperplasia of granular, light yellow, nucleated cells, as also a large number of "giant cells." Runeberg states that the urine contained numerous hyaline and finely granular casts, and adds that the finding of the latter alone speaks against a true inflammatory disease of the kidneys. The existence of cirrhotic kidneys, moreover, was excluded, and at the post-mortem it could be definitely shown that a nephritis did not exist. From the account given the possibility thus suggests itself that in this case the "albu-

¹ R. v. Jaksch. *Klinische Diagnostik*, 1896, Wien u. Leipzig.

² O. Langendorff and J. Mommsen. *Beiträge zur Kenntniss d. Osteomalacie*, Virchow's Arch., 1877, vol. xlix. p. 649.

³ A. Raschkes. *Ein Fall von seniler Osteomalacie mit Albumosurie*, Prag. med. Woch., 1894, vol. xix. p. 649.

⁴ J. W. Runeberg. *Ein Fall von medullärer Pseudoleukämie*, Deutsch. Arch., 1883, vol. xxxiii. p. 629.

mose" may have been present, but a definite conclusion can scarcely be reached in the absence of a more detailed examination.

The negative cases, in regard to which I consulted the original histories, are those reported by Lannelongue,¹ Arnold,² Litten,³ Grawitz,⁴ Waldstein,⁵ Hammer,⁶ Wieland,⁷ Kudrewetzky,⁸ and Nothnagel.⁹

In cases of multiple metastatic carcinoma involving the bones "albumosuria" never occurs.

The only instance of bone disease other than what has been described above, in which a substance has been encountered in the urine of man, which may be interpreted as being identical with the body under consideration, is a case of tubercular osteo-arthritis of the shoulder that has been briefly reported by Vidal¹⁰ before the Société de Biologie of Paris (October 29, 1898). The patient died of acute pleuro-peritoneal tuberculosis, but an autopsy was apparently not held. The reactions which were obtained from the patient's urine, and from which Vidal himself concludes that the substance must have belonged to the "primary proteoses," are the following: After the addition of a few drops of acetic acid no precipitate results on boiling. A well-marked biuret reaction is obtained. Cold nitric acid produces a milky turbidity, which disappears on boiling and reappears on cooling.¹¹

It is stated by Laache¹² and Lussana¹³ that in pernicious anæmia "peptonuria," in the older meaning of the term, is of common occurrence; but I have not been able to find any data in the literature to warrant the conclusion that the "peptone" in such cases is identical with the Bence Jones albumin. The statement, however, is of interest, as it is a well-known fact that myeloma may follow a clinical course which is quite similar to the common form of pernicious anæmia. A

¹ Lannelongue. *Gaz. des Hôpît.*, Nos. 41 and 42.

² Arnold. Drei Fälle v. primärem Sarcom d. Schädels, *Virchow's Arch.*, 1873, vol. lvii. p. 297.

³ Litten. Ueber einen in medulläre Leukämie übergehenden Fall v. primärer Anämie. *Berlin. klin. Woch.*, 1877, Nos. 19 and 20.

⁴ P. Grawitz. Maligne Osteomyelitis u. sarcomatöse Erkrankungen d. Knochensystems als Befunde b. Fällen v. perniciöser Anämie, *Virchow's Arch.*, 1879, vol. lxxvii. p. 253.

⁵ L. Waldstein. Ein Fall von progressiver Anämie u. darauffolgender Leucocythämie mit Knochenmarkerkrankungen u. einem sogenannten Chlorom (Chlorolymphom), *Virchow's Arch.*, 1883, vol. xel. p. 12.

⁶ Hammer. Primäre sarcomatöse Ostitis mit chronischem Rückfallsieber, *Virchow's Arch.*, 1894, vol. cxxxvii. p. 280.

⁷ Wieland. *Loc. cit.*

⁸ B. Kudrewetzky. Zur Lehre v. d. durch Wirbelskülen Tumoren bedingten Compressions, Erkrankung d. Rückenmarks, *Zeitsch. f. Heilkunde*, 1892, vol. xiii.

⁹ H. Nothnagel. Ueber eine eigenthümliche perniciösen kockenerkrankung. (Lymphadenia osium), *Virchow's Festschrift*, 1891, vol. ii. p. 155.

¹⁰ E. Vidal. Note sur un cas d'albumosurie, *Compt. rend. de la Soc. de biol.*, 1898, vol. l., p. 991.

¹¹ I find that another case is briefly reported in which the Bence Jones substance was apparently present in the urine. No details, however are given. Matthes, *Verhandl. d. Congr. f. inner Med.*, 1900, p. 501.

¹² Laache. *Die Anämie*. Christiania, 1883.

¹³ Lussana. *Cit. by Eichhorst, Perniciöse Anaemie, Eulenburg's Real-Encycl.*, 3d ed.

more careful search in this particular direction than I have been able to make may possibly unearth still other cases of Bence Jones' albuminuria.

From the reported cases of Bence Jones' albuminuria the conclusion may be drawn: 1. That the presence of the substance in the urine of man usually indicates the existence of multiple new-growths affecting the bones, and generally of myeloma. 2. That myeloma may apparently occur in the absence of the peculiar albuminous substance. 3. That the existence of the urinary condition in association with other diseases without involvement of the bones is not as yet proved by the report of any case in which a careful post-mortem examination has been made. 4. That malignant disease of the bones, in general, may exist in the absence of the Bence Jones albumin from the urine.

Outside of the urine, and possibly the blood and the tumor masses of the cases described in the foregoing pages, the Bence Jones albumin has apparently also been encountered in the urine of dogs during the continued administration of pyrocin (monoacetyl-phenylhydrazin). Zülzer¹ states that the "albumosuria" is practically a constant result of pyrocin poisoning in dogs, but that it only persists in its pure form for a very short time, and is soon obscured by the common form of albuminuria, which develops as the result of the inevitable nephritis. He cites one instance, however, in which the albumosuria persisted for four days, uncomplicated by albuminuria. With nitric acid a heavy precipitate occurred at ordinary temperatures, which dissolved entirely on heating. Similar results were obtained with sulpho-salicylic acid, picric acid, Almen's reagent, and acetic acid and potassium ferrocyanide. On heating the urine, after acidifying distinctly with acetic acid, a marked turbidity occurred between 50° C. and 60° C., which disappeared almost entirely on heating to 100° C. The isolated substance (precipitation with alcohol) gave the same reactions as the native urine. After drying it was soluble only in boiling water after the addition of a little soda. Millon's reaction, the biuret reaction, and the sulphur test were positive. On half saturation with salt a marked turbidity occurred (1).

A detailed account of the experiments is still lacking, but I conclude from Zülzer's paper that the "albumosuria" was associated with changes in the bones.

In Tallquist's² monograph on pyrocin-anæmia no mention is made of the occurrence of "albumosuria," but it is possible, as Zülzer suggests, that it has been overlooked, owing to the simultaneous presence of common albumin.

¹ G. Zülzer. Ueber experimentelle Bence Jones'sche Albumosurie, Berlin. klin. Woch., 1900, vol. xxxvii, p. 894.

² T. W. Tallquist. Ueber experimentelle Blutgift-Anæmien. Berlin, 1900. A. Hirschwald.

Whether or not the Bence Jones substance may also occur in normal bone-marrow, as is suggested by the experiments of Fleischer,¹ remains to be decided by future researches according to more modern methods. If so, it is apparently only present in traces. Larger amounts of a similar but apparently not identical substance were found by Virchow² in the bone-marrow of a patient dead with osteomalacia; and it has been stated by some that this was in reality the Bence Jones albumin. Virchow, however, has made no statement to this effect; and Kühne in speaking of this observation states that it merely shows "that common albumin after solution in acetic acid by boiling can give rise to a precipitate, on treating with nitric acid, which dissolves on heating and reappears on cooling." Fleischer's substance differs from that of Virchow in the fact that the precipitate caused by nitric acid dissolved upon the further addition of concentrated acetic acid.

COMPARISON OF RESULTS. Amount. On comparing my results with those obtained by others, and these in turn with each other, it is clear, I think, that all observers have been dealing with one and the same substance. They all mention that on treating the urine with nitric acid a precipitate results, which disappears on boiling and reappears on cooling. This reaction, it is true, is not characteristic of the Bence Jones substance, as it is also obtained with the deutero-albumoses which may occur in the urine under various other pathological conditions. But it should be borne in mind that whereas deutero-albumoses are usually only present in such small amounts that special methods are necessary for their demonstration, the Bence Jones substance generally occurs in quantities which are so large as to arrest attention at once. In the following table I have collected the results which have been obtained by the various observers:

| | | |
|-----------------------|--------|-----------------|
| Fitz | 0.12 — | 0.98 pro mille. |
| Ellinger | 0.25 — | 0.5 " " |
| Askanazy | 0.5 — | 1.25 " " |
| Fitz | 0.1 — | 2.25 " " |
| Huppert | 3.0 | " " |
| Fitz-Wright | 3.3 — | 3.4 " " |
| Kallscher, | 5.0 — | 5.0 " " |
| Stegmann | 6.0 | " " |
| Hamburger, I. | 6.0 | " " |
| Matthes | 4.0 — | 6.0 " " |
| Simon | 0.12 — | 5.12 " " |
| Boszolo | 10.0 | " " |
| Bradshaw | 10.0 | " " |
| Ribbink | 20.0 | " " |
| Magnus-Levy | 18.0 — | 24.0 " " |
| Bence Jones | 67.0 | " " |
| Noël Paton | 70.0 | " " |

¹ R. Fleischer. Ueber das Vorkommen d. sogenannten Bence Jones'schen Eiweisskörper's im normalen Knochenmark, Virchow's Arch., 1880, vol. lxxx. p. 482.

² R. Virchow. Ueber parenchymatöse Entzündungen, Virchow's Arch., vol. iv. p. 308.

From this table it will be seen that in some cases the elimination of the substance far exceeds the largest amounts of common albumin that may be encountered in the urine.

Behavior on Heating. All observers agree that the albuminous substance in question begins to be precipitated from the urine at a temperature which is well below that at which serum albumin is coagulated, viz., between 50° C. and 58° C. Individual variations are undoubtedly referable to a varying degree of acidity and the amount of salt present, and may indeed occur in one and the same case. Kühne's is the only case in which the initial temperature of precipitation was found below 50° C., viz., at 43° C. Naunyn states that his substance was precipitated between 49° C. and 53° C. As regards the behavior of the urine on further heating, the statements of the various observers differ more radically. Kühne and Matthes thus find that in their cases the urine cleared entirely on boiling, and it appears that this also occurred in Ellinger's case, as the faint turbidity which remained at 100° C. was here no doubt referable to the contaminating nucleo-albumin. In Ribbink's case and those of Huppert, Rosin, Magnus-Levy, Rostovsky, and myself, on the other hand, the urine did not clear on boiling. This apparent difference might suggest that the substance which the various investigators had under observation was not the same in all cases. Such a conclusion, however, is not admissible, in view of the fact that all possible variations in the solubility of the precipitate at 100° C. may be observed not only in the different cases, but even in one and the same instance. Naunyn thus reports that the precipitate which appeared between 49° C. and 53° C. cleared almost entirely on subsequent heating to boiling. Magnus-Levy, who studied the urine from the same patient later on, writes that at first a large portion of the precipitate dissolved on boiling, but that a considerable fraction remained undissolved; and still later in the course of the disease it was found that even with a neutral reaction the substance could be quantitatively precipitated by heat and remained insoluble on boiling. In my case, also, it was found that at first the urine became "clear" on boiling after previous precipitation; while later, when I had occasion to study the urine, a clearing at 100° C. could scarcely be noted with the eye. I have shown, moreover, that in my case, as in Magnus-Levy's, it was possible to *completely* precipitate the substance by heat if the elevation of temperature was carried out slowly, providing that a sufficient amount of salt was present and the urine presented an acid reaction. In such an event no solution whatever occurred on boiling. But, aside from these observations, the identity of the various substances is proved by the fact that, as Magnus-Levy first pointed out, it is possible to render the body soluble at 100° C., or insoluble, at will, by simple measures which in themselves can hardly be supposed to

change the chemical nature of the substance. A solution of the isolated substance, moreover, which has been rendered acid by acetic acid, and which contains a certain amount of salt, shows the "typical" behavior of the Bence Jones substance on heating as it was described by Kühne. Its insolubility at 100° C. in the urine must hence depend upon the associated presence of some other substance or substances in the urine, among which the salts, no doubt, are of prime but not of exclusive importance. What other factors are here of moment, however, is as yet unknown. Remembering the influence of the degree of acidity upon the phenomenon, it might be supposed that the relative amount of acid phosphates would enter into consideration, but this does not seem to be the case. Using two different mixtures of monopotassium phosphate and disodium phosphate in solution, of which the one was strongly alkaline, while the other was markedly acid, and adding varying amounts of these solutions to a given amount of a solution of the Bence Jones substance, Magnus-Levy was unable to obtain any effect whatever upon the coagulability of the body. He showed, however, that the addition of ammonium chloride in a definite proportion ($\frac{1}{3}$ per cent.) renders the substance more readily soluble at 100° C. after it has been previously coagulated, and that the addition of a certain amount of urea to the solution or to the urine may prevent heat coagulation altogether. These experiments I have repeated, and I have been able to confirm Magnus-Levy's results. I do not think, however, that the differing behavior of the urine, as noted by different observers, can be explained upon the basis of these observations alone. Other factors are here, no doubt, active, of the nature of which we are as yet in ignorance; and to the activity of such factors I have no doubt some other and minor variations in the behavior of the body that have been noted by different observers may be due. For such reasons, also, differing reactions may be obtained in one and the same case at different times. So much is certain, however, that it is no longer admissible to speak of the solubility of the coagulated substance at 100° C. as a characteristic and typical reaction, *so far as the urine is concerned. It is characteristic, however, of a solution of the isolated substance in the presence of a certain amount of acid and of salt. As the most characteristic general reaction, I should regard the coagulability of the substance at a temperature of from 50° C. to 58° C.¹*

In the above pages I have repeatedly made use of the term *coagulation* in reference to the precipitation of the Bence Jones substance by

¹ Whether or not serum albumin was actually present in the urine in association with the Bence Jones substance as often as stated by individual observers I am not prepared to decide. I should suggest, however, that in the future a special examination in this direction be made. To conclude that the common albumins are present because the urine does not clear entirely on boiling is unwarrantable.

heat or on treating with alcohol. Strictly speaking, this term is not applicable, however, as it implies a material alteration of the chemical properties of the substance, which in reality has not been effected. It is true that such precipitates are practically insoluble in cold distilled water, but they dissolve with comparative ease in boiling water in the presence of a small amount of sodium carbonate, and, as Magnus-Levy has first pointed out, the alcohol precipitate is readily soluble in very dilute solutions of ammonia. On subsequent neutralization a solution is then obtained which behaves, on heating, as the original solution, if ammonium chloride is present to the extent of $\frac{1}{2}$ per cent. The latter observation is especially important, as the common albumins which may occur in the urine are insoluble in dilute ammonia after having been precipitated by alcohol. The coagulate, moreover, is readily soluble in dilute acids, and from such solutions the substance is again thrown down upon the application of heat, providing that a certain amount of salt is present.

Dialysis. As regards the behavior of the substance on dialysis, all observers who have examined in this direction agree that it does not pass through parchment, with the exception of Ribbink, who states that a portion of his substance escaped in this manner. It is quite possible, however, that Ribbink's parchment was not altogether dense. Important in this connection is the fact that the substance is not precipitated from its solutions by dialysis, no matter how prolonged this may be.

Behavior on Salting with Sodium Chloride. Apparent differences are recorded in the behavior of the substance on salting with sodium chloride. Huppert reports that in his case the substance was completely precipitated from the urine by salting to saturation between 35° C. and 40° C. Ribbink, on the other hand, reports that even after standing for several days he was unable to obtain a precipitate by salting with rock salt at any temperature between 15° C. and 40° C. Matthes states that in his case only a fraction of the substance was precipitated by salting the urine to saturation, and then only on standing for several days. Süssmann and Magnus-Levy found that in their cases the substance could be partially precipitated from the acid urine in this manner, and Ellinger writes that on the addition of a saturated solution of sodium chloride he obtained a precipitate even at ordinary temperatures. Kühne, under the latter conditions, merely noted a turbidity. In my case no turbidity whatever occurred on treating the acid urine with a saturated solution of common salt, even when added in the proportion of 3 to 1. With an alkaline urine, however, that had undergone ammoniacal decomposition I found that a precipitate formed upon the addition of 0.4 c.c. of a saturated solution of salt to 2 c.c. of the urine. With rock salt partial precipitation occurred at ordinary tem-

peratures, while complete separation occurred on salting at from 35° C. to 40° C.

With a neutral solution of the isolated substance I very curiously found that partial precipitation occurred on salting with sodium chloride to saturation, whereas Magnus-Levy and Süssmann state that a neutral and purified solution of the body cannot be precipitated by salting with sodium chloride at ordinary temperatures. Magnus-Levy, however, has manifestly overlooked the fact that Kühne also found that his substance could be precipitated with sodium chloride from its neutral solutions. In his second communication he states distinctly that a solution of the isolated substance gave a copious precipitate upon the addition of rock salt prisms in acid, as well as in neutral and feebly alkaline solutions. The precipitate, moreover, was soluble in water.¹

These data in themselves suffice to show that the question, whether or not the Bence Jones substance can be precipitated on salting with sodium chloride, depends not only upon the reaction of the solution, but upon other factors as well, which are as yet but little understood.

The observation of Huppert, who succeeded in completely precipitating his substance from the urine by salting to saturation at a temperature between 35° C. and 40° C., may be explained by the assumption that this precipitation is not a true salt precipitation at all, but an actual coagulation, which, owing to the presence of so large an amount of salt occurs at a much lower temperature. I have accordingly also found that the precipitate, which I obtained on salting with rock salt to saturation at a temperature of about 37° C., was insoluble in water.

Limits of Precipitation with Ammonium Sulphate. The differences in the limits of precipitation with ammonium sulphate which exist between Magnus-Levy's case, Ellinger's, Fitz's No. 2, and my own are possibly owing to differences in the amount of salts which were originally present in the urine, to varying strengths of the albuminous solutions, and like factors. On this basis, at any rate, it would scarcely be warrantable to assume that each of us was dealing with a different substance.

| | |
|--|-------------|
| Ellinger (solution of isolated substance, previously coagulated and dissolved in a dilute solution of sodium carbonate) | 2.0 and 4.0 |
| Magnus-Levy (similar preparation, but subsequently neutralized) | 0.6 " 2.2 |
| Magnus-Levy (similar preparation, but dissolved in ammonia) | 2.5 " 6.0 |
| Magnus-Levy (neutralized urine). | 4.0 " 6.0 |
| Simon (neutralized urine) | 0.7 " 4.2 |
| Simon (solution of isolated substance, precipitated with ammonium sulphate and dialysed for eight weeks, neutral reaction) | 2.3 " 3.8 |
| Fitz-Wright [precipitation with alcohol, solution in very dilute ammonia (Simon)] | 1.7 " 3.6 |

Formation of "Albumosate." As regards the behavior of a solution of the substance on neutralization, after the previous addition of an acid

¹ Kühne and Chittenden. Loc. cit., vol. xx.

or an alkali, the reports of all observers who have examined in this direction, with the exception of Ribbink, are the same. Ribbink states that he could not obtain a precipitate on neutralization, but it is quite likely, as Magnus-Levy suggests, that he had previously not added enough acid or alkali. Kühne, who regarded the Bence Jones substance as an albumose, speaks of the apparent change which results on treating with an alkali, as due to the formation of an albumosate analogous to the albuminate in the case of the common albumins.

Behavior on Salting with Magnesium Sulphate. In its behavior toward magnesium sulphate a marked difference apparently exists between my substance and that of Magnus Levy. For, whereas he reports that on saturating the urine with the salt in question, at the temperature of the room, no precipitation occurred, I noted in my case that after acidifying with acetic acid the substance is promptly thrown down. My notes on this point read as follows: Upon the addition of two volumes and a half of a saturated solution of magnesium sulphate to the urine no precipitation occurred, even after acidifying with acetic acid, but on saturation of the acid solution with the salt in substance, complete precipitation occurred. Saturation without the addition of acetic acid gave no result in the native urine. But on treating the neutral solution of the isolated substance (precipitation with ammonium sulphate and subsequent dialysis for eight weeks) with two or three times its volume of a saturated solution of magnesium sulphate, and then saturating with the salt in substance, the Bence Jones body was completely precipitated on standing for twenty-four hours, at the temperature of the room. The filtrate remained clear on heating after the addition of acetic acid; no further precipitate occurred on treating with twice the volume of a saturated solution of ammonium sulphate, and the biuret reaction was negative. The precipitate itself was soluble in water and could hence not be a coagulate, but must be regarded as a true salt-precipitate. Other observers have made no notes of the behavior of the substance in this respect; but it is interesting to note that the substance which Dr. Wood sent me (from Fitz-Wright case) could also be precipitated with magnesium sulphate from its neutral aqueous solution.

Behavior on Digestion. On digestion with pepsin-hydrochloric acid all observers who have made investigations in this direction report that the substance dissolves very readily, and that the presence of "peptones" can very soon be demonstrated. Matthes noted that on prolonged digestion a jelly-like material separated out, which, after drying, was soluble in absolute alcohol. This, however, as he later admitted himself, and as Neumeister indicates, was owing to the admixture of a renal excretion. Ellinger records that on digestion with pepsin for twenty-four hours a small amount of residue was obtained, which con-

tained phosphorus. In his case, however, a nucleo-albumin was also present and had not been previously removed. Speaking of the products of digestion with pepsin-hydrochloric acid, Kühne states that after one or two hours only "peptone" was found in solution, and no precipitate was obtained either with nitric acid or with acetic acid and sodium chloride.

A more detailed examination of the resulting products of digestion according to more modern methods has been made by Magnus-Levy. As I have previously indicated, he claims to have obtained a proto-albumose, three deuto-albumoses, and two peptones, while the formation of a hetero-albumose was not observed. In view of the importance of these results in their bearing on the question of the chemical nature of the Bence Jones substance, it may not be out of place to consider in some detail the basis on which Magnus-Levy has established his claim that on peptic digestion the Bence Jones substance yields a proto-albumose. Magnus-Levy states that he employed Hofmeister's method of analysis, as described by Pick,¹ Umber,² and Alexander.³ According to this method the digestive mixture was neutralized (with ammonia or ammonium carbonate), brought to the boiling-point, and then concentrated on the water-bath to about one-fifth of the original volume. On neutralization Magnus-Levy states that he never obtained a precipitate, but that during the process of concentration flocculi separated out, which may have been due to acid albumin, but were present in too small a quantity for analytical purposes. On treating the filtered solution with an equal volume of a saturated solution of ammonium sulphate a precipitate then resulted, which Magnus-Levy regards as due to proto-albumose exclusively, as a hetero-albumose could not be obtained from this fraction, either on dialysis or on treating the aqueous solution of the precipitate with half and an equal volume of alcohol and boiling for several hours. He then further *identified* this fraction as proto-albumose by the following reactions, which he compares with those of the Bence Jones substance :

| | Sulphur test. | Millon's reaction. | Biuret reaction. | Molisch's test. | Adamkiewicz test. | Salting with rock salt. | Rock salt and acetic acid. | Dilute copper sulphate solution. |
|--------------------------|-----------------------|--------------------|------------------|--------------------------------|-------------------|-------------------------|--|----------------------------------|
| 1. Bence Jones substance | Markedly black color. | Intense. | Violet-red. | Faint but distinct. | Very pronounced. | No precipitation. | Complete precipitation. | Marked precipitation. |
| 2. Proto-albumose | Markedly black color. | Markedly red. | Purely red. | Much more marked than under 1. | Faint. | Precipitation. | Precipitation more marked than with rock salt. | Marked precipitation. |

¹ Loc. cit.² Loc. cit.³ F. Alexander. Zur Kenntniss d. Caseins u. seiner peptischen Spaltungsprodukte, Zeit. f. phys. Chem., 1898, vol. xxv. p. 411.

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The limits of the precipitation were 3.2 and 4.8 in one test and 2.8 and 4.8 in a second.

The question which of course suggests itself on reading Magnus-Levy's paper is the following: Is it not possible that the proto-albumose, so called, was in reality a portion of the Bence Jones substance which had escaped digestion? I must confess that Magnus-Levy's account of his work is not sufficiently detailed as to render such a supposition unwarrantable. He states that after two to three hours, at the latest, the original substance had entirely disappeared, and that it was then already possible to demonstrate the presence of all the various fractions which he claims to have obtained. He does not state, however, in what manner he showed that the substance was no longer present, supposing that the substance had not been digested entirely. However, in that case it would certainly have remained in solution on neutralization and boiling, as the amount of acid previously added is scarcely sufficient to insure the precipitation of the substance on subsequent neutralization; and from the neutral solution the body would not have been precipitated on boiling. Even on acidifying again I have noted that the solution remains clear or becomes at best but slightly opalescent.

On the other hand, it might be argued that the behavior of the isolated proto-albumose, as compared with the Bence Jones substance, on salting with ammonium sulphate, as also in its general reactions, as tabulated above, would warrant the identification of the "proto-albumose" as a distinct substance. This I am willing to admit to a certain extent; but I have considered it necessary, nevertheless, to attempt to prove even more conclusively that the Bence Jones substance actually yields a proto-albumose on peptic digestion. To this end the experiments already detailed were undertaken, and the results show conclusively that a proto-albumose is actually formed, and my own work thus entirely confirms that of Magnus-Levy. A hetero-albumose is not formed. This was already suggested by Magnus-Levy's observation that on hydrolytic decomposition with hydrochloric acid and sulphuric acid the Bence Jones substance does not yield glycocoll, but furnishes a relatively large amount of tyrosin, with but little leucin.

As regards the remaining products of peptic digestion which may be obtained from the Bence Jones substance, I also have found bodies corresponding to Pick's deutero-albumoses A, B, and C. A and C, however, and notably the latter, were manifestly only produced in very small amounts, while B was present in fairly large quantity. Peptone A also was certainly only present in traces, if at all, while I could demonstrate a considerable amount of peptone B. Magnus-Levy states that his deutero-albumose B showed a minimal Molisch reaction; I obtained none whatever. However, Magnus-Levy himself has placed a sign of interrogation behind his (minimal).

CHEMICAL NATURE OF THE BENCE JONES SUBSTANCE. Regarding the chemical nature of the Bence Jones substance, it is clear from the foregoing considerations that neither Magnus-Levy nor I regard the body as an albumose, and I note that Rostoski also has come to this conclusion, though he does not cite any experiments as basis for his belief.

Kühne regarded the insoluble (in neutral saline solution), coagulated substance as dysalbumose, and supposed this to have resulted from hetero-albumose, in which form the body must have been present in the urine in solution. In addition, he suggests that deuterio-albumose and proto-albumose may also have been present in traces, so that, according to Kühne, the Bence Jones substance represented a mixture of several albumoses. Huppert likewise regards his body as a hetero-albumose, especially as the substance is rendered insoluble by salting to saturation with sodium chloride at 35° C. to 40° C. The remaining observers, with the exception of Magnus-Levy, Rostoski, and myself, also regarded the substance as an albumose, while admitting, however, that it does not coincide in its reactions with any one of the known digestive albumoses. The essential points of difference are given by Matthes as follows:

1. If much salt is present the coagulated substance is not *entirely* soluble on boiling, while a precipitated albumose is soluble at 100° C. no matter how much salt may be present.

2. An aqueous solution of the substance does not pass through animal membrane, even in traces, while both proto-albumose and deuterio-albumose are dialyzable to some extent.

3. The substance in question after coagulation is still soluble on washing with distilled water, as also on dialysis, especially if this is continued until practically all salt has been removed. Hetero-albumose, on the other hand, is entirely insoluble in distilled water, and is precipitated on dialysis.

4. The substance differs from hetero-albumose in the fact that it is apparently coagulated by heat, in any concentration, in the presence of a small amount of salt and acid, while hetero-albumose only coagulates in very concentrated solutions.

In addition to these factors the following also can be urged against the albumose nature of the substance, and especially against the assumption that the body may be a hetero-albumose:

1. The fact that the solubility of the coagulated substance at 100° C. may be changed at will, and that under certain conditions the coagulate is altogether insoluble at 100° C., even though the reaction of the urine and its chemical composition have not been altered by artificial means.

2. The fact that after precipitation with alcohol or with salt and acid

at ordinary temperatures the substance rapidly becomes insoluble in distilled water and neutral saline solutions. In this respect hetero-albumose resembles the Bence Jones substance, but it is to be noted that this change cannot be effected by heat.

3. The apparent formation of syntonin and alkaline albuminate on treating with acids and alkalis. In this respect the substance resembles the true albumins, but the precipitates which result on neutralization are not altogether insoluble, as in the case of the common coagulable albumins.

4. The Bence Jones substance contains a carbohydrate group, which is lacking in the hetero-albumose—of fibrin, at least.

5. Hetero-albumose (of fibrin) contains 17.98 per cent. of nitrogen, of which 39 per cent. exists as diamino-nitrogen; whereas the Bence Jones substance contains 15.57 per cent. of nitrogen, of which 25 per cent. are referable to diamino-nitrogen.

6. On hydrolytic decomposition hetero-albumose yields glycocoll and but little tyrosin, while from the Bence Jones substance no glycocoll can be obtained, but relatively much tyrosin.

7. On peptic digestion the Bence Jones substance yields a proto-albumose, showing that its molecule must be more complex than that of the primary albumoses.

We are thus forced to the conclusion that the substance cannot be an albumose; but in attempting to classify it among the remaining groups of proteins we meet with further difficulties. The fact that many observers have found phosphorus in the mineral ash would at once suggest that the substance might be a nucleo-albumin, but opposed to this assumption is the statement of Magnus-Levy that by previously removing the phosphates from the urine with ammoniacal magnesia mixture he was able to obtain specimens which were entirely free from phosphorus. None of the previous observers, moreover, has succeeded in demonstrating the separation of a nuclein, on peptic digestion, if care had been taken to eliminate the presence of a contaminating nucleo-albumin. Matthes, who originally regarded his substance as a nucleo-albumose, later abandoned this idea, and concluded himself that the jelly-like material which separated out on prolonged peptic digestion was referable to the admixture of a nucleo-albumin with which the Bence Jones substance had nothing to do. In Dr. Wood's case, moreover, I also succeeded in finally obtaining a product which was free from phosphorus.

In the solubility of the alcohol precipitate in dilute ammonia the substance resembles the histons, but it differs from these in the fact that the body cannot be precipitated by ammonia from its acid solutions.

The solubility of the alcohol precipitate in dilute ammonia further distinguishes the substance from the true coagulable albumins which

have thus far been studied. Those, moreover, which ordinarily appear in the urine are totally insoluble at 100° C. unless the chemical composition of the urine is artificially altered, as by the addition of excessive amounts of urea, etc. Magnus-Levy has pointed out that, like casein, the Bence Jones substance lacks the hetero group, but it differs from it in the absence of phosphorus and the presence of a carbohydrate group. As his substance was not precipitated by salting with magnesium sulphate, or on passing a current of carbon dioxide through its solution, and as it proved soluble in distilled water, he concludes that the body does not belong to the class of globulins, even though in his case the limits of precipitation of the deutero-albumose A were the same as those of the corresponding deutero-albumose obtained from serum globulin. This relation and the limits of precipitation of the various other digestive products of the Bence Jones substance as compared with those of the more common albumins is shown in the accompanying table:

| | Fibrin. | Egg albumin. | Serum albumin. | Serum globulin. | Bence Jones albumin. |
|----------------------------|---------|--------------|----------------|-----------------|----------------------|
| I. Primary albumoses . . . | 2.6—4.4 | 3.6—4.6 | 4.2—4.6 | 3.6—4.6 | 2.8(3.2)—4.8 |
| II. Deutero-albumoses: | | | | | |
| Deutero-albumose A . . | 5.4—6.2 | 5.6—6.0 | 5.4—6.2 | 5.6—7.2 | 5.6—7.2 |
| Deutero-albumose B . . | 7.2—9.5 | 7.0—7.8 | 7.2—8.0 | 7.8—8.6 | 7.8—9.2 |

In contradistinction to Magnus-Levy's observation, I found that my substance could be precipitated by salting with magnesium sulphate to saturation, while at the same time there was no evidence whatever that the urine contained any other albuminous body in solution but the Bence Jones substance. Other observers, as I have already stated, failed to make a note of the behavior of the body in this respect, but with the material which Dr. Wood sent me from the Fitz-Wright case No. 2 I obtained the same result.

Why my results should differ so radically in this respect from those of Magnus-Levy I am unable to say, unless, indeed, his solutions were not altogether saturated. To insure this point I followed the suggestion of Marcus,¹ and treated the solution with from two to three times its volume of a saturated solution of magnesium sulphate, and then added the salt to saturation (Marcus, p. 567). The observation, however, is important, as it suggests that the Bence Jones substance may at least be closely related to the globulins, if, indeed, it is not a globulin itself. The fact that it is soluble in water cannot be adduced as an argument to the contrary, since it has been conclusively established that even in

¹ E. Marcus. Ueber in Wasser lösliches Serumglobulin, *Zeitsch. f. physiol. Chem.*, 1899, vol. xxviii, p. 559.

normal blood a serum-globulin exists which is water-soluble. According to Marcus, indeed, that portion which is insoluble in water represents but 9 to 23 per cent. of the entire quantity that may be precipitated by magnesium sulphate. This water-soluble portion, as would be expected, is not precipitated by dialysis. The substance gives the xanthoproteic reaction, the sulphur and biuret reactions, that of Adamkiewicz, as also that of Molisch. An estimation of the nitrogen gave 15.9 per cent.—i. e., a figure which approximates that which has been found for the Bence Jones substance, viz., 15.56 to 15.59. It is noteworthy, moreover, that in the case of a pure solution of the substance not even a turbidity is produced by means of a current of carbon dioxide, so that this point also cannot be brought forward as an argument that the Bence Jones substance should not be viewed as a globulin. On the contrary, I believe I have shown that cogent reasons exist for the assumption that the body is indeed closely related to the water-soluble globulin, as Marcus describes it. My material, however, was unfortunately not sufficient to institute a more detailed comparison between the two substances; and for other reasons also it seemed advisable to publish my results so far as they go.

Should further research bear out the correctness of my view, the previously supposed identity of Noël Paton's¹ crystalline globulin with the Bence Jones substance would appear to be rendered quite probable. Huppert,² indeed, had previously attempted to prove their identity and at one time regarded Noël Paton's substance as an albumose; later, however, he abandoned this view,³ and at present I think there can be no doubt that this substance also was in reality a globulin. In many respects the description of the body reminds one strongly of the Bence Jones substance. As in Bence Jones' case, it was eliminated in enormous quantities (15.0 to 70.0 pro mille, corresponding on one occasion to an elimination of 70 grammes in twenty-four hours), and, barring the Bence Jones substance, it is, indeed, the only albuminous substance which has ever been encountered in the urine in such large amounts. In this case, also, no tube-casts were found in the urine, and post-mortem examination revealed the absence of a nephritis. A further point of resemblance is the fact that Paton's globulin was obtained in crystalline form, and was deposited as such spontaneously from the urine. This occurred at times on standing for only one or two days;

¹ Byrom-Bramwell and D. Noël Paton. On a Crystalline Globulin Occurring in Human Urine, Reports from the Laboratory of the Royal College of Physicians, Edinburgh, 1892, vol. iv. p. 47.

² Huppert. Ueber einen Fall von Albumosurie, *Zeitsch. f. physiol. Chem.*, 1896-1897, vol. xxii. p. 500.

³ Huppert. Ueber den Noël Paton'schen Eiweisskörper, *Centralbl. f. med. Wissen.*, 1898, p. 481; cit. in *Maly's Jahresber.*, 1899, vol. xxviii. p. 302.

at other times only after weeks or months. A solution of the crystals in neutral saline solution coagulated between 56° C. and 59° C. Very suggestive also is the following observation, which is recorded in Huppert's second communication: "The coagulum dissolves in warm water after the addition of a few drops of a sodium carbonate solution, and on neutralization with hydrochloric acid a flocculent precipitate is formed, which apparently did not dissolve upon the addition of 5 per cent. of rock salt; but on heating the filtrate a turbidity occurred at 52° C." On boiling it is stated that the urine coagulated almost in bulk, and it would thus appear that Paton's substance differed from that of Bence Jones'; but, as I have shown, the behavior on boiling cannot be regarded as a criterion in differentiating between the latter and the common coagulable albumins. In other respects, it is true, points of difference apparently exist between the two bodies; but, on the whole, these are unimportant, and I believe that we may very well classify the two together. Although future researches may show that they are not identical, there is evidence to prove even now that they are at least closely related.

The case occurred in Byrom-Bramwell's practice, while the urinary examination was conducted by Noël Paton. Evidence of bone disease did not exist during life, but it is unfortunate that post-mortem a more detailed examination could not have been made.

THE ORIGIN OF THE BENCE JONES SUBSTANCE. Of the seat of origin of the Bence Jones substance very little is known that is definite. Ellinger states that he found a substance in the blood of his patient which gave the common albumose reactions and coagulated at about the temperature of the isolated body (he indicates 38° C. to 40° C.!). Ribbink and Askanazy, on the other hand, who are the only observers who made investigations in this special direction, report negative results. Ellinger does not state whether the blood in his case was withdrawn during the life of the patient, but even if this were done the result can scarcely be surprising unless, indeed, the substance was formed in the kidneys—a supposition for which there is no tangible basis.

Askanazy states that he did not find the substance in the tumor masses, but found it in a fresh extract of the bone-marrow, which, however, was the seat of extensive lymphadenoid degeneration. Ribbink, on the other hand, records that in his case it was present neither in the bone-marrow nor in the bone-substance. Ellinger reports similar findings in the case of the tumor masses, as of the blood, and Dr. Wood writes me that he obtained the same substance, which was present in the urine, from the diseased portion of the bone tissue, and that he did not find it in the bone-marrow in any other portion of the body of

or an alkali, the reports of all observers who have examined in this direction, with the exception of Ribbink, are the same. Ribbink states that he could not obtain a precipitate on neutralization, but it is quite likely, as Magnus-Levy suggests, that he had previously not added enough acid or alkali. Kühne, who regarded the Bence Jones substance as an albumose, speaks of the apparent change which results on treating with an alkali, as due to the formation of an albumosate analogous to the albuminate in the case of the common albumins.

Behavior on Salting with Magnesium Sulphate. In its behavior toward magnesium sulphate a marked difference apparently exists between my substance and that of Magnus Levy. For, whereas he reports that on saturating the urine with the salt in question, at the temperature of the room, no precipitation occurred, I noted in my case that after acidifying with acetic acid the substance is promptly thrown down. My notes on this point read as follows: Upon the addition of two volumes and a half of a saturated solution of magnesium sulphate to the urine no precipitation occurred, even after acidifying with acetic acid, but on saturation of the acid solution with the salt in substance, complete precipitation occurred. Saturation without the addition of acetic acid gave no result in the native urine. But on treating the neutral solution of the isolated substance (precipitation with ammonium sulphate and subsequent dialysis for eight weeks) with two or three times its volume of a saturated solution of magnesium sulphate, and then saturating with the salt in substance, the Bence Jones body was completely precipitated on standing for twenty-four hours, at the temperature of the room. The filtrate remained clear on heating after the addition of acetic acid; no further precipitate occurred on treating with twice the volume of a saturated solution of ammonium sulphate, and the biuret reaction was negative. The precipitate itself was soluble in water and could hence not be a coagulate, but must be regarded as a true salt-precipitate. Other observers have made no notes of the behavior of the substance in this respect; but it is interesting to note that the substance which Dr. Wood sent me (from Fitz-Wright case) could also be precipitated with magnesium sulphate from its neutral aqueous solution.

Behavior on Digestion. On digestion with pepsin-hydrochloric acid all observers who have made investigations in this direction report that the substance dissolves very readily, and that the presence of "peptones" can very soon be demonstrated. Matthes noted that on prolonged digestion a jelly-like material separated out, which, after drying, was soluble in absolute alcohol. This, however, as he later admitted himself, and as Neumeister indicates, was owing to the admixture of a nucleo-albumin. Ellinger records that on digestion with pepsin for forty-eight hours a small amount of residue was obtained, which con-

tained phosphorus. In his case, however, a nucleo-albumin was also present and had not been previously removed. Speaking of the products of digestion with pepsin-hydrochloric acid, Kühne states that after one or two hours only "peptone" was found in solution, and no precipitate was obtained either with nitric acid or with acetic acid and sodium chloride.

A more detailed examination of the resulting products of digestion according to more modern methods has been made by Magnus-Levy. As I have previously indicated, he claims to have obtained a proto-albumose, three deuto-albumoses, and two peptones, while the formation of a hetero-albumose was not observed. In view of the importance of these results in their bearing on the question of the chemical nature of the Bence Jones substance, it may not be out of place to consider in some detail the basis on which Magnus-Levy has established his claim that on peptic digestion the Bence Jones substance yields a proto-albumose. Magnus-Levy states that he employed Hofmeister's method of analysis, as described by Pick,¹ Umber,² and Alexander.³ According to this method the digestive mixture was neutralized (with ammonia or ammonium carbonate), brought to the boiling-point, and then concentrated on the water-bath to about one-fifth of the original volume. On neutralization Magnus-Levy states that he never obtained a precipitate, but that during the process of concentration flocculi separated out, which may have been due to acid albumin, but were present in too small a quantity for analytical purposes. On treating the filtered solution with an equal volume of a saturated solution of ammonium sulphate a precipitate then resulted, which Magnus-Levy regards as due to proto-albumose exclusively, as a hetero-albumose could not be obtained from this fraction, either on dialysis or on treating the aqueous solution of the precipitate with half and an equal volume of alcohol and boiling for several hours. He then further *identified* this fraction as proto-albumose by the following reactions, which he compares with those of the Bence Jones substance:

| | Sulphur test. | Millon's reaction. | Biuret reaction. | Molisch's test. | Adamkiewicz test. | Salting with rock salt. | Rock salt and acetic acid. | Dilute copper sulphate solution. |
|--------------------------|-----------------------|--------------------|------------------|--------------------------------|-------------------|-------------------------|--|----------------------------------|
| 1. Bence Jones substance | Markedly black color. | Intense. | Violet-red. | Faint but distinct. | Very pronounced. | No precipitation. | Complete precipitation. | Marked precipitation. |
| 2. Proto-albumose | Markedly black color. | Markedly red. | Purely red. | Much more marked than under 1. | Faint. | Precipitation. | Precipitation more marked than with rock salt. | Marked precipitation. |

¹ Loc. cit.² Loc. cit.³ F. Alexander. Zur Kenntniss d. Caseins u. seiner peptischen Spaltungsprodukte, Zeit. f. phys. Chem., 1896, vol. xxv. p. 411.

PARATYPHOID INFECTIONS:¹

WITH REPORT OF A CASE CLINICALLY IDENTICAL WITH TYPHOID FEVER
IN WHOSE BLOOD A PARATYPHOID BACILLUS WAS FOUND.

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SINCE Gärtner's discovery of the *B. enteritidis*, in 1888, increasing interest has attended the study of the intermediate members of the typhoid-colon group of bacilli. In 1893 Gilbert proposed the terms "paratyphoid" and "paracolony" to designate these intermediates; but Durham regards the terms as inappropriate, since he believes that the intermediates are well-defined species of organisms, and suggests that all be classified under the group name "Gärtner and its allies." From the clinical standpoint, however, the term paratyphoid is the most clearly descriptive that can be applied to infections resembling typhoid and caused by one of these intermediate bacilli.

According to Durham, the Gärtner group includes *B. enteritidis* and the similar organisms described in association with epidemics of meat-poisoning, the gas-producing "typhoid" bacilli which have been described by various observers, the *B. psittacosis*, the paracolony bacillus described by Gwyn, and the bacillus O (Cushing). Theobald Smith has shown that *B. cholerae suis* and *B. typhi murium* should be classified with this group, and Cushing suggests that the recent researches of Reed and Carroll on the *B. icteroides* of Sanarelli demonstrated that it, too, belongs here. In addition, Widal adds the bacillus of calf septicæmia of Thomassen.

Pathogenicity in the human subject has been established for certain members of the Gärtner group—*e. g.*, *B. enteritidis* in epidemics of meat-poisoning, *B. psittacosis* in a peculiar epidemic bronchopneumonia with typhoid symptoms in some cases, and the paratyphoid bacilli in cases resembling typhoid fever. Moreover, Grünbaum has suggested that febrile jaundice may be caused by one of the intermediates, since he has obtained the Widal reaction with sera from these cases.

Petrushky's bacillus *fecalis alkaligenus*, according to Durham's latest work, is not an intermediate of the typhoid-colon group, but occupies

¹ Read at a meeting of the New York Pathological Society, February 12, 1902.

a place beyond the typhoid in that it does not acidify any sugar-containing media; in a sense it out-typhoids the typhoid bacillus.

In the present paper we shall concern ourselves chiefly with cases clinically resembling or identical with typhoid fever which were apparently caused by an intermediate bacillus.

In 1896 Achard and Bensaude reported two cases which they considered due to infection with paratyphoid bacilli.

CASE I.—Woman, aged twenty-four years. Had never had typhoid fever. For several weeks had nursed a child ill with a "mucous fever." Taken sick August 24th with a chill, followed by fever, anorexia, slight diarrhoea, marked prostration, and tympanites. No spleen, no epistaxis, no rose-spots. Temperature, 103.3° F. No râles in the chest. Intestinal hemorrhage. September 11th, marked pyuria. September 14th, temperature fell suddenly to 98.6° F. without apparent cause, remounting to 100.4° F. September 20th, temperature 98.6° F., rising to 102.2° F. on the 23d, with a return of the diarrhoea and the development of right femoral thrombosis. October 2, left femoral thrombosis. October 5th, temperature normal.

Blood from vein at elbow remained sterile. Urine taken aseptically September 12th and 23d, both times gave a bacillus which was considered to be a paratyphoid. Blood aspirated from the spleen showed staphylococci, but no bacilli. Stools gave colon only.

September 9th, no Widal. September 14th, the serum reaction with same typhoid culture and the bacillus obtained from the patient's urine was positive. September 17th, two cultures of typhoid bacilli gave the reaction. October 16th, positive Widal with three of thirteen cultures of typhoid bacilli. Reaction constantly positive with paratyphoid. Constantly negative with colon. Seven weeks after the fever the patient's serum agglutinated the bacillus obtained from the urine and two cultures of paratyphoid bacilli, but was without action on five cultures of typhoid. The blood of animals immunized with the bacillus from the urine reacted with paratyphoid bacilli, and only with certain samples of typhoid bacilli.

The authors think the case presented a close analogy to typhoid fever complicated by pyelonephritis, with a relapse complicated by femoral thrombosis.

CASE II.—Infant, aged seven months. June 23d, temperature was 100.4° F., with subcrepitant râles over middle of left lung posteriorly. Temperature reached 104° F. on the 26th. The pulmonary signs disappeared, but the fever continued between 102.2° F. and 104° F. Tympanites, rose-spots in abundance. July 13th, temperature 98.6° F. Discovered swelling of right sterno-clavicular articulation, which was opened and a paratyphoid bacillus recovered from the pus. Patient recovered completely.

The authors conclude that while general infection with *B. typhosus* cannot be excluded in this case they can affirm positively that the local infection was paratyphoid in nature.

In discussing the paper, Rendu states that the authors have demonstrated that paratyphoid fever closely resembles typhoid in its course,

temperature curve, and abdominal symptoms. Cases of psittacosis infection, he says, may present the general appearance of typhoid fever, but show special pulmonary localization. He has seen three cases. All three presented signs of grave bronchopneumonia. None had diarrhoea, rose-spots, or tympanites. One died. There were no intestinal lesions at the autopsy.

In the following year, in his thesis, Bensaude contended that the bacilli isolated from these cases were *B. psittacosis* not paratyphoid as originally reported, but his bacteriological evidence only proves that the bacilli were intermediates. He did not conclusively differentiate the bacillus of psittacosis from paratyphoid bacilli. Moreover, he seems in doubt as to the real nature of the general infection in the two cases.

In 1897 Widal reported the following case: A tubercular patient, after three weeks in the ward, developed an abscess in the neck about the œsophagus, presenting at the same time slight constitutional symptoms. A bacillus was obtained from the pus, which, from its cultural characteristics, Widal considered a paracolôn. The bacillus gave a marked reaction with the serum of the patient in dilutions as high as 1:1000, the reaction steadily diminishing with convalescence. Widal concludes that the bacillus isolated was the cause of the patient's infection. The sera of animals inoculated with *B. coli communis* and with *B. psittacosis* gave no reaction with this organism.

Gwyn isolated, in 1898, from the blood of a patient suffering with a disease clinically identical with typhoid fever a bacillus which culturally corresponds with Widal's. The patient had been ill since September 17th, with headache, fever, weakness, and later with vomiting, diarrhoea, and pain in the abdomen. October 16th he had three hemorrhages from the bowel. He slowly rallied and went out of the hospital in five weeks. Rose-spots were present, the spleen was palpable, the urine gave the diazo-reaction. The temperature was that of a severe typhoid. The Widal reaction was never found. Blood cultures taken October 12th gave a paracolôn bacillus. The patient's serum reacted with this bacillus in dilutions of 1:150 to 1:200. Two months after the date of the culture there still remained a slight reaction. The same serum was without action on *B. typhosus* in any dilution above 1:1 to 1:5.

Cushing's case followed in August, 1900. M. B., colored, aged twenty-seven years. From early in June, 1898, nine months before admission, the patient had suffered with a prolonged course of fever extending over a period of ten weeks. Clinically, it was typhoid fever, with a severe but typical course. There was a relapse ushered in by profuse epistaxis. During convalescence two tender swellings developed near the breast-bone. One disappeared, the other burst, dis-

charging a large amount of pus and leaving a sinus at the level of the fifth rib. Microscopical examination showed the process to be an osteomyelitis. There was no Widal reaction. Cultures taken from the sinus at the time of the operation gave an intermediate bacillus, which Cushing calls bacillus O.

In the same month appeared Schottmueller's first case. Male, aged twenty-six years. The patient was taken ill July 23d with headache and catarrh of the upper respiratory passages. Temperature 104° F. July 27th, the patient was sleepy and tired; spleen palpable. July 28th, the sixth day of the disease, the blood furnished a bacillus resembling the typhoid, but fermenting glucose. Rose-spots present. Temperature normal, August 5th. The only symptom of typhoid lacking was the stools. August 1st the Widal reaction was negative, while the bacillus isolated from the blood reacted with the patient's serum at 1:50, but not in 1:100. Later the reaction was obtained at 1:100. With three other typhoid cultures the reaction was negative. Of the sera of four typhoid patients only one gave a reaction with the bacillus, and that at 1:20.

In 1901, Schottmueller reported five more cases, clinically like typhoid fever, in whose blood he found paratyphoid bacilli. None of the cases gave the Widal reaction. There was a sixth case which did not give the Widal reaction from whose blood he failed to obtain a bacillus, but this may be explained by the fact that the blood was drawn only three days before the end of the fever.¹

CASE I.—Male, aged sixty years. Taken ill July 7th with headache, lassitude, diarrhoea. Spleen not palpable. No spots. Temperature, 104° F. Temperature normal August 1st. Mild type.

CASE II.—Male, aged eighteen years. Taken ill July 13th with headache, etc. Temperature 105.2° F. July 18th, condition bad. July 23th, condition dangerous. September 18th, discharged cured. Severe typhoid type.

CASE III.—Male, aged nineteen years. Taken ill August 16th with headache, etc. Temperature, 104.3° F. Rose-spots present. October 1st discharged cured. Very mild type.

CASE IV.—Male, aged forty-six years. Taken ill about August 12th with headache, etc. Had been in bed six days before admission to hospital. Rose-spots present. Spleen palpable. Temperature 103° F. Discharged cured December 14th. Mild type.

CASE V.—Male, aged fifteen years. Admitted November 3d. For fourteen days had been ill with fever, lassitude, etc. Temperature 105.4° F. Many rose-spots, which appeared on the face and hands as well as in the usual situations. Patient in bad condition until November 12th. December 30th he was discharged cured. Severe typhoid.

CASE VI.—Male, aged twenty-five years. The patient was a physi-

¹ In a limited number of cases of typhoid fever we have failed to find the bacillus in the blood after the second week, and it will be noted in our case of paratyphoid infection that the blood drawn on the twenty-seventh day of the disease remained sterile.

cian who had assisted in the examination of Case II. Temperature 101.6° F. Dismissed cured September 16th. The blood did not give the Widal reaction. Blood taken three days before the end of the disease remained sterile. The diagnosis of typhoid fever was made by exclusion.

There were no complications with any of the cases, even the severe ones. Case IV. was alcoholic, Case I. was sixty years of age. In these cases, the author says, one would expect a bad prognosis in true typhoid fever, yet both were mild.

The six cases above recorded occurred in a series of sixty-eight cases which Schottmueller examined, a high proportion, if we can judge by the comparative frequency of the Widal reaction in typhoid fever.

Schottmueller applies the term paratyphoid both to the infection and to the bacillus.

Kurth also published, in 1901, five cases which he considered paratyphoid fever. These cases were found among sixty-two whose sera were tested for the Widal reaction.

CASE I.—Woman, aged thirty years. Taken sick May 17th. Temperature 103.2° F. Normal June 11th. Tendency to constipation. Widal negative except in dilution of 1 : 3. June 30th the bacillus was recovered from the urine.

CASE II.—Male, aged twenty-nine years. Taken sick August 27th, having had headache for a week, with fever and diarrhoea. Splenic tumor, rose-spots, diazo-reaction. Temperature 100.4° F. to 104.9° F. No complications. Widal negative three times. Bacillus isolated from stools September 19th. The patient's serum reacted with this bacillus at 1 : 8000, and on October 28th at 1 : 2000.

CASE III.—Male, aged twenty-five years. Taken sick September 3d with headache and vomiting. Splenic tumor, few rose-spots, diazo-reaction. Temperature up to 104° F. Widal reaction was tested three times, and was positive once at 1 : 3. No paratyphoid bacilli found either in stool or in urine. The patient's serum reacted with the bacillus from Case II. at 1 : 500.

CASE IV.—Male, aged eighteen years. Taken sick October 26th. Typhoid stools. Temperature 98.6° F. to 104° F. Normal November 27th. Rose-spots, splenic tumor. Widal negative twice. The patient's serum reacted with the bacilli from Cases I. and II. at 1 : 500.

CASE V.—Woman, aged twenty-three years. Entered hospital November 7th. No splenic tumor; doubtful rose-spots; no diazo-reaction. Irregular temperature, never above 102.2° F. Serum reaction positive.

All the cases were mild. None had complications. In Cases III. and IV. the cultures were not taken till late in the disease, which may explain the negative result.

In the course of a study on the bacteriology of the blood in typhoid fever we encountered the following case :

Louise R., colored, aged twenty-eight years, entered Bellevue Hospital in the service of Dr. Loomis, October 9, 1901. She gave the

following history: For the last two months she had been living in Elizabeth City, N. C., and was taken ill one week after her return to New York. On October 4th the patient had a chill which lasted about half an hour and was followed by fever and sweating. Her appetite has been poor and she has had frequent attacks of vomiting, the vomitus consisting of the food last eaten. There have been two attacks of epistaxis. Since October 6th she has suffered from severe headache. Her bowels have moved freely, with a slight tendency to diarrhoea. The patient feels very weak. Temperature on admission was 103.2° F., pulse 92.

Physical examination on admission. The patient is well nourished, the muscles are firm, the skin is moist and elastic. The mucous membranes are red. The tongue is tremulous, fissured, coated over the dorsum, red at the tip and edges. The pulse is small, frequent, regular, and of low tension. The heart is negative. The lungs are negative except for slight impairment of resonance posteriorly. The abdomen is soft, but gives a tympanitic note. Liver normal. Spleen not palpable. Slight epigastric tenderness. Owing to the patient's color we could not determine the presence of rose-spots.

The patient had two stools on each of the first two days in the hospital, but had been given a dose of castor oil. Subsequently there was slight constipation.

An examination of the urine, made October 10th, showed a trace of albumin, with granular and hyaline casts, blood, pus, and epithelial cells. A leucocytic count October 11th gave 10,600. The same day the blood was examined for the malarial parasite, with a negative result. The Widal reaction, tested by Dr. T. W. Hastings, was tried three times: October 10th, dilution 1:80, negative after one hour; October 16th, dilution 1:40, negative after half an hour; October 31st, dilution 1:80, negative after one hour. With another culture of typhoid bacilli an imperfect and only partial reaction was obtained at the end of an hour in a dilution of 1:20. On October 9th, the sixth day of the disease, and on October 30th, the twenty-seventh day of the disease, 10 c.c. each of blood were taken from a vein at the elbow for bacteriological examination. The first of these specimens gave a paratyphoid bacillus, the second remained sterile.

Clinically the patient followed the course of a mild attack of typhoid fever. The spleen became palpable as the disease progressed. The subsequent history was uneventful except for a sharp break in the temperature to 99.6° F. on the fourteenth day of the disease, after which the exacerbations and remissions were very marked till the twenty-second day, when the temperature fell to 98.6° F. and convalescence was definitely established. The patient was discharged cured November 20th, six weeks after entrance to the hospital and seven weeks after the beginning of her illness.

Rose-spots were sought for many times, but because of the patient's color it was impossible to decide if they were present.

To sum up: The illness was ushered in with a chill, followed by fever, anorexia, lassitude, slight diarrhoea and fibrillary tremor of the tongue. The temperature curve was continuously high to the fourteenth day of the disease, when it fell to 99.6° F., after which the curve was broken by exacerbations and remissions to the twenty-second day,

the first day of convalescence. Added to these symptoms there was splenic tumor. So that in all essential features the case was typical of mild typhoid fever.

Analysis of the Symptoms of Paratyphoid Fever. In view of the care with which the cases from whose blood the paratyphoid bacilli have been isolated were studied, it must be admitted that these organisms are capable of causing a general infection that has to be reckoned with in the diagnosis of typhoid fever. An analysis of the symptoms of the reported cases demonstrates that this infection closely resembles true typhoid infection, and may even be identical with it in clinical manifestations. Practically all of the cases of paratyphoid infection passed clinically for typhoid fever, without a Widal reaction, until careful bacteriological examination revealed their true nature.

In so far as conclusions can be drawn from the limited number of cases reported, paratyphoid infections may be mild or severe as in genuine typhoid. The symptoms of invasion are practically the same, for we may find in both headache, lassitude, anorexia, diarrhoea, epistaxis, chill followed by fever, and slight inflammation of the upper respiratory passages.

Moreover, during the course of paratyphoid infection the patient develops the typhoid state, slight or profound, accompanied by tympanites, diarrhoea or constipation, rose-spots, splenic tumor, and quite a typical temperature curve. The pulse is that of typhoid fever. The urine sometimes gives the diazo-reaction.

Abscess of the neck, osteomyelitis, pyelonephritis (?), and intestinal hemorrhages have been reported as complications. Even a relapse occurred in Cushing's case. So that the picture is more or less complete as a whole.

For the most part the cases have been mild, and Schottmueller states on the evidence of his seven cases that the prognosis is favorable.

Certain irregular typhoid cases have been reported in which the Widal reaction was absent, which we are inclined to regard as examples of paratyphoid infection. Brill has made a careful study of seventeen of these cases. In our case there was a remarkable break in the temperature curve, without assignable cause, to 99.6° F. on the fourteenth day of the disease, though the temperature afterward remounted to 101.8° F. Possibly some of the cases of the so-called fourteen-day type of typhoid fever are really paratyphoid fever, but we have been unable to obtain any data on this point. Dr. Thomas D. Coleman, of Augusta, Georgia, writes us that the fourteen-day typhoid fever is quite common in that section, and that in all essential symptoms it is identical with the twenty-one-day type. He also states that the Widal reaction was negative in one of these cases, but a bacteriological examination of the blood or feces was not made.

Brill's cases bore a marked clinical resemblance to typhoid fever, though the duration of the disease was only ten to twelve days. The serum reaction of these cases was tested with typhoid bacilli daily, with negative results. The symptoms, briefly stated, were: anorexia, malaise, headache, and general body pains for three or four days, succeeded by rigors or a chill, and sometimes epistaxis. The temperature rose rapidly to 104° F. to 105° F., after which a mild typhoid state developed. Rose-spots were generally present. The spleen was enlarged. Constipation was the rule. The temperature reached normal ten or twelve days after the beginning of the illness. Inconstant diazo-reaction. No leucocytosis. No intestinal hemorrhage. No complications or sequelæ.

In three of the cases cultures were made from the feces and *B. coli communis* obtained. In one case the spleen was aspirated, and again *B. coli communis* found. The serum of each case was tested for a reaction with the colon bacillus and found negative. And in two cases the serum of the patients was tested with colon bacilli obtained from themselves, with no reaction. In these two cases then the bacilli could not have been paratyphoid bacilli.

Case XIV. of Brill's series is especially remarkable. The patient had had typhoid fever, with the Widal reaction, six months previously and had entirely recovered. The serum reaction against the typhoid bacillus persisted up to and including part of the second illness, during the course of which it disappeared. Brill justly claims that the second attack was not typhoid fever.

The cases reported as fatal typhoid fever with intestinal lesions, not showing the Widal reaction, without bacteriological examination are not proved to have been genuine typhoid infection.

The Widal Reaction in the Light of Infections by Members of the Gärtner Group of Bacilli. Since the introduction of the serum reaction as a means of diagnosis in typhoid fever it has been a well-recognized fact that a small proportion of cases which are clinically typhoid fever fail to give the reaction. Brill, adding to Cabot's statistics, finds that of 4879 cases 4781, or 97.9 per cent., gave the reaction. Gwyn gives 99.6 as the percentage of positive reactions in the Johns Hopkins Hospital. On the contrary, in all the reported cases of paratyphoid infection the reaction has been absent. It is probable, then, that some, at least of the typhoid cases with negative reaction, were really paratyphoid. That not all of them were paratyphoid is made probable by the following remarkable case reported by H. Batty Shaw:

Adult. Headache for three days. Temperature 103° F. Typhoid fever was suspected, and on the two following days the Widal reaction was tried at 1:50, and both times failed. On the seventh day of the disease rose-spots appeared and remained visible until the nineteenth

day. Splenic tumor. Delirium and incontinence for three weeks. Temperature normal the twenty-fourth day. An abscess developed in the region of the right rectus abdominis. It was incised the thirty-sixth day of the disease and a typhoid bacillus isolated from the pus. The patient's own serum in dilution of 1 : 200 failed entirely to clump this bacillus after an hour's exposure. Another typhoid case in the hospital, giving the Widal reaction with another typhoid culture, failed to react with this bacillus at the same dilution. The serum of the patient was then tried with a typhoid culture from University College, but in dilution of 1 : 100 gave only the slightest clumping after twenty-four hours. The same culture with the serum of another patient with typhoid fever gave the reaction in two minutes.

The bacillus isolated corresponded to the following biological and other tests: 1. Decolorized by Gram. 2. Actively motile in broth—no scum. 3. Shake preparation in glucose-agar, no gas. 4. No indol in broth after ninety-six hours. 5. Growth on potato present, but not visible. 6. Did not curdle milk.

No adequate explanation offers itself in this case, except some biological peculiarity which was not demonstrated. It scarcely seems possible that the bacillus isolated was not the cause of the patient's infection.

It cannot be assumed that all cases, clinically typhoid, which have been reported as giving the Widal reaction were cases of genuine typhoid infection. The brilliant work of Durham on the typhoid colon group and their serum reactions has established the fact that certain members of this group may be mutually interacted upon by sera of infected patients and immunized animals. This is especially true of sera in low dilution, and since in the earlier years of the Widal reaction the technique had not been worked out, and dilutions were more frequently low than not, some of the cases reported as typhoid fever may have been paratyphoid. The simple fact that clumping of bacilli has been obtained is not sufficient evidence for diagnosis. Gruber and Durham have proved that a serum reaction is not in the strict sense *specific*, that it is only a *special* reaction, and that it indicates not a certainty, but a probability.

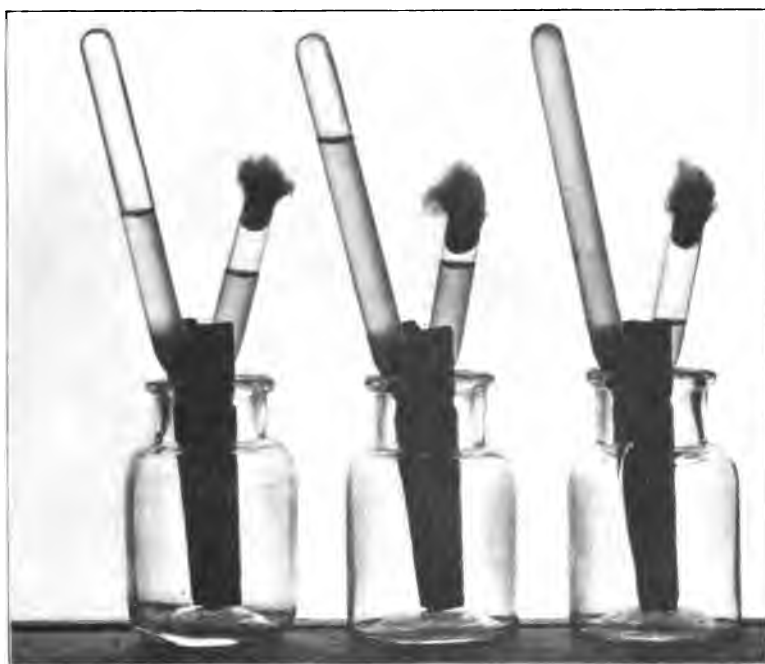
Landsteiner was the first to observe that typhoid serum reacts positively against Gärtner's bacillus enteritidis. Durham has confirmed this observation. In his study of an epidemic of meat-poisoning at Oldham he found that the sera of 5 of 29 cases of infection with *B. enteritidis* gave minimal traces of reaction against the typhoid bacillus at 1 : 100, and in lower dilution some of them gave well-marked clumping with typhoid cultures. Hence, he states that "in the absence of differential tests they might have been returned as having been obtained from typhoid fever patients." In another place he makes an even stronger statement based upon tables he gives, that, for the cases examined, in dilutions "lower than and including 1 : 100 it is imma-

terial whether true typhoid or Gärtner's bacillus is used to obtain the typhoid reaction." He adds that "sera obtained from typhoid fever patients are not potent enough to give absolute indications either for diagnosis of bacilli or the nature of serum."

In our case the serum reacted with the typhoid bacillus at 1:20, though only partially and imperfectly at the end of an hour, and failed entirely at 1:40 and 1:80 with another typhoid culture. In Gwyn's case his paracolon was agglutinated by typhoid sera of high potency

FIG. 1.

Gas formation in 2 per cent. glucose broth.



Coll communis.

Case 7.

Typhoid.

(1:300 to 1:1100) in dilutions up to 1:5. One typhoid serum (potency 1:900) gave an incomplete reaction at 1:30.

Moreover, Gilbert and Fournier and Achard and Bensaude have shown that *B. psittacosis* is agglutinated by typhoid sera at 1:10, but not at 1:40 to 60.

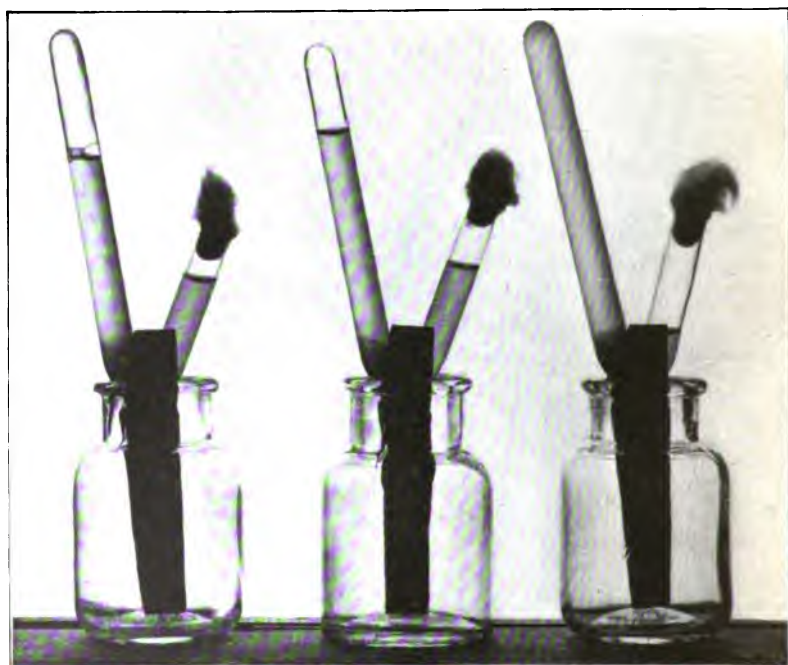
Hence, it cannot be too strongly insisted upon that the ordinary routine test for the Widal reaction give place to more reliable and scientific methods. Dilutions of sera should be high, rarely or never less than 1:50, and in the absence of a positive reaction the serum

should be tested against several of the intermediates. Not until this is done will the data of the serum reaction in typhoid fever possess scientific value.

BACTERIOLOGICAL REPORT. Although the bacteriological work on the bacillus in question, here called Case 7, has not been completed, enough has been done to show that it unquestionably belongs to the group intermediate between *coli communis* and typhoid, and that among the intermediates themselves it resembles Cushing's bacillus O

FIG. 2.

Gas formation in 2 per cent. maltose broth.



Coli communis.

Case 7.

Typhoid.

and Gwyn's (paracolon) bacillus—Durham's group C—more nearly than the *B. enteritidis* of Gärtner, hog cholera and others—Durham's group D.

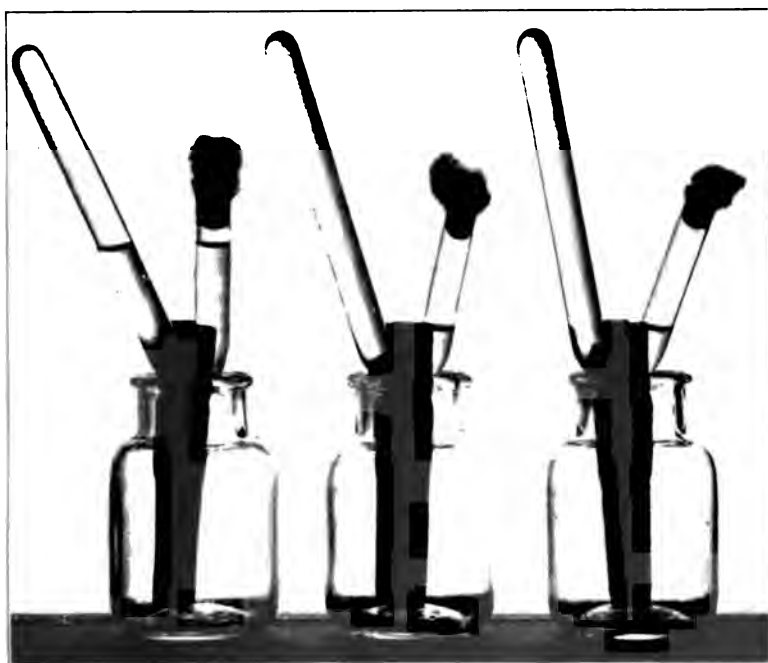
Of the 10 c.c. of blood drawn from the median basilic vein, 4 c.c. was distributed into four Erlenmeyer flasks, each containing 50 c.c. of broth; the remaining 6 c.c. being dropped into six tubes, each one containing 6 c.c. of broth. The flasks and tubes were incubated, and the following day two of the flasks appeared turbid. On the third day the other two flasks and one of the tubes were cloudy, but the remaining

tubes showed no growth either then or later. This experiment and many other similar ones undertaken by us have shown the necessity for considerable dilution of the blood if positive results are to be looked for. Cole has discussed this point very fully.

The turbidity of the four flasks and one tube was found to be due in each instance to a pure culture of the same bacillus, which at first was taken to be typhoid in spite of the fact that the patient's blood gave no Widal reaction with typhoid cultures, even in low dilutions. This

FIG. 3.

Gas formation in 2 per cent. lactose broth.



Coli communis.

Case 7.

Typhoid.

absence of reaction at that time was ascribed to the early stage of the disease, fifth or sixth day, in which the blood was taken. On testing the bacillus, however, in glucose gelatin it was found to produce gas, and this led to further investigations.

In ordinary media, agar, gelatin, and broth, the growth is indistinguishable from typhoid, nor do stained preparations, ordinary or by Gram's method, show any differences.

Hanging Drop. The bacillus is very motile, even more so than typhoid usually is, and in this resembles the bacilli isolated by Schott-

mueller, Kurth, and others. Kurth remarks that his bacilli possess flagella which are larger than those of typhoid and do not show the same peritrichal arrangement. In our bacillus, however, the flagella appear to be precisely similar to those of typhoid.

Serum Reaction. The bacillus was hardly affected by serum of the patient in the early (fifth, or sixth day) stage of the fever, but serum taken on the twenty-seventh day, at which time cultures from the blood were negative, agglutinated the bacillus in high dilutions, reacting toward typhoid cultures only in low, one to twenty dilutions, and even then, slowly and more or less imperfectly.

Later on the dried blood of the patient agglutinated cultures of Cushing's O and Gwyn's paracolon, but in somewhat lower dilutions. The blood of a rabbit immunized to Case 7 by repeated subcutaneous injections reacted strongly with the bacillus in high dilutions, and also with O and Gwyn, but was entirely negative with cultures of typhoid.

It seems, however, doubtful if the serum reaction will prove of value in distinguishing the intermediates as an entire group from typhoid on the one hand and coli communis on the other. It is true that Schottmueller found that his bacilli (six) would interact for the most part and that Kurth reports the same with his two cultures, but Durham prepared a highly potent Gwyn serum which reacted 1 : 20,000 with Gwyn, but had no effect upon O in 1 : 100 dilutions, and Cushing himself observed that the serum of his O patient reacting 1 : 8000 with O bacillus had but slight effect upon Gwyn's bacillus at 1 : 10.

These observations so far as O and Gwyn are concerned are at variance with our own, but we find with Durham that interactions between the members of the enteritidis group cannot be depended upon: Cultures which could not be distinguished either morphologically or biographically when tested by serum for agglutination showed very marked differences.

Gas Formation. The photographs show, better than a description could do, the gas formation in glucose and maltose and the absence of it in lactose broth.

In this respect Case 7 is typical of the intermediates, all observers agreeing that they do not form gas in lactose or saccharose media. So far as saccharose goes the gas test is of no value, since many true coli bacilli are also unable to ferment it. Durham, indeed, divides the coli bacilli into two groups, coli communis verus, which does not, and coli communis communior, which does ferment saccharose. The photographs show less gas formation on the part of Case 7 than of coli communis. This difference, however, is not constant, and the same may be said of the other intermediates so far tested.

Milk is not coagulated. Schottmueller lays great stress on the fact that among the intermediates there is initial acidity in the milk, fol-

lowed by alkalinity, which in two or three weeks may reach such a point that the casein is dissolved and the milk becomes opalescent. Cushing observed the same thing. This partial clearing was found to occur with *B. enteritidis* and O, but not with Gwyn or Case 7.

Potato. The growth is slight and there is no discoloration, so on this medium the bacillus resembles typhoid.

Litmus whey is a medium on which Durham places great reliance, but experiments with Case 7 have led to no particular results. It clouds the whey slightly and turns it acid, while typhoid leaves it clear and forms comparatively little acid, but trials so far have not shown that the intermediates form alkali later on, as claimed by Durham, and by itself it does not seem as if litmus whey would be of much value for differentiation.

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AN ANALYSIS OF SEVENTY-ONE CASES OF TYPHOID FEVER TREATED IN THE CHILDREN'S HOSPITAL OF PHILA- DELPHIA DURING 1901.¹

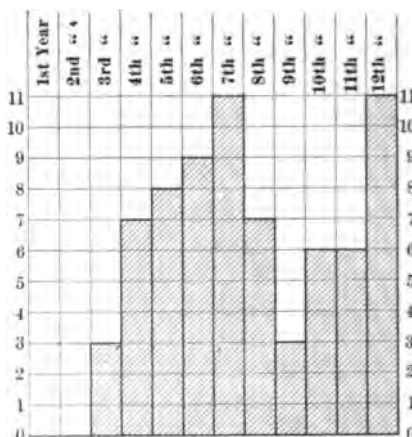
BY ALFRED HAND, JR., M.D.,
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AND

JOHN K. WALKER, M.D.,
FORMERLY RESIDENT PHYSICIAN.

"THE summer of 1901 was marked in Philadelphia by an epidemic of typhoid fever affecting children especially. The number of cases of this disease admitted to the Children's Hospital rose correspondingly, the increase being at a time when the medical wards were under our care, one of us substituting for Dr. Alfred Stengel, to whom, with the other members of the medical staff, Dr. J. P. Crozer Griffith and Dr.

FIG. 1.



Frederick A. Packard, we are indebted for the privilege of analyzing all the cases of typhoid fever that were treated in the hospital during the year. The number analyzed, 71, falls short by 2 of those in the annual report, one of the omitted cases being a convalescent remaining in the wards from the preceding year; the other was a patient admitted with a positive Widal reaction, the temperature, 100° F., falling to normal the next day, and scarlet fever developing two days later, for which she was removed from the hospital.

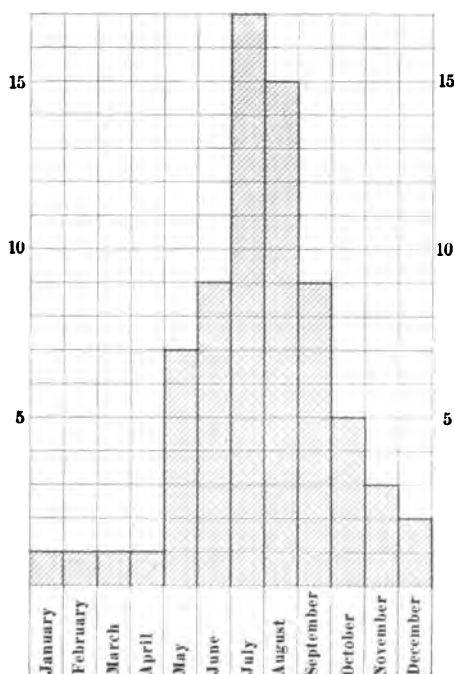
The monthly incidence of the cases is shown by the accompanying chart (Fig. 1).

The sexes showed about the same proportion as in adults, 42, or 59.1 per cent., being boys, and 29, or 40.9 per cent., girls.

¹ Read before the Medical Society of Germantown, January 27, 1902.

Figure 2 shows some interesting features with regard to the ages of the patients. In spite of the fact that the baby ward, with a capacity of twelve beds, was constantly filled all the summer, there was no case under two years of age which aroused even the suspicion of typhoid fever, although careful watch was kept for such a case. The cases showed a decided increase after the age of two years, the maximum being reached between six and seven years of age; in seeking for an explanation of this and of the drop between eight and nine years of age, it seems reasonable, although the number of cases is not very great, to attribute the increase to the digestive disturbances which are of so

FIG. 2.



frequent occurrence between the shedding of the milk teeth and the eruption of the permanent teeth. When these digestive disturbances have subsided there seems to be a period of relative insusceptibility for a year or two, but the age limit (twelve years) for admission to the hospital makes it impossible to say whether or not the second increase continues through later childhood and puberty to the highly susceptible period of early adult life.

Sixty-five of the children (91.55 per cent.) were white, the remaining six being colored. The figures are too small to form the basis of any inference as to comparative racial susceptibilities, but it is interesting

to note that the proportion of colored children (8.45 per cent.) is curiously almost exactly that present in the total number of admissions for all causes (8.49 per cent.).

A study of the city wards from which the patients came showed nothing unusual in the distribution. Dividing the part of the city between the Delaware and Schuylkill Rivers into quarters, using Broad Street as the north and south line, and Market Street as the east and west, nine patients came from the northeastern, three from the northwestern, twenty from the southwestern, and twenty-nine from the southeastern, with ten from West Philadelphia, which figures correspond closely to the general proportions in which the hospital draws its cases from these sections. The series serves, at least, to illustrate the widespread prevalence of the disease in the city and the practical impossibility of attributing the source to anything else than the water-supply. It is therefore earnestly to be hoped that the filter plants now building will be speedily completed.

With reference to the day of the disease on admission—

| | |
|--|----------------|
| 1 was admitted on the | 1st day. |
| 2 were " " " " " " " " " " " " | 2d " |
| 2 " " " " " " " " " " " " | 3d " |
| 6 " " " " " " " " " " " " | 4th " |
| 4 " " " " " " " " " " " " | 5th " |
| 8 " " " " " " " " " " " " | 6th " |
| 19 " " " " " " " " " " " " | 7th " |
| 5 " " " " " " " " " " " " | 8th " |
| 2 " " " " " " " " " " " " | 9th " |
| 2 " " " " " " " " " " " " | 10th " |
| 2 " " " " " " " " " " " " | 11th " |
| 1 was " " " " " " " " " " " " | 12th " |
| 2 were " " " " " " " " " " " " | 13th " |
| 8 " " " " " " " " " " " " | 14th " |
| 3 " " " " " " " " " " " " | 15th " |
| 1 on each of the (inclusive) | 16th to 19th " |

The prodromal symptoms as obtained by the resident physicians at the time of admission were, in the order of frequency, as follows:

| | | | |
|----------------------------------|----|---|---------------|
| Fever in | 71 | = | 100 per cent. |
| Headache in | 50 | = | 70 " |
| Diarrhœa in | 36 | = | 50 " |
| Abdominal pain in | 23 | = | 32 " |
| Constipation in | 21 | = | 30 " |
| Vomiting in | 19 | = | 26 " |
| Nose-bleed in | 13 | = | 18 " |
| Delirium in | 5 | = | 7 " |
| Chill at onset in | 2 | = | 2.8 " |
| Convulsions in | 1 | = | 1.4 " |
| Diarrhœa with blood in | 1 | = | 1.4 " |

The highest temperature observed in the hospital was 106.6° F.

| | | | | |
|-------------------------------------|---|------|-----|-----|
| In 6 cases the temperature was over | . | . | . | 106 |
| In 16 " " maximum was between | . | 105° | and | 106 |
| In 25 " " " " " | . | 104 | " | 105 |
| In 11 " " " " " | . | 103 | " | 104 |
| In 6 " " " " " | . | 102 | " | 103 |

While in one case, admitted on the fifteenth day, with enlargement of the spleen and a positive Widal reaction, the temperature was 100° F. on admission, and fell to normal in two days.

The shortest course which the fever ran was nine days in one case, while the longest was forty-four days. The temperature reached normal on an average after twenty-four and one-third days. Of the cases going on to recovery the shortest stay in the hospital was eleven days, the longest was eighty-two days, with an average stay of thirty-six days.

After admission to the hospital the patients presented the ordinary symptoms of typhoid fever in the following frequency :

1. *Widal Reaction.* This was positive in fifty-six out of sixty-four cases, no test being recorded in seven. The percentage of the cases giving the reaction was 87.5 per cent., and this would probably have been much greater if more frequent tests had been made in the negative cases. Thus in but one case were three tests made, each with a negative result, the days not being recorded. In one other case negative results were obtained on the fifteenth and seventeenth days. In the other cases but one test was made, with a negative return, on the sixth, ninth, tenth, twelfth, and seventeenth days, respectively. The reaction is thus, in our experience, the most frequent positive symptom of typhoid fever, but we hasten to add that when it is negative it is not of great value in excluding typhoid fever, provided that other symptoms point to the existence of the disease. Thus two of our patients were sisters, both looking on admission like typhoid fever; the younger one's attack ran a mild course, without spots or enlargement of the spleen, and with the Widal reaction negative until after deferescence, when it was instantly positive on the twenty-seventh day; the older sister had a severe attack, with high fever (maximum 106° F.), enlarged spleen and spots, the Widal reaction being negative repeatedly during the first attack, the first positive result being obtained on the twenty-seventh day, or the tenth day of the relapse. A similar case was that of a colored girl, aged seven years, admitted on the sixth day, having had fever, epistaxis, diarrhoea, abdominal pain, and headache; the symptoms in the hospital were high, continued fever, bronchitis, rigidity of the neck, exaggerated knee-jerks, ankle clonus, Kernig's sign negative, splenic dulness increased, but spleen not palpable, constipation, coated tongue, moderate leucocytosis, 12,600, the

Widal tests being negative on the seventh, tenth, twelfth, and sixteenth days; after a few days of apyrexia a relapse started, and the Widal reaction was found to be positive on the twenty-fifth and forty-second days from the start of the first attack.

2. *Enlargement of the Spleen.* This was present in fifty-nine, or 83 per cent. of the cases, and was usually easily recognizable, the nose of the organ being felt either just under or, more frequently, below the costal border; the largest downward projection observed was 4 cm. from the edge of the ribs. In some of the cases diagnosis of the enlargement rested on increase in the area of splenic dulness, while in a few it was necessary to rely on tenderness. Enlargement of the spleen is, however, so easily detected in children that we accept unhesitatingly the statement in the notes of twelve cases, "spleen not enlarged."

3. *Spots.* Of the sixty-five white children fifty-two, or 80 per cent., showed typical spots, the eruptions not being profuse in the cases under our care. In one case the eruption was confluent, the case being of average severity, judging from the notes and the length of the febrile course.

4. *Constipation* was present in thirty, or 42 per cent., of the cases, a slightly greater number than those with *diarrhœa*—twenty-seven, or 38 per cent.

5. *Coating of the tongue* is mentioned in but twenty-three, or 32 per cent.; the unimportance of this symptom is probably one reason for its passing unnoted, but it is not infrequent to see a child's tongue remain clean throughout the course of the disease; on the other hand, we have seen in children tongues as dry, brown, and fissured as in adults.

6. *Delirium* was sufficiently marked in eleven cases to be worthy of note, the male sex showing a preponderance more marked in the violence than in the number, seven of the patients being boys, or 16.9 per cent. of the males, the remaining four girls being 13.7 per cent. of the female cases.

7. *Intestinal hemorrhage* occurred in four cases, the amount of blood in the stools of three being very slight and scarcely enough to be called a complication; in one case there were three passages, each containing considerable blood, but without any shock or interruption to the favorable course of the case.

8. *Nose-bleed* was noted in three cases.

9. *The diazo-reaction* was tried in seventeen of our own cases, and was negative in but one, this case also failing to give a positive result in three Widal tests, but presenting a remittent fever-curve, spots, and an enlarged spleen. As an illustration of the value of the test in one case admitted as pneumonia, with consolidation of the right apex and pneumococci in the sputum, a positive reaction on the eighth day was the first indication of a typhoid infection which ran a typical course.

Thirteen, or 18 per cent., of the cases had complications. In half of these otitis media occurred, being associated in one case with pneumonia, in one case with alveolar abscess and caries of the jawbone, and in one case with a perirectal abscess. The other complications were cervical adenitis, without suppuration, in one case, and with it in another; jaundice in one case; pustules twice; furuncles in one case; abscess, vaginitis, and nephritis in one case; noma in one case, ending fatally; diphtheria in one case, which was removed to the Municipal Hospital, where it died; a prolonged cystitis existed in one case during convalescence, and the urine, collected as aseptically as possible, contained many bacteria; a culture on agar gave a growth of a bacillus which was actively motile; in a hanging-drop agglutination and cessation of motion were observed on the addition of a blood-serum known to give the specific typhoid reaction.

Relapses were seen in six patients, or 8.5 per cent., starting on the seventeenth, twenty-second, twenty-ninth, thirty-fourth, and forty-first days, respectively. In these cases the primary attack was severe in one, moderate in one, and mild in all the others.

The deaths numbered three, a mortality of 4.2 per cent. One case died of noma, one of diphtheria, and one of toxæmia, the mortality, therefore, of uncomplicated typhoid fever in this series being less than 1 per cent.

The routine elements of treatment in the cases under our care consisted of rest in bed and a liquid diet. Not knowing of any specific for the disease, the treatment was to a certain extent symptomatic. Using the temperature as an indication of the degree of toxæmia, an endeavor was made to combat the effects of the poison and to secure its elimination by hydrotherapy. This was started in the majority of cases as a tub bath, the temperature of the water being 85° F. A cloth wrung out of ice water was put on the child's head and the body immersed in the bath, the duration of which was from five to ten minutes, the influence on the temperature and on the pulse being the guide; while in the water the surface of the body was gently rubbed by the hands of the nurses. In a few cases, especially among the younger children, the tub baths were not well borne, and sponging with water of the same or higher temperature was substituted. Sponge baths were adopted at the start in some cases, but they often failed to influence the temperature sufficiently, the tub baths being used later with good effect.

As an added means of elimination we used in about twenty-five of the cases drachm doses of the solution of the citrate of potash well diluted with water, with the hope also that it would protect the kidneys from the poison as it passed out; we cannot, of course, assert that the medicine was of any more value than the water, but no deleterious effect was seen.

In those cases with loose movements an attempt was made to lessen the enteritis by giving bismuth salicylate, in doses of three grains, every three hours, sometimes combined with the subnitrate; in this we seemed successful. The salicylate was chosen first with the hope that its antiseptic properties might in some degree lessen the activity of the specific germs in the intestinal canal, but from the course of the cases we cannot see that our hopes were fulfilled.

Whiskey was given in almost every case. Without entering into a discussion as to the food value of alcohol or how it acts in fever, we feel sure that many of the cases were visibly benefited by it. The doses ranged from thirty drops for the younger children to one, or, in a few cases, two drachms for the older children every three hours. This was usually given at the same time with the milk, but sometimes the dose was divided, half being given before and half after the bath. In no case was it pushed until the odor could be detected on the breath.

Strychnine was occasionally used as a heart tonic, and one or two cases with heavily coated tongues received turpentine in five-drop doses. Children entering before the eighth day of the disease were given divided doses of calomel until a grain had been taken; after this constipation was overcome with the use of enemas.

Liquid diet was continued for ten days after defervescence was completed, the first addition to the diet being usually milk toast. Convalescence was aided by strychnine and Basham's mixture; if the tongue remained coated it was made to clear by No. 12 of the hospital pharmacopœia, a mixture of bicarbonate of soda and compound infusion of gentian. As soon as each patient was able to sit up he was sent to the country branch of the hospital, where the life out-of-doors in fresh air accomplished complete restoration to health.

In conclusion, our impression is that while typhoid fever may sometimes run a very mild or even abortive form in children, yet its clinical picture does not differ from that in adults in any essential feature save in the somewhat lower mortality.

THE FREQUENCY OF HEREDITY IN GASTRO-ENTERIC DISTURBANCES.

BY FRANZ A. R. JUNG, M.D.,
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GREAT stress is laid upon the heredity of tuberculosis, syphilis, or leprosy, but many questions are yet unanswered concerning the heredity of cancer and other diseases. Gout, diabetes mellitus, and obesity are considered a trio which can be followed through generations. The pedi-

gree of bleeders can often be traced to remote ancestry; and nervous disposition as well as mental diseases of ancestors are for many the cause of a gloomy view into the future. The pathological substrata of these hereditary troubles are yet unknown, and even the question whether the bacillus tuberculosis can be transmitted from parent to offspring has not yet been fully solved.

In order to avoid any difficulty or misunderstanding in dealing with these questions, science has introduced the expression "hereditary predisposition." Thus some people have a tendency to catarrh of the nose and throat; the same sensitiveness to "colds" was observed in their sires.

The scientific value of the expression "catching cold" was for a long time denied, until Birch-Hirschfeld, in his *General Pathology*, adduced sufficient proof of its substantial anatomico-pathological foundation.

If disturbances of circulation in the mucous membrane or anomalies of development are really traced in father and son, the same weakness of cells in one or several tissues will be common to both.

If in most nervous diseases the anatomico-pathological changes in the nerve cells have not yet been demonstrated it is to be believed that the deviation from the normal is an hereditary tendency. As a German proverb says, "The apple does not drop far from the tree;" certain laws must obtain in these cases, and no investigator dares to call it chance. Because science to-day is not advanced enough to prove this it does not follow that no light will ever be thrown upon this subject.

The expression "the power of resistance" of the organism has a complicated meaning. In the sense of Darwin's teaching this resistance is the principal factor in the survival of the fittest. At the same time another equally important factor is the "capacity for adaptation," which also is an expression for an aggregate of single facts. The study of the cell inheritance is left to the future.

Even the Roman fable teaches us that the health of the stomach was considered a factor of the first order and a principal condition of the general health. The organism, as a result of modified digestion, must needs show changes which are not visible to the naked eye. The fact that man can live a certain length of time without a stomach changes nothing. The man deprived of his stomach must succumb in the struggle for existence.

The comparative analysis of healthy stomachs, according to Boas' test meal, have shown a total acidity of 50 and 30 free HCl, together with a certain motile power and a capacity for absorption; yet a large number of people with strong deviation from this normal line do not at all consider themselves as stomach patients. They never think of consulting a specialist in order to obtain an analysis of the gastric juice. If they are ill they are treated on general principles, without special consideration of the peculiarities of their stomach, which they may have inherited from their ancestors.

The fact that I have often found in blood relations the same dyspeptic conditions induced me to study the question more closely in order to determine whether or not a direct inheritance not only of pronounced digestive troubles, but also of slight deviation from the normal, could be traced.

We speak of "predisposition," and are apt to forget that in such instances substantial pathological changes are inherited, which result in an organism with a much diminished power of resistance. If a so-called healthy stomach shows 35 total acidity only this deficiency must of necessity find expression somewhere in the system.

In order to change into useful values such terms as "disposition," "anomalies of constitution," etc., their single factors should be tested numerically. This opens a wide field to science in the future.

Unfortunately, the physician finds it difficult to persuade these so-called healthy people with an occasional disturbance of health to be thoroughly examined. Given the physiological limit of 20 to 40 free HCl, a person who is always in the vicinity of 20 cannot help being influenced by it. This is clear from a mathematical point of view. People with constantly low figures will suffer more easily from disturbances than those with an average of 30 free HCl.

On the other hand, people with 40 HCl, given the same conditions, will more readily develop superacidity.

The fact that these disturbances do not occur upon slight provocation is accounted for by the power of compensation. The personal description of the symptoms of a patient suffers usually from an involuntary want of precision.

In no histology do we see mention of a special nervous sensory apparatus of the stomach. The taste bulbs do not extend beyond the pharynx. A sweet, bitter, sour, or salty taste cannot be experienced by the stomach. Then there remain only painful sensation and tactile impressions which convey perception of temperature and pressure sensation.

Landois says, "It is highly probable that the sensory and tactile nerves possess different nerve endings."

The sensation of temperature, according to Landois, ceases in the œsophagus. Quincke states that even great variations of temperature are hardly perceived in the stomach. Pain, however, can occur wherever there are sensory nerves.

If, then, we have neither taste nor finer appreciation of temperature in the stomach, it follows that we can only perceive sensations of pain and pressure. This explains why patients cannot correctly describe sensations experienced in their stomachs.

If a strongly acid gastric juice gets into the œsophagus or reaches the tongue the patient can experience it. Further, if the pain is of a

pronounced character there is no mistake in the statement. A superacidity without eructation can only be described by the patient as a painful sensation, perhaps with the words "burning in the stomach." The other sensation is pressure, which can be caused by irritation of sensory nerves, with normal contents of the stomach, or by abnormal contents with normal nerves. A moderate degree of pain and sensation of pressure can so intimately pass into each other that they cannot be described accurately.

Einhorn has recently published six cases of achylia gastrica which presented all the symptoms of superacidity, with burning. Not only have I had similar cases under observation, but have heard patients describe a nervous subacidity and atony, with subacidity as superacidity.

In order to study the question of gastric analysis in blood relations, I have looked over the material at my disposal during the years 1898, 1899, and 1900. Analysis of test-meals showed the following:

| | | |
|--------------------------------------|----|-------------|
| Gastritis acuta | 1 | |
| Gastritis chronica | 58 | |
| Achylia gastrica | 18 | |
| Ulcus ventriculi | 5 | |
| Carcinoma ventriculi | 10 | |
| Enteroptosis (male) | | } 5 cases. |
| a. anacida | 0 | |
| b. subacida | 2 | |
| c. normalis | 1 | |
| d. superacida | 2 | |
| Enteroptosis (female) | | } 99 cases. |
| a. anacida | 1 | |
| b. subacida | 38 | |
| c. normalis | 40 | |
| d. superacida | 25 | |
| Gastropotosis (male) | | } 12 cases. |
| a. anacida | 1 | |
| b. subacida | 4 | |
| c. normalis | 0 | |
| d. superacida | 7 | |
| Gastropotosis (female) | | } 45 cases. |
| a. anacida | 1 | |
| b. subacida | 14 | |
| c. normalis | 9 | |
| d. superacida | 21 | |
| Gastroenterorrhœa hyperacida contin. | 9 | } 11 cases. |
| " " periodica | 2 | |
| Atonia ventricula | | } 64 cases. |
| a. subacida | 24 | |
| b. normalis | 23 | |
| c. superacida | 17 | |
| Ectasia | 8 | |
| Superaciditas nervosa | 83 | |
| Gastralgia-kenosis | 7 | |
| Gastralgia | 1 | |
| Regurgitatio nervosa | 2 | |
| Dyspepsia nervosa | 8 | |
| Neurasthenia a. anacid | 1 | } 42 cases. |
| b. subacid | 24 | |
| c. normal | 17 | |
| Enteritis acuta | 2 | |
| " chronic | 4 | |

| | |
|--|-----|
| Brought forward | 480 |
| Gastro-enteritis acuta | 1 |
| " chronica | 8 |
| Diarrhoea nervosa | 2 |
| Colitis membranacea hyperac. | 1 |
| " " | 3 |
| Dysenterica tropica | 1 |
| Atonia intestin. | 3 |
| Constipatio chronica | 13 |
| Ulcers recti | 1 |
| Colitis | 3 |
| Carcinoma recti | 1 |
| Cirrhosis hepatis | 2 |
| Carcinoma hepatis | 1 |
| Cholelithiasis | 7 |
| Perihepatitis | 1 |
| Stenocardia | 4 |
| Vitium cordis | 14 |
| Adipositas cordis | 1 |
| Arterio-sclerosis | 1 |
| Nephritis chronica | 3 |
| " hemorrhagica | 1 |
| Cystitis | 1 |
| Anaemia | 8 |
| " with hyperchlorhydria | 2 |
| Chlorosis | 4 |
| Hysteria | 6 |
| Epilepsia with atonia and subacid. | 2 |
| " " superacid. | 1 |
| Dementia paralytica | 1 |
| Obesitas | 1 |
| Rheumatismus | 1 |
| Arthritis urica | 4 |
| " deformans | 1 |
| Urticaria | 1 |
| Acne | 1 |
| Malaria | 2 |
| " perniciosa | 1 |
| Diverticul. cesophagi | 2 |
| Sarcoma sterni | 1 |
| Phthisis pulmon. | 6 |
| " intestin. | 1 |
| Typhus abdom. | 3 |
| Lues | 1 |
| Total number of cases | 608 |

In the order of frequency, the following table was arranged:

| | |
|---|----|
| Enteroptosis in women | 99 |
| Superaciditas nervosa | 83 |
| Atonia, both sexes | 64 |
| Gastritis chronica | 53 |
| Gastroptosis (female) | 45 |
| Neurasthenia with variable analyses | 42 |
| Achylia gastrica | 18 |
| Vitium cordis | 14 |
| Constipatio chronica | 13 |
| Gastroptosis (male) | 12 |
| Gastrosuccorrhoea | 11 |
| Carcinoma ventriculi | 10 |
| Ectasia | 8 |
| Dyspepsia nervosa | 8 |
| Anaemia | 8 |

These statistics show 166, or about one-third of all cases, to be hyperchlorhydria. This percentage is somewhat smaller than Einhorn's statistics for New York, or Hemmeter's for Baltimore. Kövesi, of Budapest, found the same. This author, in an article in vol. v., *Arch. für Verdauungskr.*, mentions 98 as the highest figure of total acidity, and thinks the figures of other authors—100 to 150—excessively high. My average figures are 80 to 110. One case came under my observation with the incredible figure 237, but this occurred only once.

Gastropptosis and enteropptosis occurred in 144 females and 17 males, or in 24 and 3 per cent., respectively, of all my cases. Boas has recently stated that atony, scientifically proved (Mathieu's method), is not nearly as frequent as is usually supposed. For the purpose of comparison I made the following observations: In Berlin I saw, within two weeks, 2 cases of atony among 70 cases at Boas' Poliklinik—about 3 per cent. In Washington I examined, in five weeks, 30 cases, according to Mathieu, and found 7 cases, with food remnants of over 200 c.c., making a percentage of 23.4.

During more extended observations these numbers will vary; so, for instance, for Washington it would be reduced to 10.7 per cent.

Having given a general outline of my material, I will now endeavor to give a short sketch of all those cases where I had the opportunity of examining blood relations.

The total acidity was determined by the usual method. The figures of free HCl have not been calculated into percentage, but are expressed in centimetres of $\frac{1}{10}$ NaOH as found in titration, and with dimethyl-amidoazobenzol as an indicator. The cases have been divided into two groups—

1. Those without similarity of analysis or disease.
2. Those with pronounced similarity of analysis, of disease, or of both.

CLASS I. *No Similarity.*

Family 1. (a) Father, aged sixty years, mitral insufficiency; has died since. Total acidity, 40; free HCl, 10; subacidity. (b) Daughter, chronic constipation and enteropptosis; total acidity, 44; free HCl, 24. (c) Son, aged about twenty-two years, complains of superacidity, mild constipation; total acidity, 80; free HCl, 45.

Family 2. (a) Mrs. C., woman in middle life; pronounced catarrh of nose and throat. Every morning her pillows are saturated with bloody mucus. The lower border of her stomach is five fingers below the umbilicus. Total acidity, 40; free HCl, 6; alizarin test, 30; enteropptosis, subacidity. (b) Daughter, nervous superacidity. Total acidity, 82; free HCl, 42.

Family 3. (a) Miss M., tuberculosis pulmonum; has died since. Total acidity, 40; free HCl, 16. (b) Brother, no tuberculosis; melancholia. Total acidity, 96; free HCl, 64; superacidity.

Family 4. (a) Mr. H., old gentleman, with asthmatic thorax. Total

acidity, 38; free HCl, 5; catarrhus ventr. chronicus. (b) Son, nervous dyspepsia. Has been cured of nervousness by hydrotherapeutics by Professor Winternitz. Total acidity, 63; free HCl, 30.

Family 5. (a) Mrs. C., medium-sized, under-nourished. Lower border of stomach three fingers below umbilicus. Right kidney palpable by one-half. Examination by diaphany. Total acidity, 80; free HCl, 48; enteroptosis, superacidity. (b) Sister of Mrs. C., subject to nervous superacidity. Total acidity, 50; free HCl, 15.

Family 6. (a) Mr. L., aged forty years. His brother, a physician, took him to Garfield Hospital, where he died of carcinoma of the lesser curvature. Tumor was palpable. No free HCl; lactic acid; autopsy. (b) Miss L., sister of deceased, enteroptosis; also scoliosis. Total acidity, 60; free HCl, 32.

Family 7. (a) Mrs. S., aged thirty years, globus hystericus, superacidity complaints, gastropotosis. Total acidity, 73; free HCl, 15. (b) Her brother, nervous, suffers from pseudo-angina pectoris. Total acidity, 62; free HCl, 48.

Family 8. (a) Mr. T., young man, with slight complaints of superacidity. Total acidity, 66; free HCl, 32. (b) His father, chorea as a child; even yet convulsive contractions of certain muscles; slight disturbances of articulation. Total acidity, 10; free HCl, 0; no ferments; achylia gastrica.

Family 9. (a) Miss B., severe enteroptosis, with acute exacerbations. Total acidity, 45; free HCl, 15; subacidity. (b) Father of Miss B., neurasthenia. Total acidity, 38; free HCl, 12; subacidity.

Family 10. (a) Miss G., suffers from constipation. Examination by diaphany, gastropotosis. Had typhoid fever in the course of the year. Total acidity, 54; free HCl, 30. (b) Miss G.'s brother, pronounced superacidity. Total acidity, 108; free HCl, 58.

Family 11. (a) Mrs. D. F. Total acidity, 3; achylia gastrica. (b) Daughter, enteroptosis with superacidity. Total acidity, 78; free HCl, 42.

Family 12. (a) Miss G., beginning enteroptosis. Total acidity, 60; free HCl, 30. (b) Brother suffers from malaria and dyspepsia. Total acidity, 53; free HCl, 26.

Family 13. (a) Mrs. S., of French descent; highly nervous. Total acidity, 30; free HCl, 0; subacidity nervosa. (b) Son, very nervous; suffers from superacidity. Total acidity, 90; free HCl, 40. (c) Miss S., floating right kidney, fourth degree; severe enteroptosis; emaciated person; no analysis.

Family 14. (a) Miss S., ansemia. Total acidity, 64; free HCl, 34. (b) Her brother, with nervous dyspepsia, complains of superacidity. Total acidity, 60; free HCl, 32. This family might be included in the next class, which deals with cases of blood relations *with similarity*.

CLASS II. *Similarity.*

Family 1. (a) Mr. D., supposed to have a sound stomach. Analysis showed total acidity, 16; no ferments; achylia gastrica. (c) Miss D., sister, has had stomach trouble for years; never yet found permanent relief. Total acidity, 10; no ferments; achylia gastrica.

Family 2. (a) Mr. M., artist by profession; subject to petit mal. Total acidity, 72; free HCl, 52; superacidity. (c) Mrs. M., mother

of artist, aged about seventy years; nervous; has suffered from digestive disturbances over a decade. Soup and starvation diet were tried in vain. Total acidity, 96; free HCl, 56; superacidity.

Family 3. (a) Mrs. W., woman in middle years, of slight frame, very thin, lower border of stomach two fingers below umbilicus. Right kidney movable; very thin abdominal walls; mother of seven children. Various rest and fat cures have been resorted to without avail, since patient seems unable to retain permanently the flesh gained during treatment. Backache and pain in right side; dyspeptic. Report from gynecologist: prolapsus uteri et ovarii dextri. Analysis, total acidity, 20; free HCl, 0; enteroptosis, subacidity. (b) Miss W., oldest daughter; medium-sized, quiet temperament, slight backache, first degree of enteroptosis. Total acidity, 38; free HCl, 16; subacidity. (c) Miss I. W., second daughter; same type as mother; nervous temperament, seems unable to gain in weight. Right kidney distinctly palpable. After violent exercise on horseback, kidney comes down much lower. Splashing stomach, lower border one finger below umbilicus. Chronic constipation. Total acidity, 48; free HCl, 17; subacidity, enteroptosis. (d) Mrs. E., sister of Mrs. W. Quite contrary to her sister, very stout, colitis chronica, gastroptosis; stomach two fingers below umbilicus. Test-meal food remnants, 220 c.cm. Total acidity, 90; free HCl, 25; gastroptosis, atony, colitis, superacidity. (e) Miss E., daughter of Mrs. E.; type of her cousin, not her mother; slender, less than medium height, nervous, anæmic. Total acidity, 50; free HCl, 14; alizarin value, 40; subacidity. (f) Miss E., niece; brother's child; chronic severe anæmia, palpitation, dyspepsia. Total acidity, 52; free HCl, 18; alizarin, 40; subacidity.

Family 4. (a) Miss F. For six years continual loss of weight. Headache, with nausea, severe constipation, amenorrhœa. Examination by gastrodiaaphany shows stomach three fingers below umbilicus; right kidney palpable. Total acidity, 88; free HCl, 43; enteroptosis, superacidity. (b) Miss F. II., younger sister; colitis membranacea, erosiones ani. (c) Miss F. III., enteritis chronica.

Family 5. (a) Mr. W., nervous young man; complains of burning in stomach, weakness, anxiety; afraid of appendicitis. Total acidity, 90; free HCl, 36; superacidity. (b) Mrs. W., mother of young man; chronic headache since childhood; has sudden sensations of hunger, vomiting of bile; fears to have inflammation in region of stomach; right kidney movable; lower border of stomach three fingers below umbilicus. Total acidity, 82; free HCl, 36; enteroptosis, superacidity.

Family 6. (a) Mr. S., man in middle life; has suffered for ten years from gastrosuccorrhœa, superacidity periodica; 300 c.cm. of clear gastric juice often present. Total acidity, 100; free HCl, 72; superacidity, gastroptosis. (b) Mrs. M., his sister; lower border of stomach five fingers below umbilicus. Total acidity, 32; free HCl, 16; gastroptosis, subacidity.

Family 7. (a) Miss R.; lower border of stomach almost at symphysis. Total acidity, 63; free HCl, 22; enteroptosis. (b) Miss J. R.; lower border of stomach two fingers below umbilicus. Total acidity, 70; free HCl, 36; kidney not movable; gastroptosis.

Family 8. (a) Colonel H., a neurasthenic old gentleman; has been repeatedly in the hands of nerve specialists in Europe; insomnia. Total acidity, 38; free HCl, 7; lower border of stomach two fingers

below umbilicus; gastropptosis, subacidity. (b) Miss H., daughter; nervous. Total acidity, 40; free HCl, 10; subacidity.

Family 9. (a) Miss A.; anæmic, chronic headache. Total acidity, 30; free HCl, 5; gastropptosis, subacidity. (b) Mr. A., brother; pronounced gastropptosis, arrhythmia. Total acidity, 64; free HCl, 33. (c) Mrs. A., mother; chronic headache for number of years; marked gastropptosis. Total acidity, 80; free HCl, 64; superacidity.

Family 10. (a) Miss M., teacher; enteroptosis; has been treated for years for displacement of uterus and ovaries. Total acidity, 54; free HCl, 27. (c) Mrs. M., mother; gastropptosis, fissura ani, gastro-enteritis chronica. Total acidity, 37; free HCl, 2; subacidity.

Family 11. (a) Mrs. P.; gastropptosis; examined by diaphany; hemorrhoids. Total acidity, 54; free HCl, 12; subacidity. (b) Miss P., daughter; pronounced enteroptosis. Total acidity, 48; free HCl, 24.

Family 12. (a) Miss G., examined by diaphany; enteroptosis; superacidity; enteritis chronica. Total acidity, 99; free HCl, 56. (b) Miss G., sister. Total acidity, 82; free HCl, 28; slight gastropptosis, superacidity.

Family 13. (a) Miss W.; enteroptosis; right kidney movable. Total acidity, 72; free HCl, 41; superacidity. (b) Mrs. W.; constipation; hyperchlorhydria. Total acidity, 78; free HCl, 42.

Family 14. (a) Miss H. P.; enteroptosis; examination by diaphany. Total acidity, 67; free HCl, 32. (b) Miss E. P.; enteroptosis; right kidney freely movable. Total acidity, 40; free HCl, 20.

Family 15. (a) Prof. B.; pronounced symptoms of superacidity. Total acidity, 96; free HCl, 60; alizarin value, 86. (b) His brother, editor of daily paper. Total acidity, 72; free HCl, 52; alizarin, 46; superacidity. It is an interesting fact that Prof. B.'s wife also suffers from superacidity. Total acidity, 70; free HCl, 36; alizarin, 64.

Family 16. (a) Miss N. Total acidity, 50; free HCl, 35; constipation, enteroptosis. (b) Miss J. N. Total acidity, 45; free HCl, 20; acne, slight gastropptosis. (c) Father; enteritis chronica.

Family 17. (a) Mr. P.; chronic constipation. Total acidity, 50; free HCl, 30. (b) Miss P.; constipation, slight gastropptosis, superacidity. Total acidity, 72; free HCl, 36. (c) Miss P., cousin; severe chronic constipation, superacidity. (d) Miss P., sister; enteroptosis of fourth degree.

Family 18. (a) Miss McK.; chlorosis, chronic headache. Total acidity, 36; free HCl, 18; alizarin, 10; subacidity. (b) Miss McK., sister; anæmia. Total acidity, 64; free HCl, 20. (c) Mr. McK., father; repeated attacks of malaria, anæmia; analysis not made.

Family 19. (a) Mrs. von D.; catarrhus chronicus. Total acidity, 30; free HCl; alizarin, 19. (b) Mrs. F., sister; suffers from enteroptosis. Total acidity, 36; free HCl, 10; subacidity.

Family 20. (a) Miss F.; chronic constipation. Total acidity, 45; free HCl, 20. (b) Mr. F., brother; chronic constipation for eighteen years. Total acidity, 47; free HCl, 24.

Family 21. (a) Mr. E., young man of large frame, wanting in energy. Total acidity, 72; free HCl, 40; superacidity. (b) Mrs. E.; acute peristaltic disturbances after excitement. Total acidity, 54; free HCl, 40. (c) Mr. E., eldest son; subject to neuralgia, superacidity; leads a gay life as a student. Total acidity, 80; free HCl, 50.

Family 22. (a) Mr. M.; suffered for many years from pain in region

of gall-bladder; the latter is very sensitive on pressure; chronic colitis. Patient rarely has severe attacks of pain; smokes and drinks heavily. Total acidity, 72; free HCl, 34; alizarin, 48. Repeated attacks of icterus, superacidity. (b) Miss M., daughter; same complaint as father, only pain more frequent in region of gall-bladder. At times acholic stools. Total acidity, 60; free HCl, 30; alizarin, 49; cholelithiasis.

Family 23. (a) Mrs. S., widow of a physician; very energetic woman, who inclines to overwork and then becomes nervous; lower border of stomach three fingers below umbilicus. Total acidity, 104; free HCl, 60; gastropotosis, superacidity. (b) Mrs. M., younger sister of Mrs. S.; very nervous in her youth; both ovaries, although healthy, were removed; symptoms remained the same. Total acidity, 86; free HCl, 34; enteroptosis, superacidity. (c) Miss S., daughter of Mrs. S.; anæmic; bruit de diable; chronic headache; lower border of stomach one finger below umbilicus. Total acidity, 78; free HCl, 26; mild gastropotosis, superacidity.

Family 24. (a) Miss B., young lady of strong physique. After violent gymnasium work develops symptoms of enteroptosis; lower border of stomach one and a half hand below umbilicus. Examination with gastroduaphany shows an almost vertical position of the stomach. Right kidney movable; right ovary displaced downward and highly sensitive. Gynecologist proposed operation, which was refused, and a successful "mast kur" was made instead. Total acidity, 46; free HCl, 23; enteroptosis. (b) Sister; dyspeptic complaints after an attack of enteritis. Total acidity, 44; free HCl, 24. (c) Second sister; tachycardia; lower border of stomach one finger below umbilicus. Total acidity, 46; free HCl, 14; mild gastropotosis, superacidity.

Family 25. (a) Miss H.; severe anæmia; very slender figure; enteroptosis; repeated operation for retrodisplacement of uterus; operation for fixing kidney. Achylia gastrica. Total acidity, 8; no ferments. (b) Mrs. H., cousin, fathers were brothers, same habitus; acute enteroptosis after being thrown during a runaway; weight, 90 pounds; has made repeated rest cures. Total acidity, 24; free HCl, 0. Alizarin, 16. Patient improves until total acidity 36, free HCl 16, is reached. Operation for fixing kidney. Prolapsus and enlargement of the uterus; congested ovaries; subacidity. It is interesting to note that cases c and d, sister and brother of Mrs. H., represent quite another type. Both are large, corpulent, weighing over 170 pounds. (c) Mrs. L. Total acidity, 78; free HCl, 46; superacidity. (d) Mr. H. Total acidity, 80; free HCl, 36.

Family 26. (a) Mr. D., a middle-aged man; chronic constipation; superacidity; attacks of vertigo. Total acidity, 75; free HCl, 50. (b) Daughter of Mr. D.; enteroptosis; lower border of stomach four fingers below umbilicus. Right kidney movable. Total acidity, 96; free HCl, 50; sensitive colon; constipation, superacidity.

Thus we have in toto 40 families, representing 597 cases—14 families without similarity (35 per cent.); 26 families with similarity (65 per cent.).

ENTEROPTOSIS AND GASTROPTOSIS. Gastropotosis, instead of enteroptosis, is differentiated whenever the stomach alone is displaced downward. Among the families *with similarity* there are 6 families with one

case of enteroptosis each, and 2 families with one case of gastropptosis each. Among the families with similarity there are 2 families with enteroptosis in at least two members of each separate family. There are 9 families in which one member has enteroptosis and one has gastropptosis. Two families show gastropptosis in two members. In 5 families enteroptosis is found but once in each. One family has only one member with gastropptosis. If we consider enteroptosis and gastropptosis as the same clinical picture we find it in several members in 13 out of 40 families, which would appear to justify the conclusion that enteroptosis in one-third of the cases is a family peculiarity. Two hypotheses are admissible as an explanation—either enteroptosis and its tendency is inherited, or similar modes of living produce enteroptosis in different members of the same family.

Among 40 families, 27, or 67 per cent., show cases of enteroptosis. Of the 27 families there are 13, or 48 per cent., families with enteroptosis in various members. In 5 families mother and daughters have enteroptosis. In 8 families sisters and cousins have enteroptosis. In 3 instances (families Nos. 6, 23, and 25) the various members live in different towns; also family No. 16 most of the time. Therefore in 3 families the conditions of life differ, and cannot be taken as an etiological factor.

In families Nos. 3, 24, 25, cases of enteroptosis give a history of violent concussion. In the same families, however, are found other cases of enteroptosis without a traumatic history. Cases Nos. 3 and 23 seem to point strongly to the theory of inheritance. In both families enteroptosis is found in two mothers (sisters) and their children, but the conditions of life are entirely different.

HYPERCHLORHYDRIA. Among 14 families without similarities of symptoms there are 10 families with superacidity. The 26 families with similarity as studied above show 10 families with repeated cases of superacidity, 5 families with one case each, and 11 families in which it is not present.

In a total of 40 families we find 25, or 62.5 per cent., with superacidity; of these in 10 families, or 37.5 per cent., several members were afflicted. Of the 25 families with superacidity 40 per cent. had more than one case in the same family. Of the 10 families showing several cases of superacidity in the same family we have 5 where it occurred in parents and children, and in the other five families in sisters, brothers, and cousins.

In 5 of the 25 families the members suffering from superacidity live in different surroundings and circumstances.

SUBACIDITY. Subacidity is found in 8 out of 14 families which showed no similarity of clinical picture. In one instance it is found among several members of the same family, and yet all were suffering

from entirely different diseases. Among the 26 families with similarity subacidity occurs in 10 families—in 3 families repeatedly in different members; in 6 families 1 each; in 1 family with achylia gastrica.

In a total of 40 families we find 19 families with subacidity—47.5 per cent. In 4 families out of the 40 it is found repeatedly in 10 per cent. Out of 19 families with subacidity it is found repeatedly in 4 families—21 per cent.

ACHYLIA GASTRICA. Among the families without similarity we find achylia twice, but no repeated cases in the same family. In families with similarity we find achylia gastrica in 2 families—in 1 family in several members and in another family with subacidity.

NORMAL ANALYSIS. A total acidity of 40 to 65 was considered normal, but the free HCl must be 20 to 40. Fourteen families without similarity show 7 families with normal figures. Among these 7 families occurred two with normal figures in two of its members, yet the symptoms and complaints are absolutely different. In one case (No. 14) showing a normal analysis the complaints are those of superacidity. The 26 families with similarity include 12 families with normal figures. Four of these 12 families have several members with normal analyses; 19 normal figures—47.5 per cent.; 6 families in several members normal analyses—15 per cent.; 6 families out of 19 have repeated cases of normal analyses—31.5 per cent.

We further find in these families repeatedly:

| | | | | | | |
|------------------------|---|---|---|---|---|----------------------|
| Anæmia in family No. 3 | . | . | . | . | . | Two cousins. |
| " " " 18 | . | . | . | . | . | Father and daughter. |
| Constipation " " 17 | . | . | . | . | . | " " " |
| " " " 20 | . | . | . | . | . | Brother and sister. |
| Cholelithiasis " " 29 | . | . | . | . | . | Father and daughter. |

RÉSUMÉ.

| | | | | | | | |
|--------------------------|---|---|---|---|---|---|--------------|
| Enteroptosis in families | . | . | . | . | . | . | 67.5 per ct. |
| Supracidity " | . | . | . | . | . | . | 62.5 " |
| Subacidity " | . | . | . | . | . | . | 47.5 " |
| Normal acidity " | . | . | . | . | . | . | 47.5 " |

Repeated cases in the same family:

| | | | | | | | |
|----------------|---|---|---|---|---|---|--------------|
| Enteroptosis | . | . | . | . | . | . | 32.5 per ct. |
| Supracidity | . | . | . | . | . | . | 37.5 " |
| Subacidity | . | . | . | . | . | . | 10.0 " |
| Normal acidity | . | . | . | . | . | . | 15.0 " |

| | | | | | | |
|--|---|---|---|---|---|--------------|
| In 27 familes of enteroptosis we find repeated cases | . | . | . | . | . | 48.0 per ct. |
| " 25 " supracidity " " | . | . | . | . | . | 40.0 " |
| " 19 " subacidity " " | . | . | . | . | . | 21.0 " |
| " 19 " normal acidity " " | . | . | . | . | . | 31.5 " |

Enteroptosis is most frequent, but supracidity presents the largest number of cases in the same family. *Of 40 families 23 (57.5 per cent.) have several cases in one family of their characteristic analyses. In other*

words, one family out of every two presents more than one case of digestive disturbance of the same nature.

The average frequency of duplicate cases of enteroptosis, superacidity, subacidity, and normal acidity lies between 27 per cent. and 48 per cent., and is consequently 34.5 per cent.

TUBERCULOUS PERICARDITIS:

FOLLOWED BY REMARKS UPON PARACENTESIS AND INCISION.

BY BEVERLEY ROBINSON, M.D.,
OF NEW YORK.

Two cases of this somewhat rare disease have been under my care within the past eighteen months. In both cases autopsies were obtained—the one complete, the other only embracing the examination of the heart and pericardium. In one of these instances the pericardium was aspirated several times; in the other it was not considered necessary or judicious to operate. The history of my first case was read at a meeting of the Practitioners' Society,¹ October 11, 1901, and the specimen shown. The case was discussed by the members.

During the past eighteen months I have also taken care of two other cases of pericarditis. Of these, one occurred as a complication of Bright's disease; the other apparently of rheumatic origin. I shall refer to these later in my remarks upon paracentesis.

In certain instances of tuberculous pericarditis it has been noted that there was no evidence of tuberculous deposit in the adhesions which were present. In those instances more numerous, where tubercles are discovered in the adhesions, they may appear like grayish areas at the line of union of the parietal with the visceral layer. In 1048 autopsies, Wells² found tuberculous pericarditis 10 times, which formed about 8 per cent. of all cases of pericarditis recorded by him. It is not limited to adults, but may occur quite frequently in childhood or infancy. It is more frequent among men than women. Baginsky reports 15 cases in 4500 autopsies, and of these 4 were purulent. In Osler's autopsies, 1000 in all, there were 7 instances of tuberculous pericarditis.³

Welch, in his report from Johns Hopkins Hospital, states he has seen 6 cases. Although often spoken of as frequent, it is not shown by references in *Index Catalogue*, *Transactions of the Pathological Society of London*, etc. Up to 1893 Osler had seen only 17 cases, and yet he writes tuberculous pericarditis follows hard upon the rheumatic form.

¹ Medical Record, November 23, 1901, p. 831.

² Journal of the American Medical Association, May 25, 1901.

³ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1893, p. 20.

In Wells' cases, where the condition was miliary and chronic, there were adhesions and no fluid. In acute miliary eruption, and in those where there was caseous deposit,¹ there was also more or less effusion in the pericardial sac.

In one instance, where no tubercles were shown in a case of acute pericarditis, the inflammation was apparently due to toxins of tubercular origin. This opinion was supported by the fact that tubercles were found extensively in other viscera. The tuberculous cases, especially those which are acute, result fatally. This seems to be true, also, of acute pericarditis following pneumonia or Bright's disease, but is not true of this complication of acute articular rheumatism.

The forms of tuberculosis as they are found in the pericardium are either of miliary form or cheesy masses. When effusion exists it is serous, bloody, or purulent. It may be moderate or considerable in amount. Whenever the condition has existed for some weeks, it is probable that the pericardial sac becomes softened and dilated and offers a very insufficient support to the heart. The clinical evidence of this softening and dilatation of the pericardial sac in disease is shown by its greater capacity to contain fluid. Experimentally, this capacity is limited to about 700 c.c. of liquid when forced into the sac.² In disease we know much greater quantities of fluid may be contained. Even when these larger amounts are present we can only recover them in part by paracentesis, owing, as will be shown later, to the position of the heart in the fluid. Soon the heart would show signs of dilatation, and this condition more surely and rapidly occurs if the heart walls are subjected to any increased strain.

In some instances of tuberculous pericarditis, as we know, adhesions with the chest wall have developed. These adhesions, if more than usually taxed, are apt, sooner or later, to be stretched and in some instances to give way. Under these circumstances the heart shows signs of insufficiency very soon.

As Sequira³ points out, the dilatation of the pericardium is very important from the stand-point of ultimate prognosis, and this we can readily appreciate when we consider the immediate effects of hypertrophous dilatation where passive congestion of the viscera is more than likely to occur. Sequira's observations are based upon the history of 130 cases of acute pericarditis and observations of 1000 cases.

In occasional instances the pericarditis seems to be due to mere extension from adjacent parts in which tuberculous lesions clearly exist. Under these conditions the pericarditis, curious to say, may occasionally be simply inflammatory. This point is affirmed by Osler. Such instances have also been noted where the extension came from a case

¹ My second case.

² Chatin. *Revue de Méd.*, June 10, 1900.

³ *British Medical Journal*, June 17, 1900.

of non-tubercular pleuritis. Likewise they have been recognized as final complications of chronic tubercular states and as a result of terminal bacteraemia. (Wells.)

In certain instances where the process in the pericardium is an acute one, tuberculosis has not had time to develop there, but later we should doubtless have discovered it if death from other organs affected with tuberculosis had not occurred. Tuberculous pericarditis heals through the formation of fibrous adhesions. In some forms, especially the caseous, the healing may be accompanied with calcification. The thickening of the sac wall may become very considerable. This is true of the parietal wall particularly. These cases are often accompanied with a deposit of numerous miliary tubercles. At the same time there is present more or less effusion, which may present different characters. Often it is distinctly bloody. Still this is not sufficient to determine the diagnosis, as it may take place in other conditions. Of the 11 cases found in literature by Sears,¹ 6 occurred in scurvy, 3 in rheumatism, 1 in goitre, 1 idiopathic, in an alcoholic subject.²

Bacilli may be found in this fluid, although sometimes it requires several careful examinations to reveal them.³ Osler reports 1 case, quoted from Kast, in which tubercles have been found in the pericardial effusion. F. C. Shattuck's⁴ second case is another. Where this examination has remained negative the bacilli are revealed solely by means of animal inoculations.⁵

The proportion of successful inoculations is considerable. It is of great value in fixing the positive diagnosis to be made. The drawback to it is the time it takes to develop tubercles.⁶ It is in the exudate, as well as in layers of pericardium, that we find tubercle. When it occurs in pericardial layers it oftener affects the parietal one and disseminates to the left pleura. Serous effusion, when present, is frequently surrounded by fibrous exudation, which in places becomes adherent.

The myocardium may be affected with tubercular infiltration at the same time as the pericardium. The miliary form is relatively unimportant as compared with the caseous. The latter penetrates deeper and sometimes perforates the cardiac walls, and may surround itself with a fibrinous clot.

Formerly primary cases of tuberculous pericarditis were reported. To-day, thanks to a more advanced knowledge of pathology, this erroneous affirmation is rarely met with. One reason is because lesions

¹ Boston Medical and Surgical Journal, 1898, p. 293.

² Churtan. *THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, 1892, p. 84; also, Michailoff, loc. cit., 1878, p. 278

³ Report of my first case.

⁴ Transactions of the Association of American Physicians, vol. xii. p. 194.

⁵ This was true in my second case.

⁶ About six weeks in guinea-pig.

formerly considered of doubtful nature are now recognized to be tuberculous. Hence, when such lesions are found elsewhere and by their structure are known to be of old date, we can readily appreciate that it is from them that the infection of the pericardium proceeds. In very many cases, without doubt, the infection is carried by the bloodvessels; in others the lymph channels are the evident way of transport.

Simple carrying, as it were, by extension of a tuberculous process through contact, although still admitted, is not so frequently allowed as heretofore. The ordinary development of tuberculous pericarditis is to run a subacute or chronic course. This course may be such an insidious one that for a long while the disease is latent and no obvious symptoms indicate its presence. Indeed, Osler reports only a single case in which, during life, "the diagnosis of tuberculous pericarditis was made with a reasonable degree of probability." If, however, there is no rheumatic history and tubercles are discovered elsewhere, with previous symptoms of pericarditis, we should think of the tuberculous form. The protracted course of the disease and the more marked irregularities of temperature may indicate its presence. Sometimes the symptoms occur, but are concealed by grave symptoms of other organs which are affected from general miliary tuberculosis as well as the pericardium. It is a fact also worthy of remark that, as a result of experimental researches about tuberculosis, while lungs, pleuræ, and mediastinal glands are usually attacked, it is a very infrequent circumstance to find the pericardium involved; indeed, it is doubtful if there be any well-authenticated cases of it. The involvement of the pericardium through the lymph channels and with transport of tubercle bacilli largely comes from the lungs, pleuræ, and peritoneum. This infection of cardiac lymphatics is doubtless carried on indirectly, so far as the former organs are concerned. Owing to the free anastomosis of the lymph vessels across the diaphragm, it is readily understood how a tuberculous peritonitis may cause directly the development of tuberculous pericarditis. Where the tuberculous pericarditis is of caseous form, brought from mediastinal glands, it is probable that later on this matter is carried to and produces secondary infection of the lungs and pleuræ.

In regard to the question of the frequency with which one layer of the pericardium is affected as compared with the other, this, it appears to me, is difficult of solution, despite the statement of Wells, and in any event of no practical moment.

What is affirmed with authority is that a large proportion of cases show tuberculous deposit in mediastinal lymph glands and pericardium at about the same time, and frequently this is true where no tuberculous deposit exists elsewhere. We are more likely to notice this dual condition where already the process has become somewhat a chronic one. Effusion into the pericardium is more likely to occur, it is stated,

where caseous masses exist on its surface than where we find the small gray tubercles.

Where the myocardium is affected the disease comes primarily, as a rule, from the pericardium. The muscle of the heart is, however, rarely thus diseased. This is especially true of the ventricle; the auricle is oftener invaded.

Tuberculous pericarditis may recover, it is stated. This is rare. In general, we may say it is not directly fatal, and death subsequently occurs from tuberculous deposit in some other important organ. At this time, that of the pericardium may have become quiescent. This, of course, occurs only in the relatively chronic forms. If it does, we may find fibrous nodules in the exudate or pericardial walls as the only direct evidence of a tuberculous lesion. It may be inferred, however, by considering the evident tuberculous condition of the mediastinal glands, and through the fact that this is the sole efficient cause of tuberculous pericarditis which can be brought to light.

The most important effects, clinically, are those which are seen upon the heart. In some instances there is no valvular lesion or structural defect of the heart muscle, and yet there is dyspnoea, cyanosis, and weakness of pulse—all pointing to cardiac distress. The effusion by its mere pressure seems to be the explanation of these symptoms, and its prompt removal is, therefore, the essential and urgent indication, so as to re-establish, as far as may be necessary, functional power. If this condition is allowed to remain, even though life may not be imminently imperilled, the heart soon shows the results of the external pressure against which it contends by becoming enlarged with hypertrophous dilatation, and by growing weaker progressively and constantly. Where adhesions exist with the thoracic walls, which is often true in these cases whenever they tend to chronicity, the duration of life is notably shortened. It is equally true, if not more so, where the two layers of the pericardium have also become adherent and the effusion has become resorbed or been removed by paracentesis. In these instances life may be measured by a few months. This is eminently true in pericarditis affecting children. Lee Dickinson¹ reports "one solitary case in which adhesion, certainly contracted in childhood, proved ultimately harmless." The late Dr. Sturges showed in these cases that acute carditis is present, usually of rheumatic origin. Peck² has ably described in several such cases a condition of "pericarditic pseudocirrhosis" resembling precisely the *mixed form of cirrhosis*³ of the liver, which is due to a latent pericarditis. The differential diagnosis is based upon: (1) Absence of

¹ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1896, p. 692.

² Ibid., 1896, p. 221.

³ A case of pseudo-atrophic cirrhosis of the liver, secondary to adherent pericardium, is reported by Dr. R. H. Babcock, of Chicago, in the Medical News, December 14, 1901, p. 924.

etiological factor of cirrhosis; (2) a history of pericarditis supported with the evidences of it from physical signs.

We should not ignore the difficulty of explaining why pericardial adhesions are occasionally followed by such changes. Wells gives several good and sufficient reasons why tuberculous synechia is less to be feared than that which occurs in purely rheumatic cases. Among these may be regarded slowness of growth, no valvular defects, no toxæmia affecting heart muscle. Moreover, he states heart dilatation occurs in rheumatism at period of inflammation or later, when effusion is being absorbed.

In the latter instance, as I have already pointed out, the pericardium becomes dilated and softened, and thus fails to give the heart proper support. In addition to this, however, if the pericardium is attached to the chest walls it simply cannot follow the heart, owing to mechanical conditions. For this reason again, the heart yields more and more to internal pressure, and consequently becomes more and more dilated, weak, inefficient, and ultimately powerless.

In these cases we often find beneath a very thick pericardium a considerable growth of connective tissue, together with a deposit of fat in the superficial parts of the heart muscle.¹ In rare instances are we able to demonstrate the existence of gray tubercles in the myocardium. Where caseous deposits are found at the autopsy they are more important because they have penetrated the heart wall deeply, and in the event of perforation they serve to explain the transport of numerous bacilli in other organs and the signs of general miliary tuberculosis, which were easily recognized, even during life.

The usual termination of tuberculous pericarditis is death. This may be brought on directly by the recurrence of a large effusion, frequently hemorrhagic, which repeated tapping has failed to relieve. Of course, at times the adhesions which have formed to chest walls, lungs, and diaphragm seem greatly to shorten the duration of life; but these again may never occur, and the heart may simply float freely in the fluid which surrounds it more or less on all sides.² Often, as I have said, the fatal ending is immediately attributable not to the heart itself, but to the general miliary tuberculosis of different viscera with which the cardiac condition is associated.

Death may occur from an embolism, but this is extremely rare.³ "Tuberculous pericarditis is generally unaccompanied by any symptoms referable to the heart, and is almost always an autopsy finding."⁴ With this statement of Wells, corroborated by Osler,⁵ judging by my two recorded cases, I should be inclined to differ.

¹ Second case reported by me.

² Case 8, reported by Wells.

³ Already cited.

⁴ See my first case—pathological report.

⁵ Loc. cit., p. 1458.

For the cases which are slowly and insidiously developed there is little or no local treatment to be advocated. This is true of the cases which terminate in synechia and where the symptoms are frequently very obscure. The systolic retraction of the nipple in these instances is a deceptive sign, as more than once I have been able to observe. In this connection Lee Dickinson states we may have pulling in of lower ribs with systole, but adds that cases with this indication are seldom available. The most characteristic of all indications "are rapid progress of the case to the fatal end and the signal failure of all known means of relief in heart disease."¹

Inasmuch as there is small effusion and no obvious inflammation, these two conditions may not require active interference. In those instances in which the onset of the disease is acute it is indicated at first to limit the amount of inflammation. Personally, I favor the local use of poultices or hot flannel stupes. To the former mustard may be added; the latter may be sprinkled with turpentine. Equal parts of soap liniment and turpentine applied for several hours on warm flannel are recommended. If cold be employed I much prefer Leiter's coil to an ice-bag. The latter is difficult to keep in place and causes distress from cold at times, which we cannot regulate easily.² A small quantity of morphine hypodermically may be called for to allay pain. In the cases that I have seen I have not thought the cautery or blisters were required in the beginning of the attack. Later, I am confident, repeated small fly-blisters may limit the amount of the exudate formed. It is also possible that an effusion already formed may thus be made to disappear, in part or wholly, and more rapidly than it otherwise would. Despite what has been said, it is far wiser in many cases not to attempt to influence the pericardial condition by any local applications. Whenever the effusion becomes considerable or excessive, paracentesis should be thought of. If the lips and fingers become cyanosed—if there is marked dyspnoea, and weak, frequent, irregular pulse—prompt withdrawal of fluid is necessary to preserve life. If before paracentesis is performed the exploring needle shows the presence of pus, it is more judicious, in many instances, to incise the sac freely and introduce a drainage-tube. Of course, if the condition of the patient is imminent, paracentesis may first be performed and subsequently the more radical operation.

The position of the heart with respect to pericardial effusions is frequently undetermined. In purulent fluids the heart is supported by or floats upon the fluid, and is carried directly against the chest walls. This has been proven experimentally. In fluids containing some blood,

¹ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1896, p. 696.

² Of course, we can put a towel or flannel compress underneath.

or in pure serum, the heart is surrounded by fluid, as a rule, of which the larger quantity is in the lower and left lateral portion of the pericardial sac.

Whenever puncture of the heart is indicated, some hesitancy arises in performing the operation on account of the danger incident thereto. There is risk of wounding the mammary artery, of entering the left pleural cavity, of puncturing the liver, of wounding the peritoneum, or, indeed, of going through the diaphragm. Perhaps the latter is of no special moment, as it is the way by which entrance to the pericardium is occasionally suggested or taken by the surgeon in a radical operation.

The classical site for aspiration of the pericardium may be regarded as the left fifth interspace, near the sternal margin.¹ Other regions, however, have been tried more or less successfully, *i. e.*, beyond the left nipple and near the outer line of cardiac dulness; in the right fourth intercostal space; in the angle of the xiphoid cartilage and the margin of the left costal arch. The right interspace is specially indicated by Dr. Rotch, of Boston, in those cases where he has found dulness over this area.² Dr. F. C. Shattuck, in his able paper before the Association of American Physicians, Washington, 1897, has tried different points for aspiration of pericardium, with satisfactory and sterile results.

On one occasion where a small quantity of fluid was withdrawn, later the autopsy showed at least one pint of fluid in the sac. It may be inferred in such cases that the fluid thus discovered was produced during the agony or after death. It seems to me more probable that it was in the pericardial sac during life, but could not be aspirated, on account of the position of the heart and the wrong point of puncture.

In this connection I am glad to be able to refer to a recent able article by Dr. O. Damsch,³ which settles definitely, it may be, most points hitherto questionable about the position of the heart, that of the effusions, and the point where we should always try paracentesis. Dr. Damsch made injections experimentally into the pericardium, the subject being in the upright position, for the purpose of determining the position assumed by the pericardial exudates. *Small amounts*, he found, collect in the lower and antero-lateral portions of the pericardium, causing approximation of the right anterior portion of the pericardium to the chest wall. The heart, when of *normal size*, was always found pressed against the posterior portion of the pericardium, the fluid occupying the

¹ Roberts states (Trans. Surg. Assoc.) that this point of election "will not assure safety to the pleura."

² The clinical conclusion from Damsch's experiments was that "first sign of pericardial effusion would be area of dulness in heart-liver angle, as taught by Rotch." (Loc. cit.)

³ Gould's Year-book, 1901, pp. 178-179.

anterior portion. In cases in which the heart was hypertrophied it was pressed *upward* and *anteriorly*. Its increased size, according to Damsch, causes it to fill the whole space between the anterior and posterior chest walls, and, therefore, since the fluid collects in the lower part (pericardium), the heart must be pressed upward. "From his experiments he decides that the best position for *puncture* is well down toward the lower part of the pericardium." He also decides that puncture in the fifth or sixth intercostal space (left) next to the sternum, directing the trocar somewhat inward, is the safest method and location of paracentesis. Thus performed there is no danger of wounding either heart or pleura. If the heart is normal the fluid is in front of it and at lower part of pericardium; if the heart is *enlarged*, which it usually is, it is floated *upward* and *out of danger*.

Aspiration of the pericardium relieves, without doubt, for a while imminent symptoms, and, as an operation of urgency, should be employed unquestionably. When we come to consider it as a curative means it is of less value. I have seen the fluid recur several times after it has been removed, and that, too, in a relatively short period. This statement is true of instances in which the pericarditis, either at that time or subsequently, was known to be tuberculous. One is thus led to ask whether in these instances it is not preferable to perform a radical operation with proper surgical technique and thorough drainage of the pericardium. In purulent cases the general consensus of good surgical judgment is to that effect. In tubercular cases where the effusion is not purulent it may be also a wiser procedure than simple puncture, because through the open wound the finger may be introduced, and large masses of fibrin extracted. These masses, if allowed to remain *in situ*, inevitably delay cure by allowing fluid to re-form rapidly. After a reproduction of fluid on one or several occasions, adhesive pericarditis may and does develop, and, sooner or later, we have to do with a seriously crippled heart. There might be a chance of obviating this by means of the canula left in place and the use through it of some form of alterative injection. Rendu¹ reports such a case of cured tuberculous pericarditis. The modifying injecting fluid employed by Rendu was a solution of pure camphorated naphthol.

What is true in cases where effusion has been withdrawn as regards some of the results to heart fibre is true where we leave the fluid in the pericardial sac and do not attempt to remove it. By and by underneath the thickened layer there comes a large layer of fat. This fat finally penetrates the heart wall between the muscular fibres, and, in connection with a deposit also of cellular tissue, leads to fatty degen-

¹ Journal of the American Medical Association, 1901, p. 1432. Also, Bull. de la Soc. des Hôpitaux de Paris, March 21, 1901.

eration of the heart structure, and later, perhaps, to combined fibroid changes.

As regards serous effusions, even though large in amount, such as we meet with as a complication, especially of acute articular rheumatism and occasionally in nephritis, these rarely require puncture, in my judgment. Whenever the indication arises, unless the condition be very imminent, I am of the opinion that one or a succession of fly-blisters over or near the heart will accomplish all that is urgently required. It is not essential, moreover, that much fluid be removed from the pericardium in these cases so as to promote absorption; a small quantity is sufficient. The proof of this is that often after aspiration of the pericardium, where very little fluid has been actually removed, a notably beneficial effect quickly follows. The same thing results from a severe counter-irritant or revulsive. How this acts (blister)¹ I am not quite sure. Evidently there is no immediate vascular connection between the skin in the præcordial region and the heart itself, and for this reason it would seem as though the blister would do as much good were it placed over a region far removed. Certainly, if mere reflex action comes into play it is possible. And yet, somehow or other, I am a believer in its good effects applied over the præcordium, and I do know that revulsion, irritation, or heat locally over the heart is of great practical value in adding to its power. I can readily conclude, therefore, that wherever effusion in large amount is partly passive, increased heart action may be remedial in a very distinct and rapid manner.

With respect to the operation of paracentesis *versus* the use of counter-irritation in those cases where the effusion is limited and probably serous, we should always have in view the possibility of change to a purulent effusion caused by the little operation itself, especially if performed with the ordinary trocar and canula. With proper aseptic precautions and the use of the aspirator, this to-day may be regarded as a negligible quantity. Moreover, it is known that but one case has resulted fatally following the puncture. "With this exception all the patients were greatly relieved by the removal even of a small amount of fluid, and many recovered completely who probably would have died if the operation had not been performed."

In purely purulent effusions it is now generally admitted that surgery with open wound, with or without drainage, should alone be considered. Porter's case² and others still prove this. Only lately, for example, H. Lilienthal has had a successful radical operation in a

¹ Dr. Shattuck never uses blisters. Drs. Tyson and Rotch would not let them go.

² THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1897, p. 458.

³ The only case ever treated by incision in Massachusetts General Hospital (F. C. S.). Boston Medical and Surgical Journal, May 6, 1897, p. 438.

case of purulent pericarditis. Here aspiration had proved insufficient. In Lilienthal's case a portion of the fifth left costal cartilage was resected close to the sternum under local anæsthesia. Forty ounces of fluid were removed from the pericardial sac.¹

In another case, reported by Ogle and Allingham, the pericardium was opened, a large quantity of pus removed, and the pericardium cleansed, without interference with the action of the heart and with decided benefit to the patient. It was shown in this instance that the heart can be handled without harm. It is therefore advisable to treat these cases precisely as we would an empyema. Indeed, the operation is more indicated than that of empyema, because the walls of the cavity are better able to contract and finally lead to complete obliteration. We know the outcome in empyema, where, to reach the best obtainable result, portions of several ribs must often be resected. Even then we have to deplore many incomplete successes.

Finally, in the operation by opening and drainage we have really the only legitimate hope of entire recovery. No one has insisted upon this operation more strenuously than Dr. J. B. Roberts since 1876. In a late paper he reiterates his findings. Very properly, he says, "the diagnosis of the purulent character of the effusion was only determined by exploratory puncture." And "this should be done at the upper part of the left xiphoid fossa close to the top of the angle between the seventh cartilage and the xiphoid cartilage."²

"The prognosis is good," says Dr. Roberts, "in pericardotomy for pyopericardium. In a table of 26 collected cases 10 recovered and 16 deaths were shown. This gave a percentage of recovery of 38.4. Of the fatal cases at least 9 were septic, and all the others who died had complicated lesions such as pleuritis, or pulmonary, cardiac or renal lesions."³

"The results obtained by incision and drainage in tubercular peritonitis suggest that drainage in tuberculous pericarditis may lead to a permanent cure. The ease with which the pericardium can be irrigated with solutions of iodoform would seemingly add to this probability of success."⁴

After analyzing different cases, Roberts writes: "These observations and other reported cases not here mentioned have almost convinced me that incision is better than aspiration even in cases not supposed to be purulent. It establishes diagnosis in dubious cases, avoids cardiac injury, saves the pleura from puncture, affords complete evacuation of effusion, permits extraction of thick pus and membranous lymph, and gives opportunity for disinfection of the sac when that is necessary."⁵

¹ New York Medical Record, November 25, 1900.

² Boston Medical and Surgical Journal, May 27, 1897, p. 522.

³ Loc. cit.

⁴ Transactions of the American Surgical Association, 1897, p. 106.

⁵ Loc. cit.

⁶ Loc. cit.

Porter cites one successful case of incision in serous pericarditis where puncture failed to relieve.¹

Personally, so far as tuberculous cases are concerned, I agree with Dr. Roberts. Ordinarily, in cases of simple serous effusion of other provenance I do not believe incision is called for, and if any operative interference is required I still prefer paracentesis. If irrigation be employed as an adjunct in incision the outflow of fluid must be unimpeded, or death may rapidly result from it.

In regard to the technique, it is not necessary to say more than to point out that the best operation is the one usually which permits best drainage in a given case. In one instance it may indicate resection of the fourth or fifth rib on the left side; in another the pericardium should be opened from below and through the insertion of the diaphragm near the central tendon.

It should be remembered, in my judgment, that the question of radical operation for pericarditis with effusion is different in one very important particular from that of pleurisy with effusion. There are two lungs; there is only one heart. A patient may do fairly well for a long time with pleuritic adhesions, a retracted chest wall, and an atelectatic and fibroid lung. No patient will continue long to be in any degree comfortable or active who has adhesive pericarditis as a sequela of large and long-continued effusion, with the pathological changes of heart walls which invariably follow sooner or later.

To those who have had small clinical experience with these cases and who may be led to believe that the liability of heart puncture is slight, I would point out that the anatomical relations of the pericardial sac, despite Dr. Damsch's researches with the chest walls and left pleura, in many instances are very perplexing and variable. As to the differential diagnosis of pericardial effusion with a heart merely enlarged, while this is often very simple and requires no great medical acumen, there are occasions when the most careful use of physical methods of exploration will leave one in a state of great uncertainty. Again, I have had to do with cases where I was confident there was a large effusion and no risk in introducing a small trocar connected with the aspirator. Unfortunately my diagnosis was incorrect, manifestly on one occasion, and instead of withdrawing fluid from the pericardium my aspirating needle penetrated the heart wall. This was obvious by reason of the fixed position, and special movements transmitted to the canula when the trocar was withdrawn. In this instance no great or immediate harm resulted. Still, it is an accident to be avoided, as far as possible, by great care and attention.

Orphüls reports a case where, at the autopsy, the end of a trocar

¹ Transactions of the American Surgical Association, vol. xv.

needle was found in the scar tissue at the upper portion of the interventricular septum. This needle probably broke off in an exploration or in a previous operation of paracentesis.¹

Loison² affirms, also, that in wounds of the heart and pericardium the "prognosis is not always grave." This judgment is strengthened by the statistics collected both by Fischer and himself. On the other hand, Rotch has "known of a case where pricking the heart with an aspirator needle has caused sudden death," and Janeway one where the aspirating needle tore the heart, causing death.

Formerly, so as to avoid just such mishaps, I had constructed for myself a modified Roberts canula.³ This is a very good instrument when kept in proper order. It is a little complicated, however, and requires to be looked after. All that precedes acquires additional interest, if we recognize, as many do, that the ordinary medicinal remedies as applied to the treatment of pericarditis with effusion (especially the tuberculous form) have very little value.

I am not aware, once pericarditis has become developed, that any remedy given internally abridges its duration or changes its course very perceptibly. I acknowledge, of course, in a few instances, that the heart needs special stimulation in view of the failure that may come on suddenly or by degrees. The alkaline treatment may prove very useful in giving strength to cardiac contraction—more, indeed, than the use of digitalis. This would be in accord with the experiments of Gaskell.⁴ I also know that in a few—a very few sthenic cases—aconite may be indicated for a short time to lower blood tension and decrease the rapidity of the pulse; but that is about all there is to do in the acute stage by way of the mouth. Locally, as I have already pointed out, the ice-bag or poultices, blood-letting (leeches and cups), are useful where there is pain or great increase of heart action. Beyond some degree of soothing or quieting thus produced, we should not count upon a great return.

The prophylactic measures to be employed have to do solely, it seems to me, with the efficient causes of tuberculous pericarditis which prevail, be it possibly pleurisy, pneumonia, sepsis of some sort, or mere exposure, fatigue, or debauch.

As we know already, tuberculous pericarditis may show itself when we have had little or no suspicion of its presence. It may also be ushered in with so few obvious symptoms or signs, local or general, that except in an accidental way it is not discovered during life. There are many examples, indeed, where the presence of the disease

¹ British Medical Journal, January 27, 1900.

² THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1900, p. 218.

³ Medical Record, March 29, 1884, p. 361.

⁴ THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1896, p. 696.

was ignored during life and only ultimately revealed by the findings at the autopsy.

Appended is an abstract of the history of my second case:

History of case of tuberculous pericarditis (No. 2). B. F., married, a hod-carrier, born in the United States, aged thirty-eight years, was admitted to St. Luke's Hospital under my care, March 19, 1901. Family and personal history negative, save that he was a somewhat excessive drinker of beer and whiskey. Present illness began four months ago, after a debauch, with pain in his right side. No cough, chills, or vomiting. The pain in the side lasted a month, when he began to cough, expectorated blood-stained mucus, had some fever, with afternoon exacerbations, and became weaker. Two weeks ago pain in the præcordial region, with palpitations, developed; also at times dyspnoea. No swelling of the feet.

Physical examination shows marked increase of cardiac dulness, rapid heart action, and a high-pitched systolic murmur, heard between the nipple (left) line and sternum. Thoracentesis gave serous fluid in right pleural cavity. Temperature, 99.3° F.; pulse, 100; respiration, 49. Urine, acid, 1020; no sugar, no albumin, few leucocytes. No tubercle in sputum.

X-ray examination shows shadow corresponding to line of cardiac percussion dulness.

Microscopical examination of blood negative; also examination of eyes negative.

April 25th. Guinea-pig inoculated with fluid from right pleura. On May 19th tubercle found in cheesy pus from enlarged lumbar glands of guinea-pig.

May 26th. Patient paralyzed on right side. Soon became comatose and died the same day. During sojourn in the hospital the patient was feverish, with pronounced irregular rise in the afternoons.

Autopsy, made by Dr. N. E. Ditman, resident pathologist, showed much thickened pericardium; sac contained eight ounces of yellow, turbid fluid. Heart enlarged, muscle pale, and covered by a layer of subpericardial fat; valves normal, save a slight atheroma of one flap of mitral. Lungs, liver, spleen, meninges, and peritoneum showed disseminated miliary tubercles.

Microscopical examination showed cheesy degeneration and numerous giant cells throughout the pericardium. In the layer of fat over the heart and beneath the pericardium were numerous small masses of small round cells, and in each mass giant cells were present. The heart muscle, superficially, was infiltrated with fat. More deeply there was a moderate small round-cell infiltration between the muscle bundles, and in places the latter are separated by connective tissue; connective tissue of bloodvessel walls increased in amount.

NOTE.—Since writing my paper a very interesting case of tuberculous pericarditis with effusion has been reported by Dr. F. R. Sabin¹ from Prof. Osler's clinic. "The points of interest are: (1) the great value of repeated tapplings in serous effusions; (2) the finding of tubercle bacilli in the pericardial exudate, and (3) the high percentage of mononuclear forms in a tuberculous exudate."

¹ American Medicine, March 8, 1902.

A CASE OF AORTIC INSUFFICIENCY, WITH PERICARDITIS, PLEURISY, AND MEDIASTITIS.¹

BY AUGUSTUS A. ESHNER, M.D.,

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THE following report of an isolated case would seem justified by reason of its unusual character, of the prominence of the physical signs, of the disparity between these and the remaining symptoms, of the etiological relations, and of the diagnostic considerations.

A man, aged forty years, by occupation a printer, presented himself at the Polyclinic Hospital, complaining since four months of constant pain throughout the entire chest and back, worse at night and also during inspiration. There was, besides, palpitation of the heart, slight cough with mucous expectoration, and some dyspnoea. There had never been hæmoptysis. The appetite was good, the bowels regular. Sleep was poor, and the frequency of micturition was increased. The lower extremities were slightly cedematous. The man was pallid, and he had lost thirty-six pounds in weight during the period of four months. He presented an appearance of distress.

The left side of the chest was comparatively prominent anteriorly. The cardiac impulse was visible in the sixth interspace on the left in the nipple line, but pulsation could be felt also in the seventh interspace out to the anterior axillary line and at the ensiform cartilage and for some distance on either side. Systolic retraction of the intercostal spaces took place over a large extent of the inferior portion of the left chest, anteriorly and posteriorly, and some degree of retraction appeared to be present also on the right side inferiorly. A thrill was palpable over the body of the heart and apparently a separate and more marked thrill in the aortic area, and also a thrill intermediate in intensity to the right of the sternum in the third and fourth intercostal spaces. The thrill was in each instance diastolic in time. The area of cardiac dulness was increased, particularly downward and to the left.

On auscultation attention was first attracted by a rough though musical murmur, audible throughout the præcordium, but heard with greatest intensity in the aortic area. It could be detected, even through the air, at a short distance from the chest, and it was heard also in the axilla and in the back, gradually losing its musical quality, which, however, became again appreciable along the left of the vertebral column. In this situation the murmur was most pronounced at the level of the spine of the scapula. This sound was followed or preceded by a dull, booming sound, synchronous with the impulse of the heart and the radial pulse. Over the vessels of the neck the transmitted murmur and a dull thud were heard in succession. At the base of the

¹ Report read and patient demonstrated before the Section on General Medicine of the College of Physicians of Philadelphia, January 13, 1902.

heart creaking sounds were audible with the cardiac sounds, being intensified by deep inspiration, and fine crackling was brought out when the patient inclined the body backward. These signs were less pronounced when the body was inclined forward. The radial pulse exhibited a water-hammer character, and this is well shown in the accompanying sphygmogram.



The right radial did not pursue its normal course, at the wrist passing on the outer side of the radius. A systolic thrill was generated by pressure over the subclavian arteries.

In the erect posture the line of percussion dulness to the right of the sternum pursued a zigzag course obliquely downward and outward across the sixth, seventh, eighth, and ninth ribs. In the middle line the lower limit of dulness was 9.5 cm. below the ensiform cartilage, and in the right nipple line 5 cm. below the costal margin. In the recumbent posture both boundaries descended from 3 to 6 cm., and a sense of resistance was appreciable along the lower border. Percussion over the area of dulness below the costal margin was attended with tenderness.

In the epigastrium, below and to the right of the ensiform cartilage, was a scar left by a stab wound inflicted three years previously. The pupils were practically equal, although the left may have been slightly the larger.

The patient stated that he had had scarlet fever and smallpox as a child and influenza at the age of thirty years. He denied venereal infection. He drank several cups of tea or coffee daily, but no alcohol. The urine was free from albumin and sugar.

There was some doubt at first as to whether the loud musical murmur heard over the heart was systolic or diastolic. Its predominance in duration and intensity over the other sound, which exhibited a dull, muscular character, suggested the possibility of its being systolic in time; but its asynchronism with the impulse of the heart, with the radial pulse, and with the thrill appreciable on palpation of the subclavian arteries, together with the sounds heard over the vessels of the neck, convinced me that the murmur was diastolic.

Some difficulty was experienced also in providing a satisfactory explanation for the thrill palpable in three situations with varying intensity. The creaking sounds at the base of the heart suggested the presence of adhesions resulting from previous pericarditis and pleurisy, with involvement of the mediastinal structures, and it was thought that the thrill palpable in the same situation might be due to circulatory disturbances in an atheromatous aortic arch possibly dilated at its origin. The thrill over the body of the heart may have been due to the same cause or to

vibrations in the anterior mitral leaflet generated by the current of blood returned from the aorta into the ventricle. The systolic retraction in the lower interspaces on both sides of the chest, but particularly on the left side, seemed to confirm the view as to the presence of pleuro-pericardial adhesions.

The liver appeared to be movable, in the recumbent posture rotating forward upon its transverse axis. The presence in the epigastrium of a scar left by a previous stab wound raised the possibility of a traumatic origin for the lesions; but as these seemed to be multiple and rather widely distributed, and in part remote in situation, such an etiological connection could be excluded and an antecedent infectious process made the responsible factor. It will be noted that there is a history of scarlet fever, smallpox, and influenza, but none of rheumatism; nor is there anything in the patient's occupation to which the lesions present could reasonably be attributed. It is now practically established that inflammation of the endocardium and of serous membranes and even of synovial membranes may attend any infectious process; and it may occur, I am sure, in the absence of any obvious primary localization, as a result of so-called cryptogenetic invasion. Acute rheumatism has long been considered by far the most common cause of chronic valvular disease of the heart; and while this view is sustained by the results of critical inquiry,¹ inasmuch as rheumatism has been shown to be a more important etiological factor than any other infectious process, the disease named has been found to be less significant in this connection than all other causative factors combined. Further, I am inclined to the belief that under the clinical designation rheumatism are included many forms of arthritis, involving one or more joints, but that among these there is one with a definite clinical course and probably dependent upon a specific micro-organism—possibly that described by Poynton and Paine;² and for this the designation acute rheumatism or rheumatic fever should be reserved, while the others are to be classed among articular infections of diverse origin.

Murmurs audible at a distance from the chest are not common, but by one of those coincidences that are not rare in medicine a patient presenting a murmur like that in the case here recorded was exhibited by Dr. Alfred Meyer at a meeting of the Section of Medicine of the New York Academy of Medicine.³ In this case also the murmur was of aortic origin, diastolic in time and musical in quality. It could be heard at a distance of from two to six inches from the chest. It was thought probable that the aortic orifice was enlarged or the root of the aorta dilated, resulting in relative insufficiency of the aortic valve. To

¹ Worobjew. *Deutsches Archiv für klinische Medizin*, Bd. lxxix., H. 5 u. 6, p. 466.

² *Lancet*, September 22 and 29, 1900, and May 4, 1901.

³ *Medical Record*, February 8, 1902, p. 237.

produce a musical murmur it was believed necessary for the surfaces over which the blood stream passed to be quite smooth. Dr. Meyer estimates the frequency of murmurs audible at a distance from the chest as perhaps 1 in 300 cases of heart disease.

NOTE.—At a meeting of the Section following that before which the case here reported was presented Dr. H. A. Hare exhibited a case of aortic insufficiency with a diastolic musical distance murmur.

THE FLUORESCENCE OF QUININE AND OTHER REMEDIES IN THE CURE OF MALARIAL FEVER.

BY A. F. A. KING, M.D.,
OF WASHINGTON, D. C.

IN a recent paper* it was maintained that the malarial parasite would not sporulate in the dark; that the light it obtained in the blood was necessarily *red*; and, following the demonstrations of Harrington and Leaming,¹ that *amœba proteus* streams in the presence of *red* light and ceases to stream in *violet* light, it was assumed that the same may be true of the *malarial* amœba; hence it was suggested that the curative action of quinine might be due to its fluorescence, producing violet rays of light in the blood. Evidence in support of this view will now be presented

First, it may be noted that two other vegetable products possessing blue fluorescence in a remarkable degree have long been known as effective remedies for intermittent fever, viz., *esculine* and *fraxine* (or *fraxinine*).

As to the efficacy of *esculine* former records furnish abundant testimony. Notably, Monveneux² reports in detail twenty-eight cases successfully treated, a report the reliability of which is accentuated by the author adding ten other cases which *esculine* failed to cure, but which were promptly relieved by quinine.

An article in the *French Dictionary of Medical Science*³ refers to the experiments of MM. Mouchon, Durand de Lunel, Diday, Vernay, and Monveneux, as demonstrating that *esculine* is comparable to quinine as an antiperiodic. The bibliography at the end of this article contains twenty-three references to *esculine*, dating from 1720 to 1858.

I find *esculine* presents, in solution, a far more brilliant blue fluorescence than any salt of quinia or cinchona. Its fluorescence is, how-

* THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, February, 1902.

ever, immediately destroyed by dilute sulphuric acid, but promptly reappears by adding a few drops of aqua ammoniæ to the solution.*

Fraxine—the other fluorescent substance named—is made from the bark of *Fraxinus excelsior*, the common ash tree of Europe. This was often given successfully for intermittent fever before the discovery of quinine, and later, one writer who reports cases cured by it, in which quinine had previously failed (?), was so convinced of its efficacy that he surnamed it *quinquina d'Europe*.⁴

The tincture and other preparations of iodine have been used successfully for intermittent fever for a long time. The literature on this subject is extensive. Sircar⁵ gives seven successful cases, and quotes Grinnell⁶ to the effect that “one hundred and forty cases were treated with the iodine, and the results were fully equal to those treated with the sulphate of quinine. The remedy seemed to act almost as by magic,” etc.

Now, while iodine is *not* fluorescent, and cannot, therefore, act (as suggested for quinine) by accentuating violet rays, yet in the body it can scarcely escape coming in contact with starch and forming the *blue or violet iodide of starch*, which is all the more likely to occur from the iodine being usually given *after meals*, to avoid its irritating effect on an empty stomach. Thus the curative influence of tincture of iodine would seem to fall in line with that of Prussian and methylene blues. One writer, Entrikon,⁷ prescribed the *iodide of starch* itself, but finally settled upon the compound tincture of iodine as the best of several preparations.

While the antiperiodic effect of the fluorescent substances esculine and fraxine favor the idea that fluorescence may be the curative property in quinine, the proof is insufficient. We need direct experiments showing that quinine actually produces fluorescence in the body, and that coincidentally the symptoms and phenomena of malarial fever disappear.

Exactly these results were actually obtained more than thirty years ago (1868) by the experiments of Drs. Edward Rhoads and William Pepper, of Philadelphia. These experiments, so long buried in oblivion apparently, were brought about in the following manner: In 1866, Henry Bence Jones,⁸ experimenting on the rapidity with which medicinal substances pass into and out of the body, was induced to use quinine for this purpose on account of the extremely minute quantity of this substance (1 grain in 1,000,800 parts—nearly 117 litres of water) that could be recognized by its fluorescence. To his surprise, he found that fluorescence existed in the blood and most of the tissues

* I obtained the drug from Merck & Co., of New York, who, in their “Index” for 1896, p. 93, describe it as an “antiperiodic. Uses: *Instead of quinine for intermittent fever.*” They inform me that little is sold, probably from its high price—three cents per grain.

of man and animals in those who had *not* taken quinine, but it was increased in those who *had* taken it. Without following him in detail, Jones obtained the result—interesting to us now—of discovering a substance in the tissues of guinea-pigs and man which possessed *all* (*italics* his) the chemical and optical properties of quinine, including fluorescence. He called it *animal quinoidine*. Early in his paper Jones remarks: "No imagination could have anticipated that this line of research into the rate and passage of substances into and out of the textures would lead to the supposition that man and all animals possess, in every part of the body, the most characteristic peculiarity of the bark of the cinchona trees of Peru;" and later in his paper he says: "Assume that a substance like quinine exists during health in the textures, can its rapid destruction and removal through the action of the marsh miasm* give rise to ague?"

Following Jones, Drs. Rhoads and Pepper² endeavored to determine "whether there might not be, as an attendant upon the pathological processes in malarial disease, a rapid and marked diminution in the amount of 'animal quinoidine' naturally existing in the tissues."

They first repeated the experiments of Jones and corroborated his results, and then proceeded to test the blood of malarial patients, with the result of finding its *fluorescence below the normal standard*. Altogether they give twelve cases, amply described and tabulated. They obtained blood by cupping over the spleen, and the degree of fluorescence was determined by comparison with the fluorescence of standard solutions of quinine of known strength—a given number of grains in 100 litres of distilled water. Thus a fluorescence of 3 would equal that of a quinine solution having 3 grains of quinine in 100 litres of water; a fluorescence of 6 would equal that of 6 grains in 100 litres, etc.

Their twelve cases may be epitomized with extreme brevity, as follows: Case I. Healthy woman. Fractured bones. Blood-test gave fluorescence of 3. Case II. Tertian ague; fluorescence, $1\frac{1}{2}$. Case III. Marked intermittent; fluorescence, 1. Case IV. Marked intermittent; fluorescence, $\frac{1}{2}$. Case V. Tertian; fluorescence, $\frac{1}{2}$. Case VI. Quotidian; fluorescence, $\frac{1}{2}$. Case VII. Quotidian and tertian; fluorescence, $\frac{1}{2}$. Case VIII. Quotidian and tertian; fluorescence, $\frac{1}{2}$. Case IX. Tertian; fluorescence, $\frac{1}{2}$.

These cases show a fluorescence of 3 in the normal woman, and of less than 1 in five of the seven cases of fever, the other 2 being severally 1 and $1\frac{1}{2}$. In these seven cases no quinine was given, and care was taken to ascertain that none *had* been given for some days before the examination.

* This was thirty-four years before Laveran discovered the malarial parasite in 1880, and before the part played by the mosquito in malarial disease was recognized in 1883.

Then follow 2 cases showing increase of fluorescence and consequent cure by sulphate of cinchona.*

Case IX. Quotidian; fifteen months' duration; spleen enlarged. Fluorescence: "none could be discovered, and it was doubtful if any existed." After fourteen days under cinchona sulphate no recurrence. Much improved; fluorescence, 1½.

Case X. Quotidian for nine days following previous irregular symptoms. Fluorescence, ½. After six days, cinchona sulphate 20 grains daily; fluorescence, 3½; no recurrence of chill.

Next come two fatal cases. One (XI.) was a quotidian of four weeks' duration. Last two paroxysms congestive; the final one at 11 A.M., death at 2 P.M. Had taken 20 grains of cinchona sulphate two days before death, and one or more doses of 15 grains each on the day of decease. Post-mortem examination revealed fluorescence of 2½ in blood of spleen and 2½ in blood of liver.

Case XII. Chronic malarial cachexia; ten months' duration; general dropsy three months; improved under cinchona and Basham's mixture, then typhoid fever, and death on ninth day of that disease. Took *large doses* of cinchona for *fifty-four days* preceding demise. Fluorescence in blood of spleen, 6, and of liver, 8.

At the conclusion of their paper the authors remark that their observations "indicate, by the uniformity of the results, a close connection between the diminution of 'animal quinoidine' and malarial disease."

In 1875 Mills,¹⁰ of Philadelphia, suggested that fluorescence may afford an explanation of cinchonism. Commenting on the experiments of Jones, Rhoads, and Pepper, he says it has been proved that animal fluorescence is increased by quinine; that it "is decreased below a normal standard in malarial affections, and that it rises in the scale *pari passu* with the giving of the cinchona preparations and the improvement in the symptoms of the diseases."

Thus, then, it is the peculiar relations of the cinchona preparations *with light* that give a clue to the explanation of their curative effect. Possibly the same may be said of *all* assimilable substances that fluoresce a *violet* or *blue* color. Some substances have a green, orange, or red fluorescence; these we should not expect to have any curative influence in malarial disease.

It remains to explain the cases in which quinine *fails* to cure—exceptions that seem to prove the rule that the malarial organism will not sporulate in the dark, and, further, that *in the dark* quinine (necessarily) cannot kill the parasite with fluorescent *light*.

In these cases the febrile paroxysm is not produced by the usual ter-

* Cinchona sulphate was used instead of quinine. Authors do not state why. Probably the quinine salt may have been too expensive for hospital use in those days.

tertian or quartan parasite, but by the "malignant" crescentic form of which Manson¹ gives three varieties, viz.: 1, æstivo-autumnal; 2, malignant quotidian; 3, malignant tertian. He calls all of these crescentic forms "malignant infection," and says "the malignant parasites *alone* form crescent bodies" (p. 65); that quinine "has apparently no influence on these bodies" (p. 66); they "may be found for days after the patient is cinchonized" (p. 44); or, "after full doses of the drug continued for a fortnight" (p. 183). The malignant parasite does *not* cause fever paroxysms, but is usually associated with marked cachexia" (p. 66); all of which is explicable by the further facts that "these crescent parasites are not generally found in the *peripheral* circulation" (pp. 15, 65, and 85), where they can get *light*, but are found in the spleen, liver, bone-marrow, and brain, than which no *darker* recesses of the body can be imagined, and consequently where the fluorescent property of quinine *must* be impotent. When they *do* appear in the peripheral circulation "it is a sure sign that a paroxysm of fever is impending" (p. 69); that is to say, having come out of their dark recesses into the red light of the peripheral blood, sporulation occurs, and with it the usual ague paroxysms. These quinine may arrest, but it cannot act upon parasites in the dark.

Possibly the malignant parasites are sickly, degenerate, or famine-stricken individuals, from the man's blood being overstocked or anæmic. The corpuscles containing them are *not enlarged* like those containing benign tertian parasites; the latter may be sometimes nearly twice the diameter (Manson, p. 62) of healthy corpuscles.

A normal capillary will just admit one normal corpuscle to pass, and while both are elastic, and the skin capillaries among the largest in the body, it is conceivable that the enlarged corpuscles with benign tertian parasites *become arrested* in the skin, on account of their size, and so remain until light causes them to break up by sporulation; on the other hand, the smaller corpuscles with crescent parasites are *not* arrested in the skin capillaries so as to be continuously exposed to light, but pass on and become arrested by smaller capillaries in the brain or elsewhere. Some of them may eventually get light enough to manage sporulation, at odd times, or continuously, but not a sufficient number sporulate *simultaneously* to generate a fever paroxysm.

In a normal case of typical tertian ague the successive events that occur during the three stages of chill, fever, and sweat are as follows: Previous to the chill a group of corpuscles lies arrested in the peripheral capillaries, their parasites exposed to sunlight preparatory to sporulation. With the sporulation is the *chill*, by which the skin arterioles are violently contracted and emptied of blood; hence, pallor and coldness of the surface; and, as if to assist in the dislodgement of the infected corpuscles and their complete disintegration, the whole fabric

of the man's body vibrates with a "shaking chill." During the *fever* the arterioles dilate again, even beyond the normal limit; an actual congestion of the skin occurs; hence its redness and heat, during which a new crop of infected corpuscles enters the peripheral capillaries. During the *sweat* a deluge of water exudes from the distended cutaneous bloodvessels, causing them to contract, close up, and shut down upon the new group of infected corpuscles, which, thus arrested, so remain until forty-eight hours of sunlight secures in turn their sporulation and the recurrence of another paroxysm. Thus a typical tertian paroxysm might be regarded as a *functional disease* (or process) of the *skin*, during which the human organism is operating, according to nature's laws, in the interest of the parasite to secure perpetuation of its species. If the process were allowed to run its natural course without interruption (without clothing, quinine, etc.) the human individuals, or their race, in the course of many generations, get their compensation by becoming black from malarial melanosis, with the same immunity that now belongs to dark-skinned peoples.

Thus regarding the malarial process as *naturalists* instead of *medical practitioners*, this new point of view presents confirmatory evidence of the rôle played by sunlight in producing the observed phenomena.

Finally, the idea that succession paroxysms of fever are produced by sporulation of successive groups of those parasites *only* whose domiciliary blood-corpuscles have become sufficiently enlarged to cause their arrest in the cutaneous capillaries is, I am aware, at variance with the generally recognized theory of Golgi that the paroxysms are produced by successive *generations* of parasites, each fit corresponding to the ripening, sporulation, and setting free of a *new brood*.¹² My idea is that only such a *part* of any generation, the blood-corpuscles of which have become large enough to get arrested in the peripheral circulation, sporulate at any one paroxysm. The others must take their turn, waiting until the corpuscles they inhabit have become increased in size sufficiently to get arrested in the skin, where sunlight may be obtained to secure their sporulation also.

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A STUDY OF HYPERPLASIA OF THE PHARYNGEAL LYMPHOID
TISSUE (ADENOIDS), WITH ESPECIAL REFERENCE
TO PRIMARY TUBERCULOSIS OF THE
PHARYNGEAL TONSIL.

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INTRODUCTION. The most important recent literature on the pharyngeal tonsil relates to tuberculosis of this tissue. While many of the newer investigations have thrown considerable light on the morphological and clinical aspects of the lesion, numerous points still remain to be settled. Perhaps one of the most interesting of these at present is the frequency with which this lesion occurs, and whether or not the lesion remains focal or may become disseminate. Thus far the results of study as to the frequency of the lesion permit of widely different interpretations; and in order to throw further light on this point we have restudied the question in a systematic manner, and have, in the course of our investigations, incidentally touched upon certain questions related to the bacterial infection and general pathology of adenoids.

THE NORMAL TONSIL. Considered either from the anatomical or physiological view-points, the various lymphoid tissues of Waldeyer's ring present fundamental features in common. Their morphology and function have best been studied in the faucial tonsil; and only very recently, and to a much less extent, have similar observations been made on the pharyngeal lymphoid tissue.

The tonsils are composed of lymphoid structures closely resembling in their general anatomy Peyer's patches of the small intestine.¹

The physiological activities of the tonsils have been variously interpreted by different investigators. The older view, now discredited, assumed that they were secreting organs furnishing a thick, viscid mucus. Later, Kingston Fox,² Hill,³ and Spicer⁴ advanced the view that these structures reabsorb the waste secretions of the neighboring

¹ For a consideration of the anatomy of the normal pharyngeal tonsil, with bibliography, consult Gottstein and Heymann's *Handbuch der Laryngologie und Rhinologie*, Wien., 1899, Bd. ii. S. 584.

² Fox. *Journal of Anatomy and Physiology*, London, 1885-86, vol. xx. p. 559.

³ Hill. *British Medical Journal*, 1888, vol. iii. p. 615

⁴ Spicer. *Lancet*, 1888, vol. ii. p. 805.

parts. More recently the question of absorption through the tonsils has been the subject of still more important researches. Studies in this direction by Ruffer,¹ Gulland,² Goodale,³ and Hendelsohn⁴ show that foreign materials, such as carmine, soot, and cinnabar, may be absorbed from the surface of the faucial tonsil in a short time after their application.

Goodale, in a series of interesting experiments in which he injected carmine particles into the tonsillar crypts, and after a time varying from a few minutes to several weeks, removed the tonsils and examined them microscopically, came to the following conclusions: 1. Absorption takes place normally through the mucous membrane of the lacunæ. 2. It follows the line of direction of the connective tissue. 3. The multinuclear leucocytes take a part in the process. 4. Bacteria are usually found in the lacunæ, but are not easily recognizable in the tonsillar tissue.

This observer's main conclusions have been confirmed by Labbé and Levi-Sirugue;⁵ and Hendelsohn⁶ has further shown that the pharyngeal tonsil also absorbs foreign particles. While Goodale claims that particles may be absorbed through the normal mucosa of the crypts, Hodenpyl⁷ concluded, from experiments made in 1891, that absorption does not occur through the normal mucosa. He believes, however, that substances finding their way beneath the epithelium, chiefly on account of the rarefaction of the epithelium, may be taken up rapidly by the lymphatics. Most recent observers favor the view advanced by Goodale.

There has also been much discussion as to the importance of different cells in the absorption of material from the tonsil surface. Statements as to the presence of foreign material within the leucocytes of the sub-mucous tissues and of the lymph-adenoid tissue of the tonsil are somewhat at variance, some authors stating that they found it within polymorphonuclear cells. The character of the leucocytes within the tonsil in health and disease is one, however, that cannot be said to be definitely settled, although the more recent work with more elaborate methods of staining suggests that the leucocytes within the tonsils are not of the form which ordinarily in other portions of the body are considered to have phagocytic action.

In order to test the significance of the migration of leucocytes, Hendelsohn⁸ injected emulsions of soot and cinnabar into the tonsils, and found that so little of the material was taken up by the leucocytes and

¹ Ruffer. *British Medical Journal*, 1890, vol. ii. p. 491.

² Gulland. *Edinburgh Medical Journal*, 1891, vol. i.

³ Goodale. *Arch. of Laryngologie u. Rhinologie*, Bd. vii., Heft 2, p. 90.

⁴ Hendelsohn. *Arch. of Laryngologie u. Rhinologie*, Bd. viii., Heft 3.

⁵ Labbé and Levi-Sirugue. *Bull. et Mém. Soc. d'Anat.*, 1889, tome lxxiv. p. 685.

⁶ Hendelsohn. *Loc. cit.*

⁷ Hodenpyl. *THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES*, 1891, vol. ci. p. 257.

⁸ Hendelsohn. *Loc. cit.*

carried to the surface that the migration of these bodies was not considered as a protective action.

Altogether, these investigations show that in the reticular tissue of the normal tonsil there are found typical lymphocytes, a few eosinophiles (varying in number with the species of animals examined), with no polymorphonuclear leucocytes. The lymphocytes being in other situations non-phagocytic, this migration of the leucocytes has not been looked upon as being actively protective. The reason for the migration is still to be discovered.

PATHOLOGY OF SIMPLE ADENOIDS. *Frequency.* According to Zarnico¹ and others, adenoids occur most frequently between the ages of five and fifteen, less frequently before five and after fifteen. They are said by Frankel² to occur at birth and are not very rare after middle age.

An analysis of six hundred cases in the children's section of the Vanderbilt Clinic shows adenoids occurring thirty-five times in all classes of disease (5 per cent.). We believe that this is too low an estimate, for, necessarily, in a general clinic these growths are often overlooked in the presence of an acute and for the moment more important condition.

An analysis of one thousand cases in the throat and nose department of the same institution, reported from June, 1900, to January, 1901, gave eighty-five adenoids, or 8.5 per cent. From the nature of the examination in this class of cases it is highly probable that none was overlooked. The ages of these cases were as follows :

| | |
|--|-------|
| Up to and including five years | 36 |
| Five to ten years | 19 |
| Ten to fifteen years | 24 |
| Over fifteen years | 6 |
| | <hr/> |
| | 85 |

Youngest case, nineteen months ; oldest, twenty-nine years.

That the condition is not frequent under one year is shown by the result of examination of one hundred cadavers of this age at the New York Foundling Hospital. In these no adenoids were found, although in this institution the condition is frequently met with between the age of three and four years. As to the frequency of adenoids in persons otherwise considered healthy, Meyer found them in 1 per cent. (1000 cases), Schmiegelow in 5 per cent. (581 cases), Wroblenski in 7 per cent. (650 cases), Rosenberg in 15,000 cases, 1350 times (9 per cent.), and Chappel found them present in 3 per cent. out of 2000 New York school-children examined.

¹ Zarnico. Lehrbuch der Krankheiten der Nasen.

² Frankel. Zeitsch. f. Ohrenheilkunde, 1881, Bd. x. S. 113.

MORPHOLOGY OF SIMPLE ADENOIDS. Adenoids have already been the subject of several illuminating microscopical studies, and, on the whole, our own studies do no more than confirm previous histological observations.¹

Adenoid vegetations consist essentially of hyperplastic pharyngeal lymphoid tissue; aside from this they present other more or less constant morphological features.

MATERIAL AND METHODS EMPLOYED. The basis of our remarks upon the histology of simple adenoids is the study of forty-six specimens. The gross features of each adenoid were first carefully noted, and then each was immediately placed in 95 per cent. alcohol, embedded in celloidin, and finally stained with hæmatoxylin and eosin, and picric acid fuchsin. Ten sections at least from as many different portions of each adenoid were examined.

EPITHELIUM. The epithelial layer is subject to considerable variation from the existence of concomitant processes but chiefly mechanical influences, such as pressure. Both the thickness of the epithelium and the morphology of the cells were different in the various specimens. In the same specimen the degree of thickness may also widely vary; thus in a portion only one or two layers of cells may be present, while elsewhere so many as ten or even more may be seen. Either ciliated, columnar, or flat, squamous epithelium may constitute this layer; or between these two forms many intermediate shapes may be noted.

McBride and Turner² state that the thickening occurs especially in children under ten or twelve years of age; on the other hand, in patients upward of fifteen years old they found no lesions of this sort. From these and other observations they conclude that changes of this kind are not necessarily dependent upon the age of the adenoid. Our own experience confirms this view, and further seems to indicate the greater frequency of this epithelial thickening in young children as compared with older ones.

Of the forty-six specimens studied, thirteen presented epithelium about normal for this portion of the pharynx; five of these showed the mucous cells described by Brindel.³ Thirty-two specimens revealed a greater or less amount of squamous epithelium on the surface; the epithelial recessions (crypts) were involved in none of these specimens. Small and superficial areas of mucous membrane necrosis were occasionally noted, with slight neighboring polymorphonuclear cell infiltration. Bacteria, mainly cocci, were usually present in the necrosed and subjacent tissue. Slight polymorphonuclear and mononuclear cell

¹ Vide Brindel. *Revue hebdomadaire de Laryngologie, d'Otologie et du Rhin.*, 1896, vol. xvi. p. 881. Also McBride and Turner, *Edinburgh Medical Journal*, May, 1897, vol. i. p. 356. Baup, *Les Amygdales porte d'entrée de la Tuberculose*. Thèse de Paris, 1900.

² McBride and Turner. *Op. cit.*

³ Brindel. *Loc. cit.*

infiltration was also observed in a few specimens between the normal epithelial cells. Baup,¹ accepting the conclusions of Labbé and Levi-Sirugue,² believes some of these cells to be of epithelial derivation. We have not seen any evidence in our specimens sustaining such an assumption.

Karyokinetic figures were occasionally noted in the deeper layers of epithelium.

Almost all the specimens examined in this study showed a more or less distinct basal membrane beneath the epithelium. In the adult, Moure³ states that there often exists connective tissue strands separating the lymphoid from the epithelial tissue. This was not observed in the specimens examined by us.

LYMPHOID TISSUE. Apart from greater or less hyperplasia of the lymphoid cells, few very definite lesions were observed in the series of specimens. The cells were for the most part closely packed, sometimes showing swelling, necrosis, or mitotic figures. Intermingled with the parenchyma cells, polymorphonuclear leucocytes were often noted, almost always in small numbers. In two specimens in which the fibrous tissue reticulum was considerably increased, eosinophile cells were present in moderate numbers, scattered irregularly through the tissues. Several lesions strikingly similar to those first described by Oertel in the lymphatic apparatus of the body in diphtheria were studied in Specimen 1; this adenoid contained the streptococcus pyogenes.

THE RELATIVE AMOUNT OF THE LYMPHOID AND THE FIBROUS TISSUES. The amount of fibrous tissue present in adenoids is variable; in many cases it is extremely scanty and in others very abundant. The relation of such an increase to the disappearance of adenoids is one of considerable interest. Therefore, we give the results of our observations in this connection of the forty-five specimens studied. Twenty-nine (64 per cent.) showed noteworthy increase of the fibrous tissue, largely perivascular in distribution; in eleven of these the increase was very great and usually associated with some thickening of the bloodvessels.

Brindel⁴ found evidence of fibrous tissue increase in forty out of sixty-four specimens examined, and McBride and Turner⁵ observed it in at least 50 per cent. of all specimens taken from children under seven years. Cornil and Ranvier⁶ and Gottstein⁷ have also described this connective tissue increase. These, the observations of Baup⁸ and our

¹ Baup. *Les Amygdales porte d'entree de la Tuberculose*. Thèse de Paris, 1900, p. 24.

² Labbé and Levi-Sirugue. *Bull. de la Soc. Anat.*, July, 1899.

³ Moure. *Bull. de l'Acad. Med.*, Paris, 1895, p. 570.

⁴ Brindel. *Loc. cit.*

⁵ McBride and Turner. *Op. cit.*

⁶ Cornil and Ranvier. *Histologie pathologique*. Paris.

⁷ Gottstein. *Berlin. klin. Woch.*, 1896, Bd. xxxiii, S. 689.

⁸ Baup. *Op. cit.*

own observations for the most part seem to indicate a greater frequency of such change in the young as compared with adults. It is to this process of fibrosis that the shrinking of adenoids has often been attributed.

Thus, H. Norval Pierce¹ states that in early life the connective tissue is embryonal in type, and matures later, when, as usual in new-formed connective tissue, it ultimately contracts, squeezing the lymph nodules and shutting off the blood supply, thus leading to atrophy. Baup considers it a reparative process. In our opinion, this is probably occasionally one of the factors inducing disappearance of the hyperplastic lymphoid tissue, but by no means the only one. Small atrophic adenoids have been studied by us, but in some we were unable to observe any large amount of fibrous tissue.

BLOOD AND LYMPH VESSELS. Fibrous thickening of the bloodvessels was noted in seventeen of the specimens; in eleven of these concomitant fibrosis of the whole tissue was present. The bloodvessels on the other hand were sometimes slightly involved and without any fibrosis of the tissues elsewhere. Swelling of the blood and lymph vessel endothelium existed in some of the specimens, but in none to a very marked degree except in one which contained lesions similar to those described by Oertel in diphtheria. Evidences of endothelial cell phagocytosis were frequently observed.

BACTERIAL INFECTION OF ADENOIDS. In order to determine whether or not bacteria other than the tubercle bacillus might be present within the hyperplastic pharyngeal lymphoid tissue, eleven medium-sized adenoids, not included in the previous series, were submitted to bacteriological examination. They were all removed from children apparently otherwise well.

TECHNIQUE. The adenoids immediately after removal were placed in sterile bottles and bacteriological examinations were begun in all instances within two hours after their removal. The surface of the tissue was seared with a hot knife and portions of tissue were then removed from the interior of the adenoid with a sterile platinum loop needle; liberal quantities were removed in each instance. The removed material was then carefully broken up into fragments in tubes of sterile broth, and from the latter aerobic agar plates of varying dilutions and tube blood-serum cultures were made. The adenoids, immediately after having removed the tissue for culture purposes, were placed in 95 per cent. alcohol. Series of sections (ten to thirty) from each adenoid were stained by Weigert's modification of Gram's method and with Loeffler's methylene blue. The classification of each identified micro-organism was based on a complete study of the morphological, tinctorial, and

¹ Pierce. *Journal of Laryngology*, January, 1901.

cultural features presented by the bacterium. Animal experiments were further made in some instances to determine the virulence of the organism studied. Cover-slip controls from the fresh tissue were made in each instance.

RESULTS. The tube and plate cultures from five of the eleven specimens remained sterile after ten days' incubation at 37° C. The remaining six specimens gave the following results :

| No. of specimen. | Amount of growth. | Predominating organism. | Bacteria found. |
|------------------|-------------------|---|---|
| 1 | Two colonies. | <i>Streptococcus pyogenes</i> . | <i>Streptococcus pyogenes</i> . |
| 2 | Very scanty. | <i>Streptococcus pyogenes</i> . | <i>Streptococcus pyogenes</i> and <i>staphylococcus pyogenes aureus</i> . |
| 6 | Abundant. | Unidentified bacillus, short, thick, not decolorized by Gram. | Unidentified bacillus; <i>diplococcus lanceolatus</i> , and <i>staphylococcus pyogenes aureus</i> . |
| 7 | Scanty. | <i>Diplococcus lanceolatus</i> . | <i>Diplococcus lanceolatus</i> and <i>staphylococcus pyogenes aureus</i> and <i>albus</i> . |
| 10 | Moderate. | <i>Streptococcus pyogenes</i> . | <i>Streptococcus pyogenes</i> . |
| 11 | Scanty. | <i>Staphylococcus pyogenes albus</i> . | <i>Staphylococcus pyogenes albus</i> . |

The *streptococcus pyogenes* cultivated from Specimen 1 did not kill a rabbit (1210 grammes) which had been inoculated intravenously with 2 c.c. of a forty-eight-hours' bouillon culture. Another *streptococcus* from Specimen 10 similarly inoculated in the same dose into another rabbit (992 grammes) induced no apparent reaction. The *diplococcus lanceolatus* from Specimen 7 failed to kill a rabbit (1355 grammes) inoculated under the same conditions.

EXAMINATION OF THE SECTIONS FOR BACTERIA. The recognition of bacterial forms was often extremely difficult and uncertain. With the exception of Specimens 1 and 6 of the above table, the bacteria were few in number and seemed to be mainly limited on the outside of or just beneath the surface epithelium. For the most part, coccil forms only were observed. They were noted in greater abundance in the crypts along with epithelial cells, leucocytes, and débris. Now and then micro-organisms were made out between the epithelial cells, especially in the crypts and where the epithelium was thinner than normal. Specimens 1 and 6 contained several foci of bacteria, mainly cocci without any distinct evidence of inflammatory reaction around them. A few polymorphonuclear leucocytes were observed in the vicinity. In both specimens the bacterial masses were near the surface. Sections from two of the specimens revealed a few bacteria (cocci and short, thick bacilli) on the surface, and here and there between the cells of the epithelial layer.

Gourc,¹ whose studies included a large number of specimens, found micro-organisms in a still greater percentage of the adenoid growths investigated. Two hundred and one specimens were studied by cover-slips and cultures on blood-serum. His results were as follows :

Twenty-five cultures contained no growths.

Streptococci (never alone) with other bacteria, 37 times.

Staphylococci alone, 69 times.

Staphylococci with other bacteria, 69 times.

Various other cocci (diplococci, tetrads) alone, 41 times.

Various other cocci (diplococci, tetrads) with other bacteria, 54 times.

Pneumococcus alone, 3 times.

Leptothrix buccalis alone, 2 times.

Leptothrix buccalis with other bacteria, 1 time.

PRIMARY TUBERCULOSIS OF THE PHARYNGEAL LYMPHOID TISSUE.

In the following résumé only the literature of primary tuberculosis of the pharyngeal tonsil is considered. For a discussion of this lesion in the faucial tonsil, consult the admirable article by Friedmann,² which contains the results of original study, and a very complete bibliography.

PREVIOUS STUDIES. Although William Meyer³ and Trautman⁴ were both of the opinion that adenoids were in some way related to so-called scrofula, the latter after careful search was unable to find any evidence of tuberculosis in the hyperplastic pharyngeal lymphoid tissue of such cases. Passing over the work of the numerous observers who found evidence of tuberculosis in this tissue in tuberculous subjects, we find several writers—Wendt,⁵ Megevand,⁶ Suchannek,⁷ Dmoschowski⁸—describing isolated cases of primary tuberculous infection of adenoids. These, according to Piffi,⁹ are the first reported observations on this subject.

In 1892 Pilliet¹⁰ studied, by histological methods, ten specimens of adenoids removed from as many persons, and found one containing giant cells and necrosis. The next investigation, also wholly histological in character, was made by Lermoyez,¹¹ who examined thirty-two

¹ Gourc. *L'Amygdale de Meyer*; *Annales de maladies de l'oreille et du larynx*, 1897, tome xxiii. p. 437.

² Friedmann. *Beitrag zur Path. Anat. u. z. allg. Path.*, 1900, Bd. xxviii. S. 66; also Labbé and Levi-Sirugue, *Gaz. des Hôp.*, 1900, No. 20, p. 193.

³ Meyer. *Op. cit.*

⁴ Trautman. *Anat. path. u. klin. Studies über Hyperplasia de Rachentonsil*, Berlin, 1896. *Handbuch d. Ohren. v. Schevartze*, 1898, Bd. ii. S. 135.

⁵ Wendt. *Ziemssen's Handbuch*, 1874, Bd. vii.

⁶ Megevand. *Contribution à l'Étude des maladies de la voute et du pharynx*, Geneva, 1887.

⁷ Suchannek. *Ziegler's Beiträge*, 1888, Bd. iii.

⁸ Dmoschowski. *Ibid.*, 1891. Bd. x.

⁹ Piffi. *Zeitsch. für Heilkunde*, 1899, Bd. xx. S. 297.

¹⁰ Pilliet. *Bull. de la Soc. Anat.*, Paris, 1892, tome vi. p. 236.

¹¹ Lermoyez. *Bull. et mém. Soc. méd.*, Paris, 1894, tome xi. p. 569.

specimens, two of which showed morphological evidence of tuberculosis. The studies of Dieulafoy¹ quickly followed and were based entirely on the results of animal inoculations. Seven (20 per cent.) of the specimens showed tuberculous lesions.

It was not until later that a real and wide-spread interest was taken in the matter.

Quickly following these reports came that of Pilliet's,² who had examined by sections another series of forty specimens. In none did he find any evidence of tuberculosis. Brieger³ examined seventy-eight specimens and found morphological evidence of tuberculosis in five. Moure,⁴ on the other hand, by a study of sections from forty specimens, succeeded in finding tuberculous lesions in only one. By similar study of one hundred specimens, Broca⁵ did not find evidence of tuberculosis in a single one. But Gottstein,⁶ with his series of thirty-three specimens, was much more fortunate. The examinations were entirely histological, but he found three specimens containing lesions morphologically characteristic of tuberculosis.

Pluder and Fischer,⁷ from the histological examination of thirty-two adenoids, found five to be tuberculous, 16 per cent. Tubercles were found only in the lymphoid tissue of the mucosa and never in the deeper portions. Bacilli were present only in the affected parts and in small numbers; they were never found in the epithelium or healthy lymph follicles. The nasal and pharyngeal mucus did not show bacilli. The writers regard these cases as true examples of primary tuberculosis, as all the patients were otherwise healthy.

Brindel,⁸ with his sixty-four cases, found 12 per cent. tuberculous. The diagnosis was based on finding giant cells, etc. Bacteriological examinations were made in but one case; inoculations in none. Luzzatti⁹ investigated by section study fifty adenoids, but failed to find any showing tuberculous lesions.

Gourc¹⁰ examined two hundred and one cases; eighteen of the individuals had hereditary (?) and seventeen acquired tuberculosis. He made use of cultures, inoculations, and histological examinations without finding evidence of tuberculosis. In none did he find any evidence of tuberculosis. He concludes that while he cannot deny the occurrence of tuberculous infection of adenoids it is extremely rare.

¹ Dieulafoy. Bull. de l'Acad. Méd., Paris, 1895, tome xxxiii. p. 437.

² Pilliet. Discussion by Cornil. Bull. de l'Acad. Méd., Paris, 1895, p. 498.

³ Brieger. Verhandl. deutsch. Otol. Gesellsch., Jena, 1895, also 1898.

⁴ Moure. Bull. de l'Acad. Méd., Paris, 1895, p. 570.

⁵ Broca. Annales de mal. de l'oreille et du larynx, 1896, tome xxii. p. 317.

⁶ Gottstein. Berlin. klin. Woch., Bd. xxxiii. 8, 689.

⁷ Pluder and Fischer. Arch. ot Laryng. u. Rhinologie, 1896, Bd. iv. S. 372.

⁸ Brindel. Revue hebdomadaire de Laryng., d'Otologie et du Rhinologie, 1896, tome xvi. p. 881.

⁹ Luzzatti. Giornale dell'Accademia Med. di Torino, 1897, Nos. 7-9.

¹⁰ Gourc. Thèse de Paris, 1897.

McBride and Turner¹ examined one hundred adenoids and found three with lesions of tuberculosis. The diagnosis of the lesion was based entirely on morphological considerations. No tubercle bacilli were found in any of the sections.

In a series of two hundred specimens investigated by Lewin,² evidence of tuberculosis was found in ten; twenty of the specimens were studied by animal inoculations, with one positive result; the remainder were examined only histologically. This observer reached the following conclusions:

1. Five per cent. of the hyperplastic pharyngeal tonsils contain tuberculous lesions.

2. The lesion resembles the so-called tumor form of tuberculosis of mucous membranes. It is characterized by the absence of all recognizable external signs—"latent" tuberculosis of the tonsils.

3. This latent tuberculosis may apparently be the first and only lesion of tuberculosis in the body.

4. It is usually associated with more extensive tuberculosis of other organs, especially of the lungs, although this at the time of operation is often not apparent.

5. Tuberculosis of the pharyngeal tonsil is relatively frequent in pulmonary tuberculosis.

6. The tuberculous process may affect normal tonsils as well as hyperplastic ones. It is doubtful whether the toxins induce the hyperplasia. The tuberculous process may delay the involution of the hyperplastic tonsil.

7. The tubercle bacillus plays very little part in the etiology of pharyngeal tonsil hyperplasia.

8. The tuberculous lesion may be entirely removed by extirpation of the tonsil.

Scheibner³ also, by histological methods, examined fourteen adenoids, in none of which he found any lesion of tuberculosis.

Piffi⁴ examined one hundred adenoids taken from clinics and private practice. Three showed histological tuberculosis, 3 per cent. No animal inoculations were made. Seven of the patients had tuberculosis. All the sections were stained for bacilli. The epithelium showed many defects and contained "becherzellen" of different sizes. One tuberculous adenoid showed tubercle bacilli, and all three specimens caseation and miliary tubercles.

Wex,⁵ in a series of examinations of stained sections from two hun-

¹ McBride and Turner. Op. cit.

² Lewin. Arch. of Laryng. u. Rhinologie, 1899, Bd. ix. S. 377.

³ Scheibner. Deutsch. med. Woch., 1899, Bd. xxv. S. 343.

⁴ Piffi. Zeitsch. f. Heilkunde, Bd. xx. S. 297.

⁵ Wex. Zeitsch. für Ohrenheilk., 1889, Bd. xxxiv. S. 207.

dred and ten cases, found seven specimens tuberculous, 3.33 per cent. In six of these bacilli were found. He found also a number of other adenoids that showed giant cells only, and which he does not consider proper to class as tuberculous. Hynitsch¹ similarly examined one hundred and eighty pharyngeal tonsils; seven were found to be tuberculous, 3.9 per cent. Tubercle bacilli were found in only three of the specimens.

Baup² made sections from forty-eight adenoids, forty-five of which he also inoculated into guinea-pigs. Only one positive result was obtained.

J. Wright³ studied fifty-one adenoids, most of them by section study only, a few by animal inoculations, but in none was he able to demonstrate any evidence of tuberculosis.

Another study of eighty-five adenoids by Dr. George Blumer,⁴ of the Bender Hygiene Laboratory, Albany, New York, revealed four containing tuberculous lesions; histological studies alone were made, and in each case tubercle bacilli were demonstrated in the sections. This gives 4.7 per cent. of tuberculous adenoids.

WRITERS' STUDIES. Seventy-five consecutive specimens of adenoids were tested for tuberculosis. About two-thirds of them were obtained from the Children's and from the Nose and Throat Departments of the Vanderbilt Clinic, New York City, and the remainder from the New York Foundling Hospital, the New York Eye and Ear Infirmary, and from several private sources.⁵ So far as could be ascertained the children from whom the specimens came were, aside from the adenoid trouble, quite healthy.

METHODS OF PROCEDURE. The material was collected immediately after operation in clean bottles. As soon as possible thereafter the whole adenoid was carefully washed by repeated shakings in sterile normal salt or Dunham's solution. It was then placed in a sterile Petri dish, cut into four equal parts with sterile instruments, using the alternate pieces for inoculation purposes; the remainder was placed in 95 per cent. alcohol for subsequent histological examination. With the exception of the first seven specimens the portion of tissue set aside to be inoculated was reduced to a pulp, the juice therefrom filtered through sterile cheese-cloth, and the filtrate was then inoculated beneath the skin of the groin of a guinea-pig. In the first seven cases the alternate pieces were merely introduced subcutaneously without

¹ Hynitsch. *Zeitsch. für Ohrenheilk.*, 1899, Bd. xxxiv. S. 184.

² Baup. *Op. cit.*

³ Wright. *New York Medical Journal*, 1900, vol. lxxi. p. 508.

⁴ Blumer. *Personal communication.*

⁵ For most of the material investigated we are greatly indebted to Drs. Huber and Simpson, of the Vanderbilt Clinic, and also to Dr. Holt, of the New York Foundling Hospital, and Dr. Mayer, of the New York Eye and Ear Infirmary.

reducing to a pulp. All the animals were kept under the same conditions.

Two animals died on the sixteenth and twentieth days, respectively ; but in both instances careful microscopical and histological study of the tissues, including seats of inoculation, failed to show any tuberculous lesion or tubercle bacilli. All the other animals died or were killed after the twenty-seventh day ; most of them, however, died or were killed after the fortieth day. The presence or absence of tuberculosis in the inoculated animals was always tested by microscopical examination of the tissues at the seat of inoculation and elsewhere. Tubercle bacilli were always demonstrated in the lesions of the positive cases.

Pieces of hardened adenoid tissues were embedded in celloidin and stained with hæmatoxylin and eosin for general morphology ; and Ziehl-Neelsen's method was employed for the demonstration of tubercle bacilli in the tissues. Tubercle bacilli were looked for in the section of only those specimens which induced tuberculosis in the guinea-pigs, since it was assumed that the far more delicate inoculation test was decisive. Sections from those specimens were persistently stained until tubercle bacilli could be demonstrated. Upward of one hundred and twenty-five sections were thus examined in cases 35 and 55, respectively, before this organism was found in the tissues, and then only in small numbers.

RESULTS OF THE ANIMAL EXPERIMENTS. Out of the seventy-five specimens tested, twelve induced tuberculosis in the inoculated animals ; hardened tissues of eight of the specimens contained both tubercle bacilli and lesions histologically more or less characteristic of tuberculosis.

The remaining four specimens contained tubercle bacilli, but presented no histological evidence of tuberculous lesions.

MORPHOLOGY OF THE TUBERCULOUS ADENOIDS. Of the twelve specimens which contained tubercle bacilli, only eight showed histological lesions of tuberculosis—giant cells, caseation, and epithelioid cells. In the remaining four specimens none of these elements were observed in those portions containing tubercle bacilli or elsewhere. In all cases the bacilli were more or less close to the surface and few in number ; the lesions of seven cases were likewise peripherally situated, close to the epithelium and quite focal in character. Four showed single tubercles ; in two of the specimens the histological lesion was diffuse but not extensive. One specimen only showed a lesion in the middle of the adenoid tissue, consisting of a single tubercle.

The morphology of the tubercles was generally characteristic ; almost all seemed to be of recent formation. Very little caseation was noted ; and giant cells were few in number or not present.

TABLE OF RESULTS.

| | Specimen. | Source. | Time animal was observed. | Autopsy finding. | Histological tubercle. |
|----|-----------|--------------------|---------------------------|---|------------------------|
| 1 | 4 | Foundling Asylum. | Killed 32d day. | Generalized tuberculosis. | Several. |
| 2 | 9 | Foundling Asylum. | Died 27th day. | Generalized tuberculosis. | Several. |
| 3 | 23 | Vanderbilt Clinic. | Died 33d day. | Tuberculosis at seat of inoculation and abdominal glands. | One. |
| 4 | 27 | Dr. M. | Died 24th day. | Tuberculosis at seat of inoculation. | None. |
| 5 | 36 | Vanderbilt Clinic. | Killed 49th day. | Generalized tuberculosis. | None. |
| 6 | 38 | Foundling Asylum. | Died 20th day. | Tuberculosis at seat of inoculation, and abdominal lymph nodes. | Several. |
| 7 | 42 | Foundling Asylum. | Died 30th day. | Tuberculosis at seat of inoculation, spleen, lymph nodes. | One. |
| 8 | 49 | Foundling Asylum. | Died 38th day. | Generalized tuberculosis. | None. |
| 9 | 51 | Dr. B. | Killed 27th day. | Tuberculosis at seat of inoculation, and lymph nodes. | One. |
| 10 | 55 | Vanderbilt Clinic. | Died 16th day. | Tuberculosis at seat of inoculation. | None. |
| 11 | 63 | Vanderbilt Clinic. | Died 25th day. | Tuberculosis at seat of inoculation, and lymph nodes. | Several. |
| 12 | 71 | Vanderbilt Clinic. | Died 29th day. | Generalized tuberculosis. | One. |

Unless the lymphoid hyperplasia, at all events a part of it, be considered in the four specimens presenting no typical tuberculous lesion as having been induced by the bacillus tuberculosis—and such tissue changes following tubercle bacillus infection are by no means uncommon, especially in lymph nodes—then in these cases no lesion can truly be said to have developed. Nearly all observers agree as to focal character of the lesions.

The following gives the percentage results :

| | |
|--|--------------------|
| Total number of specimens tested | 75 |
| Number containing bacilli and showing histological lesions of tuberculosis | 8 = 10.67 per ct. |
| Number containing bacilli but no histological lesions | 4 = 5.33 " |
| | <hr/> 12 = 16.00 " |

THE VIRULENCE OF THE TUBERCLE BACILLI. The tubercle bacilli present in Specimens 4, 38, and 63 were especially studied with a view to determine their virulence.

METHODS OF PROCEDURE.¹ Cultures were usually obtained from the deep inguinal or sublumbar lymph nodes of guinea-pigs previously

¹ Vide Lartigau. A Study of the Variations in Virulence of the Bacillus Tuberculosis in Man. Journal of Medical Research, 1901, vol. vi. No. 2.

inoculated with tuberculous material. Cultivation was made on 6 per cent. glycerin agar-agar. From fifteen to thirty tubes were always inoculated, for very often, especially with the more virulent bacilli, development took place in one or two of the tubes only. In all cases the growth was continued until it became voluminous (four to eight weeks).

For purposes of comparative study by inoculation with the guinea-pig and rabbit, the solid masses of culture were scraped off from the moist agar, especial care being taken only to remove bits of culture free from any of the medium. Usually 5 milligrammes of the tubercle bacillus were then carefully weighed and finely suspended in 5 cubic centimetres of an indifferent fluid, such as physiological salt solution, until the density was quite uniform and microscopical examination showed no (or very few) clumps. The mixture was quickly used when prepared. Occasionally, when large amounts of the tubercle bacillus were to be inoculated, 10 or more milligrammes were weighed out instead and suspended in the same amount of fluid. If, on the other hand, small amounts of the bacillus were to be utilized, then the 5 milligrammes were suspended in 10 cubic centimetres of the salt solution. Thus, in the latter case, 10 cubic centimetres of the emulsion would equal 5 milligrammes; 5 cubic centimetres would equal 2.5 milligrammes; 1 cubic centimetre would equal 0.5 milligramme; $\frac{1}{2}$ cubic centimetre would equal 0.25 milligramme, etc. Dosage under these conditions was then always fairly accurate.

The comparative method of inoculation recommended by Arloing¹ was in other respects rigorously adhered to when this was in view; the guinea-pigs and rabbits were inoculated in pairs in the same region, with the same quantity of culture and at the same time; all inoculated animals were kept under essentially the same conditions.

The presence or absence of tuberculosis in the inoculated animals was always based on both naked-eye appearances and microscopical examinations; identification of the tuberculous lesion was always confirmed by finding tubercle bacilli in the affected tissues. Cultures were obtained from the tuberculous lesions of the livers or spleens of the guinea-pigs, except when the tuberculosis was confined to the seat of inoculation; when the latter was the case, the cultivation, perforce, was made from this focus. To recapitulate, the method of procedure in the animal tests was as follows: The inoculations were made in series, one guinea-pig and one rabbit usually constituting the animals for each set. A definite and small amount of culture was inoculated into each animal under the same experimental conditions. From the guinea-pig of the first set cultures were again obtained and a second set of animals

¹ Arloing. *Leçons sur la Tuberculose et certaines Sépticémies*, Paris, 1892, p. 161.

reinoculated under the same conditions; from the guinea-pig of the second set cultures were prepared and inoculated into a third set. This was not carried beyond the third set. The channel of infection was subcutaneous.

EXPERIMENTAL RESULTS. The results of the animal inoculations showed that the tubercle bacillus from Specimen 4 was moderately virulent. Doses of 10 milligrammes introduced beneath the skin induced only slight general tuberculosis. The bacilli from Specimens 38 and 63, on the other hand, were both of low virulence; doses of 10 or 20 milligrammes inoculated subcutaneously induced little tuberculous inflammation, usually limited to the seat of inoculation.

| | Series. | Animal. | Inoculated, dose. | Died. | Autopsy findings. |
|------------------------------------|---------|------------------------|-------------------|----------------------------|---|
| Tubercle bacillus from specimen 4 | 1 | Rabbit, 1105 grams. | 20 mg. | 41st day | Moderate tuberculosis of lungs and spleen and inoculation seat. |
| | | Guinea-pig, 455 grams. | 20 mg. | 23d day | Moderate generalized tuberculosis. |
| | 2 | Rabbit, 1080 grams. | 10 mg. | 48th day | Tuberculosis of lungs and inoculation seat. |
| | | Guinea-pig, 485 grams. | 10 mg. | 18th day | Tuberculosis inoculation seat, and abdominal lymph nodes. |
| | 3 | Rabbit, 1210 grams. | 10 mg. | 53d day | Slight tuberculosis of lungs, kidney, spleen, and inoculation seat. |
| | | Guinea-pig, 575 grams. | 10 mg. | 30th day | Tuberculosis of inoculation seat, abdominal lymph nodes, lungs, and spleen. |
| Tubercle bacillus from specimen 38 | 1 | Rabbit, 1840 grams. | 20 mg. | 38th day of other disease. | Tuberculosis inoculation seat and one nodule right lung. |
| | | Guinea-pig, 620 grams. | 20 mg. | 24th day | Tuberculosis inoculation seat, abdominal lymph nodes, and liver. |
| | 2 | Rabbit, 1160 grams. | 10 mg. | 62d day killed. | Tuberculosis inoculation seat. |
| | | Guinea-pig, 580 grams. | 10 mg. | 33d day | Moderate tuberculosis inoculation seat, and abdominal lymph nodes, and liver. |
| | 3 | Rabbit, 940 grams. | 30 mg. | 56th day | Moderate tuberculosis inoculation seat, spleen, liver, and lungs. |
| | | Guinea-pig, 575 grams. | 30 mg. | 26th day | Moderate tuberculosis inoculation seat, liver, lungs, and abdom' lymph nodes. |
| Tubercle bacillus from specimen 63 | 1 | Rabbit, 1280 grams. | 20 mg. | 48th day | Tuberculosis inoculation seat. |
| | | Guinea-pig, 785 grams. | 20 mg. | 39th day | Tuberculosis inoculation seat and abdominal lymph nodes. |
| | 2 | Rabbit, 990 grams. | 10 mg. | 81st day | Tuberculosis inoculation seat. |
| | | Guinea-pig, 610 grams. | 10 mg. | 53d day | Tuberculosis inoculation seat, abdominal lymph nodes, and one lung. |
| | 3 | Rabbit, 1275 grams. | 30 mg. | 55th day | Slight tuberculosis inoculation seat and lungs. |
| | | Guinea-pig, 485 grams. | 30 mg. | 24th day | Generalized tuberculosis. |

REMARKS ON PRIMARY TUBERCULOSIS OF THE PHARYNGEAL TONSIL.

From the review of previous studies upon tuberculosis of the pharyngeal tonsil, one is much impressed with the lack of thoroughness of many of the investigations and incompleteness of others. The tuber-

culous lesions are usually so focal and discrete, and the tubercle bacilli so few in number in the tissue, that most searching study is necessary to reveal them. This is especially true for lymphoid tissue tuberculosis. The only method, then, which may permit of accurate conclusions is the use of animal inoculations controlled by careful study of many sections of the tissue. Unfortunately, investigations of this kind are too few in number to allow at the present time definite conclusions as to the frequency with which primary tuberculosis of the pharyngeal tonsil occurs. It is especially desirable that in the future the method of investigation be along these lines and uniform, if accuracy is to be attained. It is probable from our experience that many later investigations will show primary tuberculosis in 10 per cent. or over of the clinic cases. Several other factors of importance are to be considered: the evidence of tuberculosis elsewhere in the body and the age of the individuals. The source of the material should also always be mentioned. Obviously, individuals from the poorer classes would show a higher percentage than those living under more hygienic conditions.

SOURCES OF THE BACILLUS TUBERCULOSIS IN TONSIL INFECTION. As pointed out by Dmoschowski, the sources of infection may be the inspired air, sputum, blood, and lymph.

Inspired Air. The hyperplastic pharyngeal lymphoid tissue lying directly in the path of the inspired air is naturally exposed to infection from this source. That this may be an important source of infection has been shown by Straus,¹ who, in 1894, inoculated a series of animals with the nasal secretion from twenty-nine individuals (themselves not tuberculous) frequenting places where tuberculous patients were living. The enormous number of 40.9 per cent. of all persons experimented upon contained tubercle bacilli in their nasal cavities. Recently, Noble W. Jones² has performed similar experiments, using, however, nasal secretions from healthy persons, who, to the best of his knowledge, had not been in contact with tuberculous individuals. Animal inoculations with the secretion from thirty-one persons were made, three animals becoming tuberculous, a percentage of 9.7. The objection that has been brought against this mode of testing in the case of adenoids, viz., that the patients are mouth-breathers, would seem at first glance to be well taken. Pluder and Fischer,³ however, have shown that mouth-breathing, especially in lower grades of hyperplasia, is not constant. Charles A. Parker found in 82 per cent. of sleeping children that the respiration was nasal, and in 16 per cent. at least partly so. The former writers have also shown that in cases of adenoids the soft palate does not act as a perfect barrier against infection coming through the

¹ Straus. Bull. de l'Acad. de Méd., Paris, 1894, tome xxxii. p. 18.

² Jones. Medical Record, 1900, vol. lviii. p. 285.

³ Pluder and Fischer. Loc. cit.

mouth. They also point to the fact that in many cases of adenoids the nasal passage is widened, thereby increasing the possibility of infection from this source.

The histological study of tuberculous faucial tonsils removed from individuals free from tuberculosis of the lungs also indicates that the process generally extends inward from an infection on the surface.

Sputum. This mode of infection presupposes a primary focus of tuberculosis in the lower respiratory tract or lungs. Secondary infection under these conditions has been frequently demonstrated in cases of pulmonary tuberculosis with free expectoration, notably by Strassmann, Dmoschowski, Schlenker, Kruckmann, Suchanek, Wendt, Megevand, Piffi, Habermann, and Fraenkel.

Lymph and Blood. Infection through the lymph and blood would naturally be most likely to occur in connection with acute general miliary tuberculosis. Habermann and others believe that infection can take place from the middle ear, presumably through blood or lymph channels.

Dmoschowski¹ has demonstrated bacilli in the lymph channels connecting the tonsils with the cervical glands, and Piffi² has described an ascending tuberculous infection of the lymph passages from the mediastinum. Scheibner³ and others have observed tuberculous infection of the tonsil in cases of miliary tuberculosis of short duration, in which, of course, infection from the sputum must be excluded, but which the writers believe to have been true cases of blood and lymph infection. Until more accurate observations have been made, especially with regard to the relative ages of coexisting lesions, the frequency of this mode of infection cannot accurately be determined. In general, it may be said that the majority of observers are agreed that in the case of tuberculous individuals with free expectoration the infection of the pharyngeal tonsil in all probability practically always comes from the latter source, while in individuals without lung tuberculosis the factors previously mentioned, together with certain histo-pathological findings, go to show that the source of infection is almost always from the inspired air.

THE TONSILS AS SEATS OF INFECTIONS AND PORTALS OF ENTRY OF MICRO-ORGANISMS. *Miscellaneous Bacteria.* The nasopharyngeal cavity presents several anatomical and physiological peculiarities which predispose to primary infection: 1. The presence of lymphoid tissue whose surface is not smooth, but which is provided with crypts and irregular depressions. 2. The surface is not lined with ciliated epithelium throughout. In the crypts, especially in the young, flat epithelium

¹ Dmoschowski. *Beitrag z. path. Anat. u. z. allg. Path.*, Jena, 1891, Bd. x. S. 481.

² Piffi. *Loc. cit.*

³ Scheibner. *Deutsch. med. Woch.*, 1899, Bd. xxv. S. 343.

may occur ; and (3) catarrhal conditions often arise in the nasopharyngeal mucous membrane, leading to epithelial denudation. The great liability of the tonsils to mechanical lesions necessarily also increases the predisposition to infection. These considerations apply to the pharyngeal tonsil as well as to the faucial, and especially to the former when it is in a condition of hyperplasia. The epithelium of the crypts (faucial) and so-called Stohr's canals (pharyngeal) is frequently in young persons changed to the pavement variety instead of the normal ciliated, as McBride and Turner and others have shown. These writers ascribe this change to the friction of opposing surfaces of the tonsils in a condition of hyperplasia and to the pressure of surrounding walls.

Dmoschowski believes that the ciliæ in this region tend rather to work toward the pharynx than outward. Although Hodenpyl's investigations led him to believe that the normal tonsil cannot readily be infected, it has been shown that the tonsils frequently afford places for the lodgement of bacteria because of their irregularity of contour, the presence of lacunar spaces, the rarefaction of the epithelium lining the crypts (Hodenpyl), and the exfoliation of the epithelium which leaves places of denudation. The crypts and canals are deepened and multiplied in the state of hyperplasia, which ensures a better resting-place for infected secretions and dust coming through the nose and mouth. The tonsils are, therefore, constantly exposed to pathogenic organisms contained in the nasal and buccal cavities, and to those in the inspired air and ingested food.

Important protection may be afforded by the wandering leucocytes passing to the tonsil surface, as suggested by the observations of Hugenschmidt.¹ From his experiments he concluded that the saliva possesses positive chemiotatic properties, which explain the diapedesis taking place in the normal tonsil to destroy bacteria. But more important light has been thrown upon this side of the question by the extremely interesting experiments of Manfredi.² In a most elaborate article, this investigator argues from the results of his studies that the function of lymphoid tissue is one of protection. He brings forward sufficient evidence to make it probable that such protection occurs in three ways: First, by filtration of the bacteria; second, by an attenuation of the virulence of micro-organisms that reach them; and third, by the whole body obtaining a greater or less immunity while the first two processes are in operation.

As to the capacity for absorption of micro-organisms by the faucial tonsil, Ribbert³ found that the bacillus of diphtheria is not absorbed in rabbits by the buccal mucous membrane, but may be by tonsils

¹ Hugenschmidt. *Annales de l'Inst. Pasteur*, 1896, tome x. p. 545.

² Manfredi and Viola. *Palermo*, 1898.

³ Ribbert. *Deutsch. med. Woch.*, 1897, Bd. xxiii.

whose epithelium shows more or less desquamation. Buschke¹ also concludes that the tonsils are portals of entry for pus-producing organisms. Pluder² contends that tonsillar protection to the invasion of bacteria is in reality small, since the tonsils are frequently diseased, and hence portals for the entrance of micro-organisms.

These observations indicate that the faucial and pharyngeal tonsils in the more or less marked pathological condition in which they are often found are frequently open to the invasion of bacteria.

The recent observations of Plottier³ are of the greatest etiological interest. A series of 268 cadavers of children was examined in respect to adenoid vegetations, and they were only found seven times in fifty-four cases of measles; once in twenty-five cases of gastro-enteritis, in five of scarlet fever, and in twelve of tuberculous meningitis; not at all in five cases of hereditary syphilis and sixteen of athrepsia. On the other hand, they were found six times in twenty-seven cases of bronchopneumonia; five times in twenty cases of pulmonary tuberculosis; three times in seven cases of whooping-cough; and twenty-nine times in fifty-three cases of diphtheria.

BACILLUS TUBERCULOSIS. Nearly all observers are agreed that hyperplasia of the pharyngeal lymphoid tissue is very commonly associated with enlargement of the lymph nodes, especially the submaxillary and cervical lymph nodes. Meyer⁴ himself vaguely expressed the consideration that tonsillar lymphoid hyperplasia might possibly stand in some relation to scrofula. When the investigations of Baumgarten,⁵ Sims Woodhead,⁶ and Kruckmann,⁷ showing conclusively that micro-organisms may pass through the epithelium of the throat into the cervical lymphatics, are considered in connection with the fact that tubercle bacilli have been demonstrated in a certain number of adenoids, the recent observations of Moore become of great interest. Moore⁸ examined superficial lymph nodes removed from twenty cases (eighteen cervical, two axillary). Twenty of these cases were tuberculous; and six of them presented inflammatory lesions of the faucial or pharyngeal tonsil. In one case adenitis appeared to follow adenoids. He believes that in most cases the infection comes from the nose, throat, and mouth, and does not ascend from the mediastinum, the pharyngeal, lingual and faucial tonsils being the chief portals of entry in cervical lymph-node tuberculosis.

¹ Buschke. *Deutsch. Zeitsch. für Chir.*, 1894, Bd. xxxviii.

² Pluder. *Loc. cit.*

³ Plottier. *Revue Hebdomadaire de Laryngologie* (Bordeaux), October 14, 1900.

⁴ Meyer. *Loc. cit.*

⁵ Baumgarten. *Vide Moore.*

⁶ Sims Woodhead. *Report of Royal Commission on Tuberculosis*, vol. iii. p. 115.

⁷ Kruckmann. *Virch. Arch.*, 1894, Bd. cxxxviii., S. 184.

⁸ Moore. *Journal of Pathology and Bacteriology*, 1900, vol. vi. p. 94.

Jesser¹ believes, as does Zarniko,² that in many cases scrofula is the result of an infection from the tonsils, more especially the pharyngeal tonsil. Jesser quotes four very interesting cases in which, after the removal of adenoids in young children, the symptoms of scrofula subsided. The etiological relation of tonsil infection to scrofula is also maintained by Morgan³ and Gallois.⁴

Delepine and Yonge,⁵ on the other hand, examined the tonsils in seventeen cases of tuberculous lymphadenitis without pulmonary tuberculosis, and in none did they find tuberculosis of the tonsils. Like Cheyne,⁶ they tend to minimize the importance of tonsil infection in scrofula.

Baup, by introducing tubercle bacilli into the pharynx of dogs, always induced tuberculosis of the cervical lymph nodes; he, therefore, also believes that the tonsils are frequent portals of infection in cervical lymph-node tuberculosis.

Certain writers have also been disposed to lay stress on the tonsils as portals of infection in middle-ear tuberculosis, or Pott's disease (Busse), or meningitis (Demme, Heller, and Ziem).

CONCLUSIONS. 1. Adenoids consist essentially of hyperplastic pharyngeal lymphoid tissue. The epithelium and fibrous tissue changes are inconstant and variable, and independent of the age of the patient. The new-formed fibrous tissue is largely perivascular in distribution. It may occasionally be one of the factors in the process of disappearance of the adenoid.

2. The hyperplastic pharyngeal tonsil often contains micro-organisms, and these are mainly pyococcal forms. The bacteria for the most part lie near the surface; and the infection usually occurs from the surface, with or without demonstrable lesion of the epithelium.

3. Primary tuberculosis of adenoids is probably more common than most previous studies show. Sixteen per cent. of our series contained tubercle bacilli, 10 per cent. with characteristic lesions of tuberculosis. The tubercle bacilli were present in small numbers.

4. The lesions in primary tuberculosis of the adenoid are generally close to the epithelial surface and focal in character. Occasionally they may be found in the deeper parts of the pharyngeal lymphoid tissue.

5. The pharyngeal tonsil may be a portal of entry for the tubercle bacillus and other micro-organisms in localized or general infections.

¹ Jesser. Münch. med. Woch., 1898, Bd. xiv. No. 23.

² Zarniko. Loc. cit.

³ Morgan. British Medical Journal, 1899, vol. ii. p. 458.

⁴ Gallois. Bull. Med., 1897, p. 1161.

⁵ Delepine and Yonge. Vide Moore, Journal of Pathology.

⁶ Cheyne. British Medical Journal, 1899, vol. ii. p. 1659.

A CASE OF INTRATRACHEAL COLLOID STRUMA; OPERATION;
RECOVERY.*

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AMONG the problems that the throat specialist is called upon to solve there are few, perhaps, that present more difficulties than some of the conditions causing tracheal stenosis.

A large majority of these cases of tracheal stenosis are due to pressure on the trachea by tumors external to it, to old syphilitic adhesions or gummata, and to certain intratracheal new-growths, benign and malignant.

One of the rarest forms of intratracheal growth, usually causing great stenosis, and making operative procedures imperative on account of the alarming dyspnoea by which it is accompanied, is, without question, the tumor springing from the thyroid tissue in the lower laryngeal cavity and the upper part of the trachea, sometimes called an intralaryngeal or tracheal "accessory" thyroid tumor.

There are now on record, including the case which I have the pleasure of reporting to you this evening, ten positive and two doubtful cases of intralaryngeal and tracheal tumors, originating in thyroid tissue: Eight of these were observed in different parts of Germany.

The author's case is the second reported in the United States, and the tenth on record. The other case was reported by Freer, in the *Journal of the American Medical Association*, March 30, 1901. There is no doubt that other tumors of this nature, in which the diagnosis was not verified by operation and microscopical examination, have been observed, but a very thorough search of the literature failed to reveal the report of any other case (with the exception of Freer's) like the author's.

I may mention in passing, in order to show the rarity of this form of benign intratracheal growth, that Bruns,⁷ up to 1898, had collected altogether ninety-one cases of benign tumors occurring in the trachea, and of these only seven were cases of intratracheal struma. Since then two others, those of Baurowics and of Freer, have been reported, making nine only on record.

There have been, of course, a considerable number of accessory thyroid glands and thyroid tumors reported, occurring in more or less remote

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situations, viz.: the cases of intrathoracic struma, those that are substernal in position and connected directly with the thyroid gland, the accessory thyroid tumors at the base of the tongue, the parathyroid glands found in the upper third of the anterior surface of each lobe of the thyroid, which Gley has found in almost all animals, and some in still other parts of the air passages. (Osler,¹⁰ Gruber,¹¹ Gley,¹² Warren,¹³ Schadle,¹⁴ Virchow, Wolf,¹⁵ Butlin,¹⁶ and others.) These cases are only mentioned here because they are of some interest in connection with the subject under discussion.

The history of the writer's case, which in some respects differs from any of the other recorded cases, is as follows:

Mrs. M. R., aged thirty-two years, was referred to me by Dr. J. B. Harvie, of Troy, N. Y. Her father died of some kidney trouble at the age of thirty-three years; her mother is living and well. Her two brothers and two sisters are living. One sister, it is interesting to note, has a goitre of considerable size. One sister and brother died in childhood. She had the usual diseases of childhood, but otherwise has always been well. Her menstruation commenced at the age of fourteen. She has always been regular, but has usually had considerable pain the first two days of each period. Her last menstruation was in June, 1901. She is now five months pregnant, and has two children, both of whom are living and in good health. About seven years ago the patient noticed a contraction of the muscles of the left side of the neck, and that the glands on the same side were swollen. Three weeks later she first noticed a difficulty in breathing, which was increased by the least exertion. The dyspnoea, which had irregularly but steadily increased, particularly during her present pregnancy, finally became so alarming that she was sent to the Troy Hospital, November 12, 1901, by her physician, Dr. Harvie. I saw her, with Dr. Harvie, on the following day for the first time. She was breathing with considerable difficulty, and there was some cyanosis of the lips.

On examining the neck a goitre of moderate size was found, the left lobe of the thyroid being somewhat, and the isthmus decidedly, enlarged. There was a marked inspiratory stridor, and on palpation over the upper part of the trachea a decided inspiratory thrill could be felt, a symptom that appears to be pathognomonic of high tracheal stenosis. No other enlarged glands in the neck were found. The examination of the lungs was negative, with the exception of a somewhat prolonged expiratory murmur at the left apex. Other organs normal. An examination of the nose, nasopharynx, pharynx and larynx, showed normal conditions.

On laryngoscopic examination, however, a tumor of considerable size, springing from the posterior and left lateral walls of the trachea, almost completely filling its lumen, was seen during deep inspiration. (Fig. 1.) The growth appeared to extend from about the first tracheal ring downward along the posterior wall. The tumor was regular in outline, and covered with a perfectly smooth, normal-looking mucous membrane. Several vessels were seen running over its surface.

During the next few days iodide of potash was given, 30 grains every three hours. The dyspnoea, however, becoming more serious every

day, an operation was considered imperative, and was performed November 16th.

Under ether, with the patient's head low, an incision extending from the lower margin of the thyroid cartilage to a short distance above the episternal notch was made. On account of the enlarged isthmus which covered the parts it was not considered wise to attempt to perform a preliminary low tracheotomy. The isthmus was found to be much enlarged, making the exposure of the trachea difficult. At this time the patient's breathing became very bad, and a high tracheotomy was performed, the isthmus being pushed down as much as possible. In cutting through the tracheal wall a small incision was accidentally made in the tumor, causing a most troublesome hemorrhage, which was finally controlled with a sterile solution of adrenalin chloride, 1:1000. The thyroid isthmus was then firmly ligated on both sides with catgut, and divided. The trachea being now thoroughly exposed a low tracheotomy was performed, the upper tube taken out, and the tracheal rings above the tube were split, as was also the cricoid. The tumor, which

FIG. 1.



was over 5 cm. long, extended from the first tracheal ring downward along the posterior wall, and almost filled the tracheal lumen. It was resilient to the touch, and was found to be firmly attached, by a very broad base, to the posterior and left lateral walls of the trachea, from which it could not be separated. It was, in fact, part of the tracheal wall. As much of the growth as possible was removed with snare and curette, the adrenalin solution being used continuously, on account of the severe and troublesome hemorrhage.

Because of the hemorrhage, and because the patient's condition was not entirely satisfactory, attempts to separate the portion of the tumor that remained from its attachment to the posterior wall had to be finally abandoned. The trachea was brought together with catgut sutures, the external wound being closed by a continuous subcutaneous silkworm-gut suture. A gauze tampon was inserted above the tracheotomy tube to prevent blood from oozing down.

Patient's condition was very satisfactory for several days after the operation, but on the fourth day her temperature, which had been nearly normal, suddenly went up to 102° F., and she developed a

double pneumonia. Nitroglycerin was administered, at first $\frac{1}{10}$ grain every hour by mouth, with $\frac{1}{10}$ grain of strychnine every three hours hypodermically. Whiskey was also given every hour. On the following night, November 21st, patient was in a very dangerous state, several times being in a condition of extreme collapse. Hypodermic injections of 5 minims of a 1 per cent. solution of nitroglycerin every twenty minutes during the collapse were each time followed by the most marked results, the pulse improving in quality within a few minutes after each hypodermic.

The next morning, November 22d, the patient again collapsed, respiration almost ceasing. It was evident that the trachea below the tube was filled up. The tube was taken out, the patient held over the side of the bed with the head very low, and with a long, narrow curved forceps I was finally able to remove from low down in the trachea several masses of almost solid mucus which nearly filled its lumen, and which she could not have expelled through the tube. The patient's breathing improved at once, and from this time on she steadily improved. For nearly two weeks, however, from the onset of the pneumonia her heart acted badly, the pulse being rather irregular, not of good quality, and never getting below 120. During this time nitroglycerin was administered continuously at hour intervals, with strychnine $\frac{1}{10}$ grain hypodermically every four hours. The nitroglycerin had to be stopped at times for part of a day when the patient complained too much of throbbing and pain in the head, and digitalis was given instead, but I would like to mention at this time that I do not believe there is any doubt that the large amount of nitroglycerin the patient received, and particularly the large doses given hypodermically during collapse, saved her life. She was discharged from the hospital in excellent condition on the twenty-ninth day after the operation, still wearing the tracheotomy tube. She was at that time about six months pregnant.

The portions of the growth removed were sent to Dr. Blumer for examination, who reported as follows:

BENDER HYGIENIC LABORATORY,
ALBANY, N. Y., December 21, 1901.

I enclose report on the tumor from the interior of the trachea:

The specimen submitted consists of ten small fragments of growth, averaging half a centimetre in diameter. The tissue is pinkish in color and somewhat translucent. Microscopical examination shows that covering the growth on one side is normal tracheal epithelium. Beneath this is submucosa, but tracheal glands are not present in the section, and presumably lie behind the tumor. The tumor itself is made up of tissue which may be described in brief as exaggerated thyroid tissue. The alveoli are in arrangement exactly similar to normal thyroid, and consist of spaces lined by cubical epithelium and containing quantities of colloid. They differ from normal thyroid in the great variation in size and shape of the alveoli. (Fig. 2.) The amount of connective tissue between the alveoli is about that normally seen in the thyroid gland. The connective tissue contains a large number of bloodvessels.

Diagnosis. Colloid struma, originating in thyroid tissue situated beneath the submucosa of the trachea.

GEORGE BLUMER.

It was not considered safe to permanently take out the tracheotomy tube until after her confinement, on account of the danger of a sudden swelling of the part of the tumor that had to be left. When the last laryngeal examination was made I found that this remnant had contracted considerably, so that she breathed without difficulty when the tracheotomy tube was temporarily removed, and I have no doubt that after she is confined it will almost disappear.

The origin of intralaryngeal and tracheal thyroid tumors was up to the time of Paltauf's investigations one of the most interesting enigmas in medicine, and for its solution it will be necessary to briefly review the reported cases.

FIG. 2.



Drawing of section; low power.

CASE I.—(Ziemssen's,¹ 1875.) The patient, a shoemaker, aged thirty years, developed laryngeal dyspnoea during the latter part of October, 1875, which rapidly increased. He had a goitre of moderate size. A tracheotomy was performed for the relief of the dyspnoea. Death on the following day of erysipelas developing in the wound. At the autopsy a tumor was found in the larynx, left side, which extended from the middle of the cricoid cartilage, and was about 2 cm. long and 1 cm. thick. It had a perfectly smooth surface and an intact mucous membrane. Microscopical examination disclosed that the tumor was made up of thyroid tissue, and, also, that the goitre had penetrated on the left side, between the cricoid and thyroid cartilages, into the lower part of the larynx.

CASE II.—(Reported by Bruns.²) The patient, a male, aged thirty-two years, had had since his seventeenth year difficulty in breathing, which had gradually increased. Examination of the neck showed a slight enlargement of both lobes of the thyroid. On laryngeal exam-

ination a tumor was seen in the lower part of the larynx, almost filling its lumen, and springing from the right lateral wall and part of the posterior wall. Operation, laryngo-tracheotomy. Growth extended from the fourth tracheal ring upward to close under the right vocal cord. On microscopical examination it was found to consist of thyroid tissue, with slight colloid degeneration.

CASE III.—(Heise-Bruna,³ 1877.) Case of a girl, aged fifteen years, who for three years had had difficulty in breathing. Lateral lobes of the thyroid were not enlarged, but the isthmus could be felt as a node as large as a cherry. On laryngeal examination a subglottic tumor was seen on the posterior and right lateral walls of the larynx, slightly nodular, and covered with intact mucous membrane, taking up two-thirds of the lower laryngeal cavity. Operation, laryngo-tracheotomy. The tumor was found to extend from the second tracheal ring to close under the glottis, and had a very broad base. On microscopical examination it was found to consist of thyroid tissue.

CASE IV.—(Heise;⁴ also from the Tübingen clinic, 1885.) The patient, a male, aged twenty-six years, had had slight dyspnoea after exertion, since his twenty-fourth year, which gradually got worse. The thyroid was not enlarged. Laryngeal examination, followed by tracheotomy, showed the presence of a tumor, 5 cm. long, extending from the first tracheal ring downward along the posterior tracheal wall. On microscopical examination the tumor was found to be made up of thyroid substance, showing slight colloid degeneration.

CASE V.—(Roth's.⁵ Reported by Bruns, 1888.) Case of a woman, aged forty years. At the autopsy a small tumor, covered with mucous membrane, was found under the cricoid cartilage. On microscopical examination it was found to consist of small follicles with fatty degenerated epithelium.

CASE VI.—Paltauf's,⁶ 1892.) A young woman, aged twenty-nine years, was brought to Albert's clinic, in Vienna, on account of alarming dyspnoea, and an immediate tracheotomy was performed. Four weeks before difficulty in breathing had developed, and became alarming three days before the operation. Examination of the neck showed the presence of a struma of moderate size. On laryngeal examination several red tumors were seen under the vocal cords, nearly closing the laryngeal lumen. Three weeks after the operation a large abscess of the left thyroid lobe developed, death resulting a week later. At the autopsy a small tumor was found, extending from the lower border of the thyroid cartilage to the cricoid cartilage. Microscopical examination showed this to be typical thyroid tissue.

CASE VII.—(Bruns,⁷ 1898.) Case of a girl, aged twenty-four years, who had a small median goitre. On laryngeal examination a tumor was seen on the anterior wall of the lower laryngeal cavity and upper part of the trachea. Operation, laryngo-tracheotomy. An adhesion of the isthmus to the trachea was found, and *directly opposite* in the trachea the tumor was situated. On microscopical examination it was found to be a struma colloides.

CASE VIII.—(Baurowics',⁸ 1898.) Case of a girl, aged twenty-one years, who had had an operation for tracheotomy. Under the left vocal cord a tumor was seen attached to the left lateral and posterior walls, filling the lumen of the lower laryngeal cavity, and extending down to the fourth tracheal ring. Operation, laryngotomy. On microscopical

examination the growth was found to consist of thyroid tissue, with slight colloid degeneration.

CASE IX. (Freer's,⁹ 1901.)—This patient, a woman aged thirty-two years, had a thickening of the mucous membrane below the cords, forming a ledge which projected into the laryngeal lumen around its whole circumference. This thickening extended down the trachea to the third or fourth ring, forming a prominent tumor on the posterior wall. On examination of the neck the thyroid could be felt. A tracheotomy was performed on account of a sudden subglottic swelling. Portions of the intratracheal growth were afterward removed through the tracheal wound, and were found to be made up of thyroid tissue. An operation for the removal of the growth itself was not performed.

Doubtful cases. Radestock's¹⁷ case was formerly included under this head. In his case a tumor was found low down in the trachea, at the entrance to the main bronchus, closing its lumen entirely. Since the publication of his case it has been claimed by Paltauf and Kolisko¹⁸ that it was not a true case of intratracheal struma, but that the microscopical examinations proved it to be a case of adenoma. Radestock's case was undoubtedly similar to the one reported by Scheuer¹⁹ in 1893. Scheuer's case also turned out to be a true adenoma on the posterior wall of the trachea, extending from the sixth to the eighth rings. The other doubtful case was reported by Ball.²⁰ In this there was a tumor filling the upper part of the larynx and complicating a goitre. The laryngeal tumor was probably malignant, although Semon was of the opinion that it might have consisted of hypertrophied thyroid tissue. Mayer's case,²¹ in which there was a struma nodule on the right wall of the trachea which had grown through the wall from a large struma, was also proved to be a malignant adenoma. For the reasons given, and because the clinical data were insufficient, Radestock and Ball's cases were not included in the above table. The statement made by Freer that his case is the *tenth* case on record is then *not* correct. His was the *ninth*. He also stated that five cases have been reported from Bruns' clinic. Only *four* have been reported from the Tübingen clinic, two by Bruns and two by Heise.

The two theories as to the origin of this most interesting form of tumor that have received the most consideration are those of Bruns and Paltauf. Bruns and Heise,² the chief exponents of the embryonal theory, held that in *intra-uterine* life a small accessory thyroid lobule from aberrant embryonic rudiments of the thyroid gland (*Angeborene Verlagerung von Schilddrüsengewebe*), must have, in such cases, been present in the foetal larynx or trachea. This lobule, developing about the time of puberty, resulted in the true intralaryngeal or tracheal "accessory thyroid tumor." Paltauf⁶ was able to prove in his case, by microscopical examinations, that this theory was not tenable. In his case there was a connection between the intratracheal growth and the thyroid gland externally. The thyroid was so firmly attached to the cricoid cartilage and upper three tracheal rings that it could not be separated from them. The space between the cricoid and first tracheal ring, and a portion of the external lateral lobe of the thyroid, which

was firmly adherent to the cartilage at this point, were examined microscopically, and he was able to prove positively that the thyroid tissue had penetrated (through the interstitial membranes) to the perichondrium and submucosa on the inner surface of the trachea, the cartilage itself remaining intact. So it is not necessary, as Heise thought, that in order for the thyroid tissue to penetrate into the trachea there must be a perforation, or at least a defect in the tracheal wall. Ziemssen,¹ too, observed that the struma had penetrated the lower cavity of the larynx between the cricoid and thyroid cartilages, and Baurowics was able to demonstrate the same origin of the intratracheal growth in his case. This observation of Paltauf's is the first instance on record of normal thyroid gland tissue penetrating to the interior of the larynx and trachea, although Orth,² in his *Pathological Anatomy*, makes the statement, "that strumas, but particularly the *malignant* neoplasms, could penetrate into the air passages, i. e., into the larynx."

The only way that this penetration of normal thyroid tissue in Paltauf's case can be explained is by the intense adhesion of the right and left lateral thyroid lobes to the upper tracheal rings and cricoid. They were really part of the tracheal wall. This probably took place during foetal development, although the thyroid tissue in all probability did not penetrate into the larynx until during extra-uterine life, and at a time when enlargements of the thyroid commonly take place—the period of puberty.

Paltauf's conclusions, that these intralaryngeal and tracheal tumors springing from thyroid tissue do *not* owe their origin to an intra-uterine deposit of thyroid tissue are of great interest. They should therefore *not* be called *accessory* thyroid tumors. They originate in extra-uterine life by penetration of the gland tissue between the cricoid and thyroid cartilages, between the cricoid and first tracheal ring, between the upper tracheal rings, and through the interstitial tracheal membrane itself, *from without*. When this occurs it should be considered as a direct extension of an enlarged thyroid gland—a parenchymatous struma. Proof of this assertion is that the thyroid gland has really grown fast to the cricoid cartilage, interstitial membranes, and upper tracheal rings. This abnormal adhesion can neither be explained by pressure, nor by an inflammatory process, but can only have occurred during the formation and development of the thyroid gland, and only in the way that the *foetal* gland, in these cases, must be united with the perichondrium of the cartilage and the interstitial membranes.

Gruber's³ investigations, perhaps, would give additional support to Paltauf's theory. He found, after examinations of many Russians and Bohemians, that there was often an accessory or really an extra lobule extending downward from the lowest posterior margin of the lateral lobes and lying very close to the trachea. These extra lobules may

also lie in the crico-thyroid space. The only parallel cases that possibly give some support to Bruns' theory are those observed by Streckeisen.²² He found on sections through the hyoid bone seven times the so-called glandulæ intrahyoidea, that is, thyroid tissue was shut in the bone. He believes that this was shut in during the ossification period.

In my own case it could not be determined during the operation whether the left lateral thyroid lobe, which was enlarged, was adherent to the trachea or not. The isthmus was not, because in ligating it the ligatures could be readily passed between it and the trachea. It is a rational deduction, however, from the fact that the intratracheal growth was really part of the tracheal wall, and springing as it did from the posterior and left lateral wall, that in this case, too, the intratracheal tumor was really an offshoot from the left lateral thyroid lobe.

An analysis of the ten cases of this rare condition brings out a number of interesting points: (a) The location of the tumors in all the cases, with the exception of Bruns' last case, was characteristic. They were all situated in the lower part of the larynx and upper part of the trachea and attached to the lateral and posterior walls. Bruns' last case was the only exception to this rule, the growth being attached to the anterior wall. (b) This point decides another interesting fact before mentioned, that these tumors, for this reason, should not be called *accessory* thyroid tumors, because they are really offshoots from the thyroid, as proved in the majority of the cases by the adhesion of the thyroid to the tracheal wall, and in Paltauf's case, microscopically, by the infiltration of the interstitial membranes with the follicles of the thyroid gland. (c) They were all observed early in life, from the fifteenth to the thirty-third year, with the exception of Roth's case (the fortieth year). (d) A goitre of moderate size was present in all except in one of Heise's and in Freer's case. (e) They occurred in both sexes—three in males, and seven (including the author's) in girls and young adult women. A special consideration of my own case reveals some additional interesting points: It is the only case of which I could find any record in which the intratracheal struma was present in a pregnant woman; it is the second case on record in which the struma was confined to the trachea, extending from the first ring downward, the other case being one of Heise's. It is one of the largest, if not *the* largest recorded intratracheal tumor of this kind, extending as it did down the posterior tracheal wall for a distance of over 5 cm., or nearly two inches. The fact that it was present in a pregnant woman suggests another rather interesting connection between the intratracheal struma and pregnancy. I do not think there is much doubt that the pregnant condition had a great influence on the development and increase in size of both the extratracheal and intratracheal struma, because the dyspnoea was not nearly

so great before she became pregnant. It is well known that the thyroid frequently enlarges during pregnancy to cope with the increased metabolic changes of that period. Freund,²⁴ in his classical article on the relations between the thyroid gland and the female genital organs, brings out the fact that the gland is frequently enlarged in pregnant women, and that it almost always increases markedly in size during the birth of the child. This was true in forty-five out of fifty women examined by him. Tait²⁵ found an enlargement of the thyroid body in twenty women who had borne children. In his cases the increase in size developed during the latter months of pregnancy, and subsided very much after delivery. Wölfler²⁶ has shown that in the normal thyroid gland embryonic masses of thyroid cells are found here and there, particularly toward the periphery of the gland, and these, under certain stimuli, take on a kind of normal growth. Such a change occurs during pregnancy.

PROGNOSIS. The prognosis, on the whole, may be said to be favorable. Tumors of this nature, when completely removed, do not appear to have a tendency to recur.

TREATMENT. When the growth is large enough to seriously interfere with respiration a laryngo-tracheotomy for its removal should be performed as soon as possible, on account of the peculiar capacity tumors of this kind have for increasing very suddenly in size. I could not find any record of internal thyroid medication being used for intratracheal struma, but should think that it might be of value in certain cases.

CONCLUSIONS. 1. Cases of intratracheal struma are, perhaps, not so rare as the few cases on record would indicate. If laryngeal and, as far as possible, *tracheal* examinations of all cases of goitre were made, particularly when the thyroid is only *slightly* enlarged and the dyspnoea is severe, more cases would probably be discovered.

2. The term *accessory*, when applied to such tumors, is *not* correct, because they really are "off-shoots" of the thyroid gland.

3. Pregnancy, as in the writer's case, undoubtedly has a great influence on the development of the intratracheal struma.

I wish to take this opportunity to express my thanks to Dr. Harvie for his valuable assistance at the operation, and also to my clinical assistant, Dr. Fairweather, for so carefully following out the after-treatment of the case.

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EXENCEPHALIC (INIENCEPHALIC?) MONSTER WITH BILATERAL HARELIP AND CLEFT PALATE.¹

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THE monster which I wish to present this evening is a seven months' white female foetus which was stillborn, being the first child of an extremely neurasthenic woman of seventeen and a half years, whose family history I cannot obtain, but whose pregnancy up to the time of the miscarriage was uneventful. The father is about twenty-five years of age, healthy and robust, but having a strong family history of tuberculosis, his father having died of phthisis, and his mother is now suffering with the same disease.

About one week before the miscarriage the mother fell directly forward upon her abdomen, followed by severe lumbar pain and uterine

¹ Read at the meeting of the Philadelphia Pathological Society, held February 13, 1902.

hemorrhage, but continued to feel foetal movements up to within two days of the birth. Hydramnios was not present, as has been frequently reported in cases of this malformation, and labor was not long or severe.

Following the classification of Isidore Geoffroy Saint Hilaire, whose work upon teratology has practically been accepted and followed by all writers upon this subject, this monster belongs in the general class of exencephalus, which is described as "characterized by a malformed brain situated at least in part without the cranial cavity, the bony walls of which are themselves imperfect." This general class is further subdivided into (1) the notencephalus, in which the cranial contents are in large part without the skull, resting upon the back of the neck, the tumor protruding through a fissure or aperture in the occipital bone. This deformity is usually associated with hydrocephalus of greater or less degree; (2) proencephalus, in which the same deformity as just described exists upon the anterior portion of the skull; (3) podencephalus, and (4) hyperencephalus, in these the cranial deformity is upon the vertex and the tumor protrudes from above. The podencephalus differing from the hyperencephalus only in degree; (5) iniencephalus, in this subclass the deformity of the skull is the same as in notencephalus, with the addition of a spinal cleft of varying degree; in some cases the failure of union extends as far downward as the coccyx, leaving the spinal canal open throughout its entire length. In this class lordosis and compensatory kyphosis are common—the head is tilted strongly backward, causing the face to look almost directly upward, which, with the faulty development of the vault of the cranium and apparent, rather than real, protrusion of the eyes, has suggested the term "frog-faced" monster, although this term is not exclusively applied to the iniencephalic subclass.

In 1896 Dr. H. F. Lewis presented a specimen of this monstrosity to the Chicago Pathological Society, and at the same time collected all the recorded cases which, with two other specimens, he described at the same time and which had not previously been recorded, brought the total number to twenty-five. I have been able since that time to find the report of only four other cases, although the search through the records has been cursory rather than exhaustive.

No mention could be found in any of the recorded cases of an associated harelip or cleft palate (unless the case described by Dr. Wood could be looked upon as an attempt at harelip formation), with the exception of a single sketch in Ahlfeld's *Atlas* which quite closely resembles the photograph of the present specimen.

The present specimen is an under-developed female foetus; skin and visible mucous membranes normal. The neck is entirely absent, the malformed head being placed directly upon the upper portion of the thorax in such a position as to turn the face directly upward, so that

the plane of the face is at a right angle to the plane of the body, and to cause the deformed or under-developed occipital bone to rest directly back upon the anteriorly curved cervical vertebræ (Figs. 1 and 3).

FIG. 1.



The general appearance of the monster. The match-stick (a) has been passed into the opening in the malformed brain cavity; (b) the remains of the brain, meninges, and fringes of skin rolled up as a ball; (c) area over which there was failure of development of true skin. The constriction about the upper portion of the thorax and the arm was produced by a tight string used to suspend the fetus in the preserving fluid.

FIG. 2.



Photograph of monster, showing the characteristic formation of the face, and in addition the bilateral harelip, with the greatly protruding intermaxillary bone. The two white spots seen just below the centre of the tongue were caused by moisture on the lip at the time the photograph was taken.

The entire upper portion of the skull above an imaginary plane beginning on a level with the superciliary ridges and passing backward

FIG. 3.

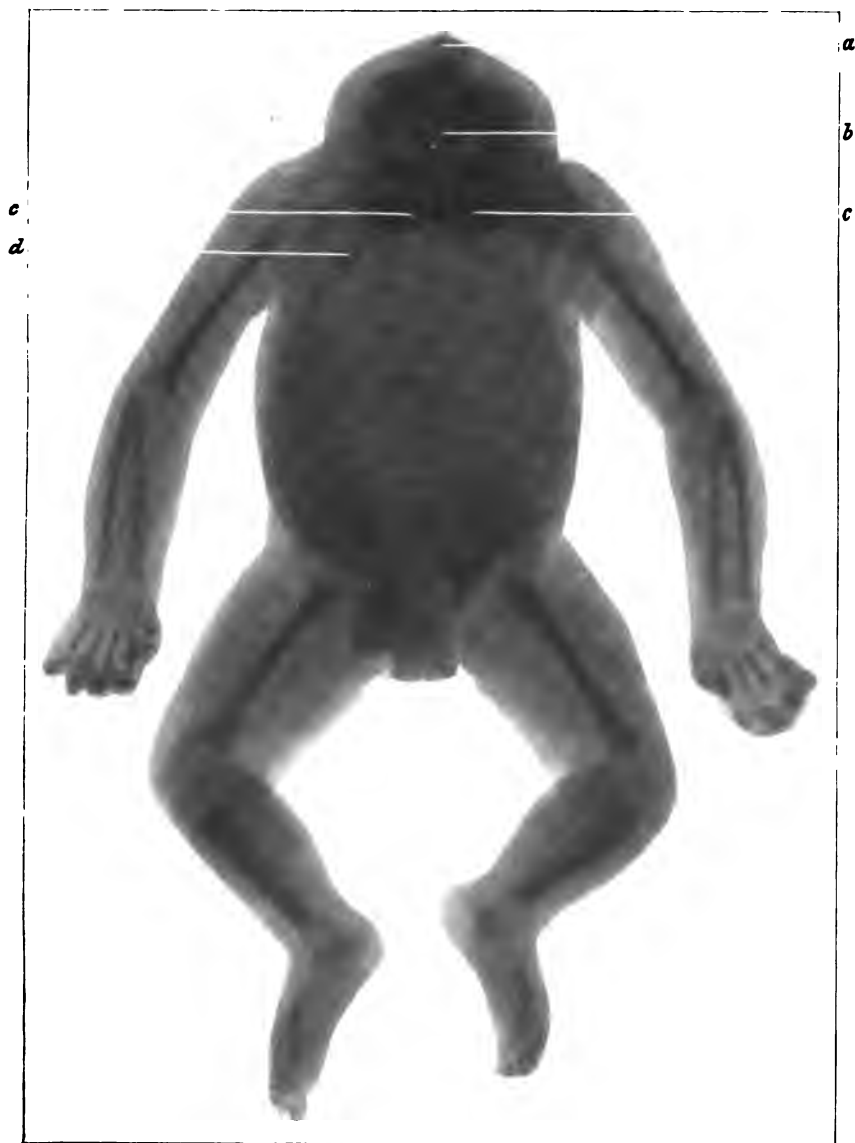


"X-ray" photograph, showing (a) the characteristic cervical lordosis. The remains of the brain and meninges appear as the faint shadows surrounding the optical bones (b).

at a right angle to the plane of the face is entirely absent and replaced by a solid bony plate covered by hairy skin. At the junction of the

occipital bones and spinal column is an opening into the skull large enough to admit the end of one's little finger (in the photograph is shown a match-stick passed into the opening), from the margins of this open-

FIG. 4.



"X-ray" photograph, showing (a) the protruding intermaxillary bone; (b) failure of union of hard palate; (c) abnormal position of malformed and separated optical bones; (d) scapula and scoliosis.

ing are extending the brain membranes and remains of the brain, together with fringes of skin (in the photograph these are seen rolled up as a ball).

Extending over the back from the opening into the skull to the lower lumbar vertebræ and laterally about 2.5 cm. to each side of the spinal column is an area over which the skin has not grown, the tissues being absent down to the muscular structures, through which the ribs and closed spinal column can be plainly seen (Fig. 1). This is not an area of abrasion as at first appears, but one devoid of true skin. The distance from the opening into the skull to the coccyx is 10 cm.

The ears, so far as can be seen, are normally developed, and are behind and slightly below the shoulders. They are 2.5 cm. long and 1 cm. wide. The eyes are normally located and appear normally developed, the palpebral fissure measuring 1.5 cm. and the distance between the inner canthi is 1.5 cm.

The mouth, which is 2 cm. wide, is the seat of a well-marked bilateral harelip, the cleft on each side extending well up into the nostril of the corresponding side, the intermaxillary bone being quite prominent (Fig. 2). The bony cleft extends backward into the mouth and involves both hard and soft palate to a considerable degree (Fig. 4). The distance between the alæ nasi is 1.5 cm. The tongue is quite large and prominent, protruding from the mouth. With the exception of the above-noted abnormalities the fœtus is normal.

Synopsis of Collected Cases.

CASE I.—(Case of Dr. Albert C. Bowerman.) Patient, aged twenty-four years, gave birth, about two years ago, to an apparently normal child, but it died in ten months of some cerebral trouble. Present pregnancy advanced to the eighth month. Hydramnios present. Female fœtus, weighed three pounds, lived about one hour. Body and limbs fairly well developed, though somewhat disproportionate, lower limbs especially long. No neck. Ears well developed and are behind and rather below the shoulder joints; face well formed and directed upward, eyes prominent. Cranium open in occipital region and continuous with the unclosed spinal canal as far down as the lower lumbar vertebræ. Protruding from the upper end of this canal were the congested lobes of the cerebellum much enlarged and exposed across the base of the sacrum. Length of the back from the occiput to the sacrum not over three inches. Child had appearance of crescent with the hollow at the back.

CASE II.—(Case of Dr. B. Onuf.) Duplicity of the face and the anterior part of the head; the duplicity begins proximally from the petrous portion of the temporal bones, including the sphenoid, frontal, nasal, supramaxillary and inframaxillary bones. Two pharynges are present which communicate with each other. Craniorrhachischisis involves the entire vertebral column.

There is the interposition of a peculiar os sacrum-like vertebral

formation, and two longitudinal bones (each divided into anterior and posterior portion) between the occipital and temporal bones of each side. In the proximo-distal direction these intercalary formations form the transition between the bones of the skull and the vertebra. There is also absence of the bony sacrum.

CASE III.—(Case of Dr. W. Atkinson Wood.) Mother, aged twenty-three years; father, aged twenty-six years. Female fetus born dead, but the tissues appeared healthy and fresh. Entire scalp from the superciliary ridge to the position of the occipital protuberance and from one mastoid region to the other was a mass of dry gangrene studded with pear-shaped fibrous outgrowths. Both eyes protruded markedly, and the mouth in the mid-line was quite absent, being represented by two lateral slits into which a probe passed easily for half an inch. Both ears were well developed. At the third or fourth lumbar vertebra was a myelocoele. The lower extremities were fully flexed at the hips, so that the feet which were in the position of talipes spasticus, or dorsalis, were fitted and pressed accurately under the position of the chin.

The vertex was removed on a level with the superciliary ridge, and the bones were so fused together as to appear as one bony mass. The petrous portion of the temporal bone and mastoid bones were fused into one mass; on the inner side of this mass were two cavities containing brain matter, each about the size of a bean. In the substance of these bony masses the ossicles were well developed. The sphenoidal cells were well developed, and the nasal chambers connected directly with the anterior fossa of the skull.

The vertebral column had no direct communication with the skull, being kept in apposition only by skin and connective tissue. The spinal canal opened directly at the depression on the posterior part of the skull, the spinal cord being thus exposed to the air.

The pharynx and larynx were well developed. Tongue and tonsils were contained in a membranous sac on the anterior surface of the spinal vertebrae. The internal carotid was doubtfully developed (which the author ascribed as the probable cause of the cranial deformity).

The lower and upper jaws were ankylosed together; the buccal cavity was divided into two lateral halves. The alveolar processes of the upper jaw were fused with the hard palate, leaving a slit-like opening between.

CASE IV.—(Case of Dr. Barton C. Hirst.) The doctor presented a photograph of the monster, with no description.

The specimen shown this evening will be presented to the Wistar Institute of the University of Pennsylvania.

I am indebted to Dr. Clifford B. Farr for the translations of the foreign references, and to Dr. Thomas S. Stewart, who furnished the "X-ray" negatives.

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A CASE OF HYPERNEPHROMA OF THE KIDNEY COMPLICATING PREGNANCY.

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AND

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THE following case of tumor of the kidney appears to deserve mention both because of the rarity of the tumor and the peculiar manner in which it manifested itself.

Mrs. T., a negress, aged thirty years, was admitted as an emergency case to the Medico-Chirurgical Maternity Hospital, on November 3, 1900. Examination of the patient on admission showed her to be about six months pregnant. She complained of pain over the uterus, and an internal examination showed evidence of a threatened miscarriage. More interesting than the pregnancy was the presence of a tumor which could be felt at the lower border of the liver. Dulness over this tumor extended into the right flank.

We were unable to elicit any previous history throwing light upon its origin. The patient was anæmic, but had not the characteristic cachexia of malignancy. The urinalysis was negative, the temperature elevated. Five days after admission she spontaneously miscarried, giving birth to a six months' fœtus. After delivery the tumor stood out more prominently and appeared to be rapidly increasing in size.

A tentative diagnosis of either abscess of the liver or a large growth of the kidney was made. The tumor had not the feel of a cystic kidney, nor were the clinical symptoms suggestive of liver abscess. She still continued to have fever and was rapidly growing worse, and on November 17th was etherized and an exploratory incision made. The tumor occupied more of the region of the liver, and upon deep palpation fluctuation was noticed.

An incision was made over the most prominent portion of the growth through to the peritoneal cavity. What was then found was supposed to be a retroperitoneal abscess, so gauze was packed around the wound to shut off the general peritoneal cavity, and an incision was made into this bulging mass. A large quantity of thin, dark, very offensive fluid escaped. It had the odor of urine. As the patient was now in an extremely critical condition, a large drainage-tube was inserted. The abscess was drained, and the next day the patient seemed a trifle improved, but she commenced to lose ground again, and died December 17th, a month and a half after her admission to the hospital.

Autopsy Notes. December 19, 1900. Body of a much emaciated young colored woman of middle stature, about thirty years of age. The face bears an expression of suffering. Rigor mortis is absent. Inspection of the body shows an open wound in the right hypochondrium, large enough to admit a finger, and communicating with some part of the intestine, forming a fecal fistula. The edges of the wound are indurated, the surface necrotic and foul. In addition to this intestinal fistula, there is a sinus which descends deeply toward the right

side of the abdomen and communicates with what appears to be an abscess cavity, as puriform matter bathes the exploring finger. When closely examined, this matter was curdy and much resembled the pus of a cold abscess. The body was opened by suitable incisions.

Thorax. Pericardium normal; pleural adhesions on both sides. Heart: brown atrophy. No valvular lesions. Aorta normal, except for beginning atheroma. Lungs: slight congestive oedema.

Abdomen. Chronic peritonitis chiefly localized about the area of disease on the right side. Recent peritonitis near the external wound. Spleen, small; capsule thickened, substance indurated, vessels large. Adrenals apparently normal. Left kidney normal in size and appearance. On section, mild parenchymatous changes of the cortex. Ureter normal. Right kidney (see below). Ureter normal. Bladder normal. Uterus: enlarged puerperal uterus. No sign of any pathological condition. Ovaries normal. Pancreas normal, except for inflammatory adhesions to the surrounding viscera. Stomach and duodenum normal. Small intestine normal. Large intestine, operation fistula communicating with the transverse colon. The transverse and ascending colon are adherent to surrounding structures by inflammatory adhesions. Vermiform appendix normal and free of adhesions. Liver normal. Gall-bladder normal. Nervous system not examined.

The only lesion of interest or importance is to be found in the *right kidney* and its contiguous structures.

The sinus descending from the external wound was found to communicate with a cavity above and internal to the right kidney. The appearances suggested that this cavity had once been large, but had collapsed where the external wound was made. It seemed to arise either from the kidney or the retroperitoneal tissues. The kidney together with the surrounding peritoneal tissue, forming the entire walls of the somewhat indefinite sac of the abscess (?), were removed and carefully dissected. The ureter and pelvis of the kidney were now found to be normal. Little by little the kidney was dissected free from the surrounding cellular tissue, which was thickened and infiltrated until gradually the lower three-fourths were exposed, after which it was split longitudinally along the convex surface. The substance of the organ was now found to be free from disease—a condition which was entirely compatible with the fact that no abnormal condition of the urine was discovered during life.

Tracing down the sinus from above and laying it open, it was discovered that the inner surface of its wrinkled and contracted wall was thickly studded with more or less rounded nodules, suggesting some neoplasm. These became larger as the upper part of the kidney was approached. In the superior, external, and posterior portion of the kidney there was a rounded excavation about as large as a walnut. In this excavation lay a neoplasm nearly as large as the excavation itself, and projecting so as to form a rounded mass from the bottom of the excavation into the sinus. The color of this neoplasm, as well as the color of the nodules in the neighborhood, was grayish-yellow, the surface smooth, the consistency rather firm and somewhat friable.

A section passing through the neoplasm and the adjacent kidney substance showed the tumor to be sharply demarcated from the kidney substance, being separated by a band of fibrous tissue.

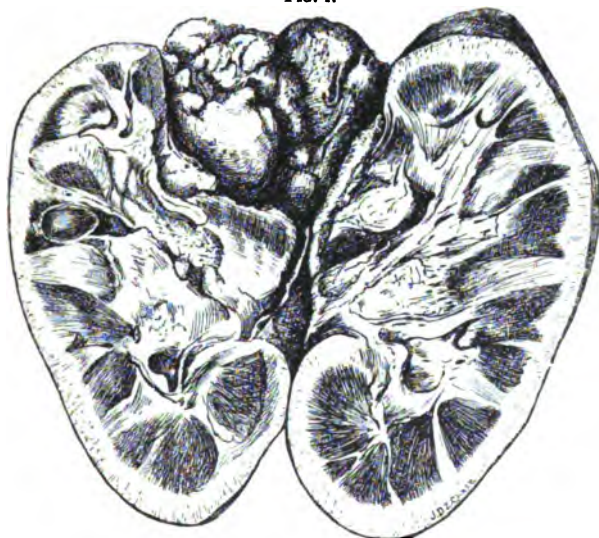
The presence of the neoplasm in the kidney, the numerous nodules

in the sinus, and the absence of evidences of any inflammatory process, beyond that depending upon the operation fistula, entirely changed the aspect of the case.

It now appeared as if the condition was one of primary neoplasm of the kidney with enlargement and softening. The puriform matter that escaped when the operation was performed, and continued to escape subsequently, was the softened contents of the neoplasm enclosed in its capsule of perirenal tissue. The escape of this softened tissue was followed by collapse of the sac and the necrosis of the remaining part of the growth with the formation of the rounded eminences found in the walls of the sinus.

Concerning the nature of the growth the naked-eye appearances are not clear. It undoubtedly arises from the kidney, yet its growth away

FIG. 1.



Hypernephroma of the kidney. Organ split longitudinally, and showing the tumor in the upper left-hand half.

from the kidney substance and its sharp demarcation from it are peculiar. The growth shows no disposition to infiltrate neighboring organs or tissues, and has given no metastases. These facts seem to warrant the assumption that the growth is a *hypernephroma*—i. e., a growth developing from a fragment of the suprarenal body included within the kidney tissue during the embryonal differentiation of the tissues.

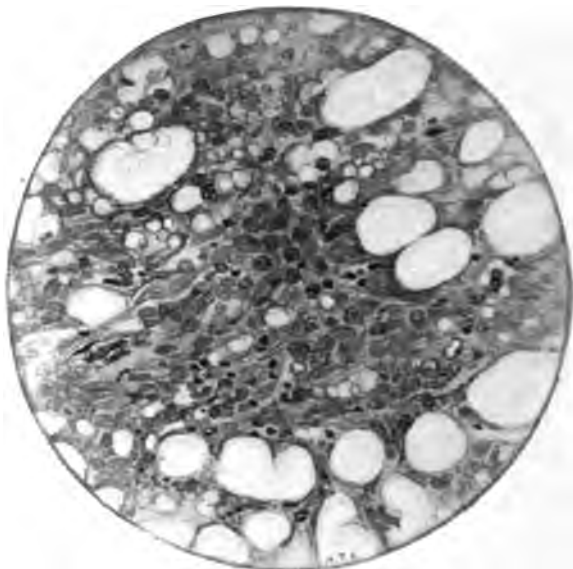
Microscopy. The fragments of tissue were hardened in formaldehyde, embedded in paraffin, and stained for study by various methods.

Three difficulties immediately presented themselves and made the determination of the nature of the growth difficult. First, the entire growth was in a condition of advanced retrogressive change; second, it was evidently distinctly modified by the acute and chronic inflammatory processes that had taken place in and about it; and, lastly, it was a peculiar, atypical growth.

Beginning the examination with that part of the growth which is attached to the kidney and occupies the cavity in its upper portion, we find, though the largest tumor mass is firmly attached to the kidney, it does not blend with its substance anywhere, but that between the tumor tissue and the kidney tissues there is a fairly sharp line of circumscription, consisting in part of a fibrous tissue belonging to the tumor and in part of fibrous tissue resulting from atrophy of the parenchymatous elements of the organ.

The kidney structure is quite normal until the described line of demarcation is reached, when a sudden and complete pressure atrophy is observed. From the study of sections of this kind it becomes evident that the growth has not taken place from the kidney tissues.

FIG. 2.



Hypernephroma of the kidney, showing the indefinite structure and the presence of considerable fat. This section passed through the centre of the main tumor nodule. (Zeiss, Ocular II., Objective D D.)

The tissue forming the tumor is very largely in an advanced condition of *fatty infiltration*. This is true not only of that part in contact with the kidney, but also of the remote parts. So wide-spread is this infiltration that it becomes impossible to decide upon the nature of the cells, nearly every one of which is involved in the process. Among the fatty globules thus formed and thickly distributed in the trabeculae of fibrocellular structure, and about large bloodvessels which are present, there are cells of various kinds, most of which are unfortunately altered by degenerative changes:

1. *The proper cells of the tumor.* These cells are rather large, for the most part irregularly ovoid, and mononucleated. They are closely packed together, without any arrangement, the cytoplasm is universally injured by retrogressive changes and prone to contain vacuoles and

granules. The nuclei were originally round, large, and vesicular, but almost every variation from the normal presents itself. There are fragmented nuclei, vacuolated nuclei, and giant pale nuclei. Occasionally a large cell with several nuclei is observed. In general the cells bear more distinct resemblance to sarcoma cells than those of any other type, yet the extensive fatty metamorphosis and the original scanty amount of chromatin are distinctly contrary to this view.

2. *Connective-tissue cells.* Spindle cells, derived from the stroma of the tumor and from the bloodvessels are very numerous. Indeed, certain areas consist almost exclusively of them, their large size, arrangement in bundles, etc., forming an embryonal tissue closely suggesting spindle-cell sarcoma. These areas are, however, not numerous, and proximity to the trabeculæ, etc., from which the cells are derived, usually sufficiently explains them. Spindle cells, endothelial cells, and irregular cells evidently derived from the connective tissue and bloodvessels are scattered irregularly throughout the growth and often mingle with its proper cells.

3. *Plasma cells.* There seem to be a good many of these scattered about among the tumor cells, but their numbers are greatest in the areas in which the connective-tissue cells predominate.

4. *Leucocytes.* Leucocytes occur in small numbers throughout the tumor. They are most numerous in the connective-tissue areas surrounding the bloodvessels.

There is no regular framework of connective tissue supporting the cells of the tumor.

The bloodvessels are numerous, large vascular trunks more numerous in proportion than the capillaries.

Degeneration. In addition to the wide-spread fatty infiltration of all parts of the growth there were numerous areas of necrosis in which no definite structure was any longer visible. Here and there, and especially in the connective-tissue areas, pigmentation of leucocytes and connective-tissue cells with hemosiderin was observed, but nowhere in the growth were hemorrhages found.

When other nodules of the neoplasm, from the wall of the sinus and from the perirenal tissue, were examined the advanced fatty infiltration of the cells was the most marked feature. In nearly all of these fragments the retrogressive changes were so marked that the cellular structure could not be studied.

It is evident, therefore, that the histological structure of this tumor conforms to no regular tumor type. It is a growth attached to the right kidney and partly embedded in a fossa in the organ, yet developing from some other structure and not invading the kidney. Its peculiar cells are subject to fatty infiltration. The tendency of the growth is to increase in size without metastatic distribution and without infiltrating contiguous structures. All of these facts seem to point to its development from abnormal tissue. It did not grow from the right adrenal itself, however, as that organ was found normally placed and reasonably normal. Therefore if of adrenal tissue it must have been from a "rest" or fragment misplaced and in apposition with the kidney. The preservation adopted did not permit us to determine the presence or absence of glycogen, but the facts already given, and especially the marked fatty infiltration, lead us to believe that the tumor was correctly called *hypernephroma*.

OBSERVATIONS UPON RECENT METHODS OF TREATING CORNEAL ULCERS, WITH ESPECIAL REFERENCE TO THE USE OF CARBOLIC ACID AS A NOT INFREQUENT SUBSTITUTE FOR THE ACTUAL CAUTERY.

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THE general and more definite recognition of the important rôle which micro-organisms play in the etiology of corneal ulcers has led, as might be supposed, to decided changes in the therapeutic measures employed in the treatment of this often troublesome condition, and, it may be added, to modified views as to the beneficial action of some of the older and well-tried remedies.

In the text-books upon eye diseases of twenty years ago we find but scant reference to bacteria as factors in the causation of conjunctival or corneal inflammations, and practically nothing as to the employment of bactericidal agents, as such, in their treatment. Then, as now, nitrate of silver was used in purulent conjunctivitis, and, at Gayet's and Fuchs' suggestion, the actual cautery was beginning to be employed in the more dangerous types of corneal ulcers; but the good effects of the former were ascribed to its "astringent and caustic" action, and of the latter, by one authority, at least, to its supposed influence in reducing intra-ocular tension.¹

At the present day, while it is conceded that the efficaciousness of nitrate of silver is in part due to its direct action upon the conjunctival inflammation, it is in larger measure attributed to its bactericidal properties, and especially to its power of penetration, which enables it to reach and destroy the gonococci that have found lodgement beneath the superficial layers of the conjunctival epithelium, as well as those that are free in the conjunctival sac. And, in like manner, as to the actual cautery in the treatment of corneal ulcers, its good effects are now ascribed mainly to its powerful antiseptic action, as was first pointed out by Sattler.²

At the period referred to, if, in spite of atropia, warm fomentations and a pressure bandage, a corneal ulcer continued to progress and threatened the integrity of the eye, reliance was placed chiefly in paracentesis of the anterior chamber, in incision through the base of the ulcer (Saemisch), or in the performance of an iridectomy. Now, when

¹ Diseases of the Eye, Soelberg Wells, 1883, p. 233.

² Transactions of the Fifteenth Annual Meeting of the Heidelberg Ophthalmological Society, 1883, p. 104.

a threatening ulcer of the cornea is encountered, one asks what exceptionally virulent micro-organism is causing the unfavorable condition, and by what means it can be eradicated and the ulcer rendered aseptic, or, on the other hand, if the micro-organism be not of exceptional virulence, what has lowered the resisting power of the corneal cells, or of the phagocytes whose function it is to assist in their defence, and how it can be brought up to its normal standard. So, while such drugs as atropine, eserine, and the more recently introduced holocaine are still employed in the treatment of ulcerative processes of the cornea, and are held to be of undoubted value, germicidal agents, such as the actual cautery, carbolic acid, tincture of iodine, formalin, chlorine water, iodoform, and boric acid, and the mechanical removal of the bacteria and the corneal tissue which they have invaded by curetting, are regarded as of the first importance.

And this brings me to the matter which chiefly induced the writing of this paper, namely, to speak of the good results which, in my own hands and in the hands of several of my colleagues, have been obtained from the use of carbolic acid in the treatment of threatening ulcers of the cornea—the application of the pure acid to the ulcer for its germicidal effect and, in large measure, as a substitute for the actual cautery. It is, perhaps, hardly necessary for me to say that this use of carbolic acid is not novel, and that I am fully aware that others have employed it in the same manner and with favorable results.

I must confess that I have not been one of those who, upon small provocation, are disposed to use the actual cautery for the control of corneal ulcers, and that my inclination has been to employ, instead, some more easily managed and less dangerous procedure; and this has been not merely because I am aware that the application of the actual cautery to the cornea has been known to cause permanent opacity of the lens, and must almost invariably leave an indelible leucoma of the cornea, nor because I have had occasion to observe how easily it may lead to an unintentional opening of the anterior chamber, but, rather, because I feel that whenever it is possible for the surgeon to substitute a simpler and safer surgical procedure for one that is less simple and less safe it is his duty to do so, provided, of course, it is equally efficacious.

That the application of carbolic acid to a corneal ulcer is a simpler and safer procedure than its cauterization by the galvano- or thermocautery goes without saying. But what of its efficacy? I am not prepared to assert that it will accomplish in every case what the cautery will do, or that it should wholly supplant the latter; but I believe that in many cases in which it is usual to employ the cautery carbolic acid may be substituted with advantage, and that in most cases

it is judicious, at least, to make trial of it before resorting to thermal cauterization.

In applying pure carbolic acid to the cornea it is, of course, important to limit its action carefully to the affected part. To facilitate this the eye should be anæsthetized by cocaine, which renders the procedure entirely painless. I have found it convenient to make the application by means of a pointed toothpick, about the tip of which a very small quantity of absorbent cotton has been wound. If much cotton is used an excess of the acid will be taken up, and it will be almost impossible to prevent its spreading over healthy portions of the cornea. To the surface of the ulcer the acid should be thoroughly applied by a gentle rubbing movement, which is, in effect, a sort of curettage. When the ulcer is foul and its walls are lined by infected and necrotic material, this should be removed with a small curette before the acid is applied. When, however, this condition is less pronounced, aided by the loosening action of the cocaine, the cleaning of the ulcer may be effected satisfactorily by means of the toothpick, armed with a wisp of dry cotton. After the acid has been allowed to remain in contact with the ulcer for a few moments, the lids meantime having been held apart, its further action should be arrested by flushing the cornea with sterile water, normal salt solution, or a saturated solution of boric acid. After the effect of the cocaine has passed off some smarting or discomfort may be felt in the eye, but usually this is not pronounced.

The effect of carbolic acid upon the surface of the cornea is rather startling, for it attacks the epithelium energetically, and in an instant renders it opaque, causing it to assume a milky appearance. Bowman's membrane, it would seem, is much more capable of resisting its caustic action, for the superficial opacity which it produces quickly disappears.

It seems hardly necessary, and yet it is, perhaps, best, to say that the employment of carbolic acid in the manner described is not to be regarded as part of the routine treatment of corneal ulcers; on the contrary, the cases in which it is called for are distinctly the exception and not the rule. When the ulcer is the product of infection with one of the less virulent bacteria, such as the staphylococcus aureus—as is commonly the case in phlyctenular keratitis—the yellow oxide of mercury, supplemented, perhaps, by boric acid, commonly meets the condition most satisfactorily. It is the dangerous ulcers—the serpiginous ulcer, the ulcer complicated by hypopyon—which are commonly due to the presence of the pneumococcus and less frequently to the more virulent streptococcus, that call for the energetic germicidal action of carbolic acid and in which its good effects are manifested.

I shall not prolong this paper with detailed accounts of cases in which

the efficacy of carbolic acid has been shown ; but, in justification of the views I have expressed as to its value, will simply mention that I have employed it with gratifying results in hypopyon ulcers (in the early stages, especially), in suppurating ulcers of traumatic origin following oyster-shell injuries, etc., and in dendritic keratitis, and that recently one of my colleagues, Dr. James Bordley, applied it to a threatening corneal ulcer complicating gonorrhœal conjunctivitis, and which, it seems probable, was due to a secondary infection, with the result that the rapid progress of the ulcer was at once arrested and the process of repair quickly established.

In conclusion, and to prevent possible misapprehension, I wish to add that while in what has gone before I have dwelt upon the importance of using energetic antiseptic agents in the treatment of the more dangerous types of corneal ulcers, I am far from holding that they should be our sole reliance, that they should be employed to the exclusion of other remedies. Atropine, holocaine and, exceptionally, eserine and opium, as a lotion to be applied over the closed lids, are as useful to-day in controlling suppurative and ulcerative processes of the cornea as they ever were, and the same may be said of the liberal administration of quinine, which, unquestionably, augments the resisting power of the cornea, and so may determine, as we would wish it, the outcome of the combat between the invading bacteria and the phagocytes which hasten, literally as well as metaphorically, to their circumvention.

PRIMARY SYPHILITIC OPTIC NEURITIS.*

By C. A. VEASEY, A.M., M.D.,
OF PHILADELPHIA.

PRIMARY syphilitic optic neuritis is an affection so rare that its existence is even denied by some authors. Thus Hock¹ refers to syphilitic neuritis only as a symptom of cerebral syphilis. Hughlings Jackson² does not believe that primary syphilitic optic neuritis exists, but that neuritis manifests itself always as a result of cerebral gumma.

Undoubtedly, syphilitic inflammation of the optic nerve manifests itself most frequently in the form of papillitis resulting from some form of cerebral syphilis ; but certainly in a number of rare instances the nerve itself seems to be primarily affected by the syphilitic poison.

* Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, April 15, 1902

Leber² makes a distinction between the papillitis arising in consequence of a cerebral gumma and the true syphilitic optic neuritis appearing primarily in the trunk of the optic nerve. Foerster⁴ and Alexander⁵ also believe in the existence of a primary syphilitic neuritis, the latter stating that he has studied two such cases. Horner⁶ and Knorre⁷ have found upon pathological examination of such cases that there is sometimes marked inflammatory thickening in both optic nerves from the optic foramen to the chiasm, and even beyond. Ole Bull⁸ has observed hyperæmia and opacity of the optic nerve and surrounding retina in the early stages of syphilis, but believes that marked papillitis is to be found in the later stages of the disease. Januskiewicz⁹ states that he has seen a case of primary specific optic neuritis two and a half months after infection. Horstmann,¹⁰ in a most excellent paper upon the subject, from which the writer has quoted quite freely, believes in the existence of a primary syphilitic optic neuritis, and reports eight cases of this condition. In his cases all the patients had suffered from syphilis and had undergone specific treatment for a shorter or longer period. The ophthalmoscopic examination revealed in each some swelling of the optic disk, which encroached but little upon the surrounding retina. In some of the cases the papillitis was monolateral, and in those cases in which both nerves were affected one side was usually much worse than the other. There was hyperæmia, together, with more or less opacity, as is ordinarily found in a moderate degree of papillitis. In all the cases there was found concentric contraction of the visual fields, but in none was a central defect observed. The author concludes, therefore, that the condition could not have been due to descending neuritis, and, moreover, there was no evidence of any existing orbital or cerebral disease. In addition, in all of the cases the disease was either removed or improved by the administration of anti-syphilitic remedies.

In this same class the writer is inclined to believe the two following cases belong :

Mr. —, aged forty-five years, was referred to me for an examination of his eyes on November 5, 1901, complaining of an aching pain in and immediately behind the eyeballs, and an inability to see well, especially with the left eye. Vision had been growing less acute for two weeks, and for a few days prior to the examination of his eyes it had become rapidly diminished in the left. Vision of the right eye equalled 6/9; of the left 1/100. There was a history of occasional attacks of headache, always preceded, as the patient expressed himself, by "shooting stars," that it is believed were migrainous in character, and which had existed for a long time, and still exist.

The pupillary reactions were normal. An ophthalmoscopic examination showed that both media were clear; there was a moderate

papillitis with very little involvement of the surrounding retina, and with but little exudate showing on the surfaces of the disks. There were no hemorrhages in either eye, and in the left the papillitis was much more marked than in the right, the latter disk presenting only slight veiling of the margin.

The patient acknowledged the initial lesion of syphilis three months before the eye examination was made, and this statement was corroborated by the physician who had him under treatment at this time. There were present mucous patches and a moderate eruption of the skin, and he had been taking mercury irregularly.

The field of vision for the right eye showed concentric contraction for both form and colors, but no scotoma could be detected. The vision of the left eye was so reduced that the patient could not distinguish large test objects, but with a candle flame the limit was also found to be concentrically contracted.

The patient was immediately placed upon mercurial inunctions and rapidly increasing doses of potassium iodide, and in one week showed a moderate degree of improvement. In two months' time the neuritis had entirely subsided, and the vision in the right eye equalled $\frac{6}{5}$, and in the left $\frac{6}{7.5}$, the fields being normal. Thirteen days after the examination was made of this patient he brought his wife in to see me, who presented the following history:

Mrs. —, aged forty-three years, had been complaining for a few weeks of inability to see well, together with some pain in and behind the eyeballs. She had always worn glasses of her own selection, obtained from an optician. The vision of the right eye equalled $\frac{6}{7.5}$ —, and of the left $\frac{6}{7.5}$ —. Both pupils reacted promptly to light, convergence, and in accommodation.

The ophthalmoscopic examination revealed in the right eye an oval disk with its edges very slightly veiled, but no other changes. In the left eye there was distinct papillitis, the nasal edge being most affected, with but little involvement of the surrounding retina. No hemorrhages were found.

The interesting history was ascertained that the patient was also syphilitic, presenting mucous patches and a skin eruption, and that the initial lesion was acquired from her husband, whose primary lesion had been acquired only three and a half months before. During this period, like her husband, she had been using mercury irregularly. The field of vision for the left eye was concentrically contracted in a moderate degree; the field of the right eye was not affected. She was at once placed upon mercurial inunctions and rapidly increasing doses of potassium iodide, and in six weeks' time the papillitis had almost entirely subsided, and the patient's vision equalled $\frac{6}{5}$ — in each eye, with normal visual fields.

It seems to the writer that these two cases of papillitis, occurring so early after the initial lesion of syphilis, and the circumstances attending the same, must be placed in the class in which the optic nerve is primarily affected. There was no history of any cerebral complication, and it hardly seems probable there could have been any such condition without corresponding symptoms. In addition, the early appear-

ance of the papillitis, within two and a half months after the appearance of the initial lesion in patients, would seem to exclude cerebral disease, as pointed out by Horstmann. The concentric contraction of the visual field would seem to argue against the extension of the inflammatory process from the sheath to the substance of the optic nerve. It is certainly a most unusual and interesting fact that the husband acquired the initial lesion of syphilis, inoculated his wife, and that both presented papillitis within three months from the beginning of the disease, the left nerve being principally affected in each case.

The manner of the production of primary syphilitic papillitis is not definitely determined, but may possibly be brought about by syphilitic disease of the central vessels. The very fortunate result obtained in the above two cases is believed to have been brought about because they came under observation very early after the beginning of the disease, as it has been shown that when primary syphilitic neuritis occurs in a patient whose infection dates back more than a year the prospect of complete recovery is small.

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REVIEWS.

THE PRACTICE OF OBSTETRICS, BY AMERICAN AUTHORS. Edited by CHARLES JEWETT, M.D., Professor of Obstetrics and Gynecology in the Long Island College Hospital, New York. Second edition, revised and enlarged. 8vo., pp. 776. New York and Philadelphia: Lea Brothers & Co., 1901.

THIS composite work appears in its second edition, with changes in its contributors and with increased and revised contents.

The character of the articles and their assignment are scarcely consistent with the title of the volume. In a book whose title would indicate that it is devoted entirely to a description of the practice of obstetrics the first 100 pages are given to anatomy, physiology, and embryology. In contrast to this the section upon Obstetric Surgery is allotted but 87 pages. In the distribution of articles the immediate repair of lacerations of the generative tract, the induction of abortion and premature labor, the delivery of the retained and adherent placenta, Cæsarean section, the Porro operation, symphysiotomy, and the subject of ectopic gestation are assigned to gentlemen whose titles and practice inform us that they are interested in gynecology only. In view of the fact that there are in the United States a number of competent obstetric surgeons who have had abundant experience in these subjects, and whose records have been published, it is remarkable that none of them was selected to write upon these topics. The inconsistency displayed in the arrangement and management of topics and the title of the volume call attention to this as a composite book, a type of publication largely created for commercial interest. In arranging for such a volume, publishers select as large a number of writers as possible connected with teaching institutions, thus insuring a sale for the book. Scientific consistency and a uniform standard of excellence may be sacrificed by this arrangement. While composite volumes contain many articles of permanent value, they must be encyclopædic in scope, or are, we think, inferior to a consistent treatise by one writer, experienced in all topics of the subject upon which he writes.

Medical education has now advanced to the point where anatomy, physiology, and embryology are subjects for study in the early years of a medical course. They receive their best consideration in separate text-books, and the best literature of modern times will not find it necessary to begin a volume with an anatomical or physiological or embryological epitome which has little scientific value. A purely practical consideration of obstetrics would certainly have given a greater space to obstetric surgery than to anatomy, physiology, and embryology, and the practitioner depending for guidance upon such a volume desires the experience of one accomplished in Cæsarean section and the other operations of obstetric surgery.

While we thus take issue with some important features of the book, and believe it more a trade book than a scientific volume, we have no intention of slighting the work of its accomplished editor and many articles contained in the volume. While we may differ with some of the methods of treatment advised, and while our methods of study may be slightly different in some respects, we have nothing but admiration for the papers of Dr. Edgar, Dr. Williams, and the contributions to the volume from its editor, from Dr. Dickinson, and others associated with him. The diagnosis of pregnancy has received no better exposition than that given by Dr. Dickinson, enriched by excellent illustrations. For the general practitioner, the keystone of the obstetric arch is the management of normal labor, and this receives full and adequate exposition at the hands of Dr. Jewett. Dr. Robb indulges in reminiscences of his medical career before he became a specialist in gynecology by writing upon the puerperal state and its management, while the care of the newborn child is assigned to Dr. Bartley, who gives an excellent résumé of artificial infant feeding. We recognize the sound surgical judgment of an old friend in Dr. Henrotin's article upon ectopic gestation, while Dr. Cameron and Dr. Webster contribute an interesting section in that portion of the volume devoted to the pathology of labor. Among the best expositions of eclampsia, its causation and treatment, which we have seen is Dr. Edgar's article, in which he describes with abundant illustrations his method of rapidly dilating the cervix to secure prompt delivery.

Dr. Williams' paper upon puerperal infection is an authoritative monograph upon the subject to which practitioners and teachers as well may refer with profit. In discussing the treatment of puerperal septic infection attention is properly called to the value of strychnine and alcohol. The uselessness of sublimate injections to secure deep penetration of the tissues is clearly shown, and the danger of intra-uterine douches of antiseptics, and especially of bichloride of mercury, receives ample consideration. It is urged that bacteriological examination of the lochia and of the interior of the uterus be made whenever possible. If streptococci are found, then local treatment of the uterus is of little value, while if the germs of putrid infection are present, thorough douching with sterile salt solution is indicated. The uterus must be made to contract, if necessary, by the use of ergot. Antipyretic drugs should not be given, but excessive fever should be treated by the use of cold. As regards surgical interference, pus-tubes not adherent may be removed through the abdominal wall, while if extensive adhesions are present they should be punctured and evacuated through the vagina, with subsequent packing of the abscess cavity with gauze. Hysterectomy for puerperal sepsis has a very restricted field.

In the section upon Obstetric Surgery the repair of recent lacerations of the vagina and vulva are illustrated with some very good drawings. The immediate repair of the lacerated cervix is considered permissible and as affording a fair chance for success. In the induction of abortion and premature labor several obsolete methods are given, and also those which have received the sanction of wide experience. In cases where the uterus must be promptly emptied in early pregnancy dilatation and curetting are very properly advised as the method to be preferred. In cases of adherent placenta the advice is given to try faithfully Crede's method first, and then to remove the placenta by the

hand, douching the uterus with normal salt solution afterward. In the use of forceps the indications and dangers are clearly stated, and the operation of forceps delivery is recognized as attended with difficulty and danger. Although the advice is given to sterilize the forceps by boiling, there is no mention made of a portable and convenient sterilizer in which this may be done. A basin of bichloride solution and one or two squares of cheese-cloth comprise the instrumental armamentarium for antisepsis. The use of sterile glycerin or vaseline is advised as a lubricant for hands and instruments. We have in creolin and lysol lubricant antiseptics which, in our experience, should be employed for boiling the forceps and as lubricants in all manipulations. It is possible that the illustrations do not clearly represent the practice of the writer, for the forceps blades are shown guarded but by the tips of the fingers and pushed in by the thumb and other hand. The writer prefers to remove the forceps when the head is on the pelvic floor so low down that it can be expelled by light pressure through the perineum. We are glad to know that the introduction of the finger into the rectum is thought inconsistent with aseptic precautions. Various devices are shown for making axis traction with the hands and with several forms of instruments. In the treatment of occipitoposterior rotation attention is called to a manipulation often neglected, the rotation of the entire body of the fetus anteriorly. Under full anæsthesia this is often successful and is accompanied by anterior rotation of the occiput. It is so safe and valuable a procedure that it should not be neglected.

In treating of Cæsarean section the writer takes what we believe to be the proper stand, that the limit of the operation has been advanced through perfection in technique. We cannot agree that the better method consists in keeping the uterus within the abdomen, for the disadvantages claimed by turning out the womb are more than compensated for by the better control of hemorrhage and the removal of all uterine fluids from the abdominal cavity. The uterus is emptied much more rapidly and easily when turned out of the abdomen than if it be retained. The rubber ligature is seldom necessary if an intelligent assistant be available. In closing the womb the majority of experienced operators prefer silk, and as the uterus must contract in involution, a permanent suture is especially desirable. The manner of dressing a patient who has had a Cæsarean section is of considerable importance. The writer refers to a suitable bandage, by which we infer that the many-tailed flannel bandage commonly used after abdominal section is advised. In our experience in difficult cases, where a patient is very restless after operation, or where the patient coughs or vomits, this bandage is inefficient. We have seen the best results by covering the abdomen completely with strips of adhesive plaster encircling two-thirds of the body. In this manner an absolutely firm and unyielding support is given to the abdominal wall during coughing or straining. It seems to us that the writer gives, in a book addressed to general practitioners and intended to describe the practice of obstetrics by physicians, dangerous advice as regards the choice of time for Cæsarean section. It is said that it is not necessary to wait until labor pains come on nor for marked dilatation of the cervix. The surgeon may sometimes be compelled to operate during labor. Experience has shown that pelvimetry, palpation, and the estimation of the size of the fetus are not in all cases reliable guides for the performance of the Cæsarean

operation. The test of labor must be awaited in many of these cases. The majority of obstetricians, we think, agree in stating that the patient should come into labor and should have sufficient uterine contractions to determine by actual test whether the head can or cannot enter the pelvic brim. If it does not under favorable conditions and the patient under close observation, then operation must be immediately performed. There are some notable instances where surgeons have announced Cæsarean operations for a given time, and summoned an audience, to have the patient disappoint them by spontaneous delivery. The Cæsarean section is an operation rarely practised by a gynecologist, belonging, as it does, to the obstetrician, or in emergency to the general surgeon. Experience has taught the obstetrician that only the test of labor justifies so important a decision, and the surgeon in this regard may profit by obstetric experience.

The treatment of the Porro operation is unsatisfactory because cœliohysterectomy with intraperitoneal treatment of the stump is included under the head of the Porro operation. Under the heading of Cæsarean Section the excellent advice is given that if the uterus has been infected before the operation the whole organ should be removed. Under the paragraph upon the Porro operation it is stated that this method of procedure has been superseded by hysterectomy. These statements place the Porro operation in its proper light, namely, as a wellnigh obsolete surgical procedure. It would have been better had the writer described complete removal of the uterus and cœliohysterectomy, as both are logical and satisfactory operations, and both are now performed in place of the Porro operation. Considerable space is given to symphysiotomy, and the method by direct incision, as practised by Farabeuf, and Ayers' subcutaneous method are described. We have had satisfactory results in eight cases by the subcutaneous method as originally practised, and have obtained excellent apposition by immobilizing the pelvis with a single broad strip of adhesive plaster.

We cannot close a review of this volume without again calling attention to the excellent work of its editor. It is our regret that he did not write more of the volume, and that the requirements of publication should have hampered him in the arrangement and choice of the contents of the volume.

The book is clearly printed, and most of the illustrations are clear and good. Some of the colored illustrations are not remarkable for excellence. The book contains a complete and efficient index.

E. P. D.

AN AMERICAN TEXT-BOOK OF PATHOLOGY. Edited by LUDVIG HEKTOEN, M.D., and DAVID RIESMAN, M.D. Illustrated. Philadelphia and London: W. B. Saunders & Co., 1901.

IN criticising an American text-book of pathology the reviewer must take into account the fact that the publishers claim that it is "the most representative treatise on the subject that has appeared in English," and that it is the outcome of the desire of the editors and publishers to place in the hands of the medical student and physician "a comprehensive text-book on the essential principles and facts in general pathology

and pathological anatomy." The question, of course, is to what extent the assertion is justified and the desire realized. As a representative text-book upon American pathology we should expect to find among the list of contributors to the volume the names of certain illustrious workers in this field. These, however, do not appear. It must not be supposed that the men who have contributed are incapable or unknown, but the omission of the others to whom we allude must be accounted a defect in the work. There is another feature in the book which we think merits criticism. It has been the custom for some time in German and American text-books to add to each section, either by way of a preface or appendix, a list of the more important articles which have been published on the subject treated, and this is particularly important in a text-book of pathology, a subject whose field is so vast that practically all men in it are specialists. In the present book, had the literature been added, and particularly the literature that has been contributed by American investigators, not only would the book have been improved, but a great service would have been done to American pathology; for perhaps because we are so prone to quote German authorities we know too little regarding the work of our colleagues in the United States. Another fault that, however, is not found in all the articles is a tendency to omit allusion to other authors or to quote authors without reference. In a subject as theoretical as is pathology, concerning the simplest facts of which there is at the present day so much discussion, it is not sufficient to know the opinion of any one man, no matter how authoritative his opinion may be; but we expect to be informed, at least in a general way, concerning the ideas of other workers, and some of the articles in which this fault is carried to extreme are more suitable for quiz compend than for a formal text-book of pathology, for they are a mere statement of the current teaching without any critical discussion of the subject. In other cases the author chiefly used in the preparation of the article is entirely too obvious, and as a result there is a lack of independence.

On the other hand, some of the articles are most admirably treated. Authorities are stated frequently, references are given, and the subject is treated in a scholarly and thorough manner. This is particularly true of the articles by the editors, of Dr. Collins' section on the pathology of the nervous system, and Dr. Beyea's article upon the female genital tract, although in the last two articles references are too frequently omitted. Among omissions, some of which are perhaps inevitable in the first edition of any text-book of this character, we may note the discussion of certain pathological cell-forms that are now exciting considerable interest in pathology. We refer particularly to the plasma cells, mast cells, to the significance of the eosinophile cells in tissues, to the different varieties of giant cells, etc. The discussion of the pathology of the lymph glands and spleen, which might have been more properly classified, is entirely more adequate. A more thorough account of the present status of the infectious theory of cancer would have been desirable, for although it cannot be said that we have reached any definite conclusions, the results of experiments have contributed largely to our knowledge of the subject of cancer and even to our knowledge in some other directions. The subject of pseudoleukæmia receives scant attention, particularly in view of the recent advance in the pathology of this disease. Some of the illustrations deserve

criticism. Many of them are borrowed; the sources are various, and this gives rise to more or less confusion in appearance. This is particularly true of the illustrations in the article upon bacteriology. It is always desirable, if possible, to have the illustrations in a text-book prepared according to some definite and uniform plan. It is almost supererogation to cite Ziegler's text-book as a model to be followed in this respect.

The book is well printed. The errors of proof-reading appear to have been singularly few, and an earnest and largely successful effort has been made by the editors and contributors to include the very latest knowledge upon each subject. J. S.

PROGRESSIVE MEDICINE, Vol. I., 1902. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by HOBART AMORY HARE, M.D., Professor of Therapeutics and Materia Medica in the Jefferson Medical College of Philadelphia. Octavo, 452 pages, 5 illustrations. Philadelphia and New York: Lea Brothers & Co.

THE first issue of *Progressive Medicine* for the current year reviews the literature of 1901 under the heading of Surgery of the Head, Neck, and Chest; Infectious Diseases; the Diseases of Children; Pathology; Laryngology and Rhinology, and Otology.

The division on Surgery, by Charles H. Frazier, the longest in the volume, covers 128 pages. Especially interesting is a very complete discussion of the advances in the surgery of the Gasserian ganglion and the fifth nerve, to which Dr. Frazier, in conjunction with Dr. William G. Spiller, has recently made a noteworthy contribution. The X-ray treatment of skin-cancers is a topic of timely interest, receiving due attention, while a section of fully ten pages is devoted to a very complete discussion of our present knowledge of enlargements of the thyroid gland, their pathology and their surgical treatment. Considerable space also is devoted to the recent literature of the surgical treatment of injuries and surgical diseases of the heart, pericardium, and large vessels, a branch of surgery which bids fair to achieve brilliant and, until lately, unhopèd-for success.

The article on Infectious Diseases has been entrusted to Frederick A. Packard, who contributes seventy-seven pages, twenty-four of which are devoted to the year's literature upon typhoid fever. The complications of the disease, many of them of quite unusual occurrence, are very fully discussed. Four and a half pages are devoted to diphtheria, which fairly indicate the extent and importance of the most recent literature of this disease, upon which, until lately, so much has been written since the introduction of the serum treatment. Indeed, activity in work upon diphtheria apparently for the time has been exhausted, and there seems little more to be done for some time to come. Varicella, smallpox, measles, and dysentery together occupy nine pages, which sufficiently cover new points of interest. The section on Tuberculosis is mainly devoted to Koch's startling paper, read before the British Congress on Tuberculosis, and to Ravenel's somewhat contradictory conclusions on

the same subject. Important work upon the insect-borne diseases, yellow fever and malaria, receives notice, and short sections upon Influenza, especially its nervous complications, upon scarlet fever, and upon cerebro-spinal meningitis complete the review.

The Diseases of Children, by Floyd M. Crandall, occupying fifty-seven pages, is a significant indication of the activity of work in this increasingly important branch of medicine. Infant feeding, as usual, claims considerable attention, and the recent advances are well outlined in a section covering eight pages. The general acceptance of the percentage basis of feeding is clearly indicated in all the contributions to the subject, though the tendency to simplification of methods, and consequently less regard for accuracy, seems to be at present in favor. The rest of the article, which is divided according to the diseases of the various systems, adequately covers the new work of the past year. Passing mention may be made of the fact that, as shown in a discussion upon summer diarrhoeas, the leading authorities of the country are very closely in accord as to the essential treatment of these diseases.

Pathology, reviewed by Ludvig Hektoen, in an article of eighty-seven pages, the second longest of the volume, is restricted to the original investigations in serum pathology, immunity, pathogenic micro-organisms, general pathological histology, and oncology. Lack of space forbids more than general mention of the vast amount of new work that is here very satisfactorily sketched, the number of references far exceeding those of any other division of the volume. Considerable space is devoted to the recent important studies of the etiology and pathology of cancer.

Laryngology and Rhinology are considered in an article of fifty-one pages, by St. Clair Thomson. Especially important is a very complete presentation of the general therapeutics of suprarenal extract and adrenalin, which have lately given promise of their value in throat and nose diseases.

The volume concludes with the section on Otology, by Robert L. Randolph. While naturally the shortest division of the six, it presents an adequate review of the progress of the year in this branch of medicine, especial mention being deserved for the sections on vibratory massage and upon the surgery of the mastoid.

Referring, in conclusion, to the volume as a whole, it may be said that the present issue of this very valuable periodical is fully up to the high standard maintained in the previous issues, and that it constitutes an indispensable adjunct to the working library of all who labor in the arduous field of advanced medical knowledge. T. S. W.

A TEXT-BOOK OF DISEASES OF WOMEN. BY CHARLES B. PENROSE, M.D., Ph.D., formerly Professor of Gynecology in the University of Pennsylvania, Surgeon to the Gyncean Hospital, Philadelphia. Fourth edition, revised. Philadelphia and London: W. B. Saunders & Co., 1901.

THE fourth edition of this book has many new features, both in the text and by the addition of instructive drawings, which place it in a still higher plane of excellence than before. For the student and the general practitioner it is especially trustworthy, dealing with each subject in a clear, concise manner, presenting the best teachings of

modern gynecology without the tedious rehash of antique methods, and outlining in the main only one mode of treatment, which is to the student a distinct advantage. The chapters on the diagnosis and repair of tears of the perineum and cervix are of especial value. These conditions are of so much importance that they deserve more attention than is ordinarily given to them in text-books on gynecology. Penrose's description of their appearance and their diagnostic features is excellent, while his outline of the operative treatment is rational and clear. The methods of Emmet have been carefully revised, and, while still followed in the main, many new points have been added, increasing their efficiency. In the chapter on "Prolapse of the Uterus" the author's treatment of the subject is most wise. It is very well described in the text, and a series of drawings showing the different steps of the operation for its relief elucidate it clearly. The pathological features of each condition and disease have received much more attention in this than in the previous editions of the book. The reason that this side of the work is perhaps not as complete as it might be is given by the author in his preface when he states that "he has, as a rule, omitted all facts of anatomy, physiology, and pathology which may be found in the general text-books on these subjects. In the chapter on "Cancer of the Uterus" cancer of the ovary has been added, and the insertion of new plates in this chapter makes it an excellent one. The withdrawal of many old plates and the substitution of new ones more clearly illustrating the admirable quality of the text would be wise, as some of the drawings are certainly not up to date.

The chapters on "Diseases of the Ovaries" have been carefully rewritten and improved, and a description of teratoma of the ovary has been added. Endothelioma of the ovary is also a useful addition to this chapter.

The chapter on "Diseases of the Urethra and Bladder," while not as full as it might be from the practitioner's stand-point, is nevertheless sufficiently adequate for the student.

The chapter on the "Technique of Gynecological Operations" has been brought well up to date, and very clearly and decisively written. The author's large and successful experience as an operator make it of great worth. The post-operative treatment of cases is somewhat old-fashioned.

In a few years the third edition of this book has been exhausted; that it has been very successful, and that its popularity is well deserved, is unquestioned.

R. F. W.

MODERN OBSTETRICS, GENERAL AND OPERATIVE. By W. A. NEWMAN DORLAND, A.M., M.D. Second edition, revised and enlarged. Pp. 797. Philadelphia and London: W. B. Saunders & Co., 1901.

THIS is an enlarged edition of a manual of obstetrics originally designed to facilitate a review of the subject by students. The present book is a literary résumé of the subject in which a fair review up to the time of publication is given. The book contains little original matter, and is not enriched by the results of an extensive experience. In type and illustrations attention has been paid to economy at the expense of the volume.

E. P. D.

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Diabetes and Pregnancy.—HERMAN (*Edinburgh Medical Journal*, February, 1902, p. 119) reports a case of pregnancy complicated by diabetes. The patient was a Jewess, aged thirty years. She had been married twelve years and had had seven children, the last three of which had been born dead. At about the third month of her eighth pregnancy she began to complain of weakness, pruritus, thirst, and emaciation. She had suffered from thirst during her previous pregnancies. She was informed by her physician about this time that she had diabetes. At about the seventh month she began to have uterine pains, associated with severe vomiting. She was admitted to the London Hospital in an almost moribund condition on March 26th. She was practically unconscious. The following day the membranes were ruptured, and she was later on delivered of a dead child weighing five and three-quarter pounds. The amniotic fluid was excessive. For the next few days the patient's condition varied markedly. She had rigors and fever, and gradually became weaker, and died on April 6th, but apparently not of diabetic coma. The urine averaged about 70 ounces daily. The amount of sugar ranged between 50 and 70 grammes. One examination failed to show any diacetic acid. The autopsy revealed that there was a round-celled sarcoma involving the right pleura. The right pleural cavity contained 2½ ounces of pus. The pancreas was not examined.

According to Herman, Matthews Duncan was the first to study the effects of diabetes and pregnancy on one another. Recently the subject has been carefully studied by Kleinwächter. According to the latter, two-thirds of the cases of diabetes in women do not begin until after the menopause. The disease is consequently infrequent as a complication of pregnancy, and when it does occur in a child-bearing woman it usually suppresses menstruation and

sometimes produces atrophy of the uterus. Our knowledge of the clinical history of this complication of pregnancy is based on but a few cases.

The symptoms of diabetes coming on during pregnancy may disappear within a short time after delivery, and recur again in subsequent pregnancies. This indicates that the pregnant condition favors the production of the disease. Statistics show that prognosis in these cases is more favorable than in those in which diabetic patients become pregnant. Some of the cases of pregnancy associated with intermittent diabetes have recovered from the latter after the period of child-bearing has ceased. When a diabetic woman becomes pregnant, however, the disease usually becomes aggravated and its progress hastened.

Premature delivery, due to intra-uterine death of the child, has occurred in about two-thirds of the published cases of pregnancy with diabetes. Hydramnios is rather frequent. The children have often been large. In two cases the liquor amnii contained 3 per cent. of sugar, and in one the fetal urine contained sugar.

The question as to whether pregnancy should be interrupted or allowed to go on is apparently not yet a settled one among obstetricians. According to Herman, the early termination of pregnancy will prove the best treatment for a pregnant diabetic. If pregnancy be allowed to go on the following are the possibilities:

1. The chances are two to one that the child will die in utero. Its life is therefore not of much account.

2. It is possible that when the pregnancy is over the diabetes may get well. This is more likely to take place the earlier the pregnancy ends.

3. The patient may die from collapse and coma soon after delivery. This is more likely to occur the longer that pregnancy has gone on. One of these three events has occurred in most of the published cases; those in which pregnancy and labor were gone through with without influencing the diabetes or being influenced by it are in the minority.

Kleinwächter advises against premature delivery, on the ground that there is no evidence of its benefit. Apparently, however, it has only been induced once in the published cases, and then in a case in which the mother was moribund. As stated, Herman advises acting on probability and inducing premature delivery.

Apparent Contagion in Rheumatism Due to Meyer's Diplococcus.—ALLARIA (*Revista Critica di Clinica Medica*, 1901, vol. ii., p. 805) states that the remarkable observations of Meyer in Leyden's clinic have been reported in a previous number of this journal. This observer succeeded in cultivating a diplostreptococcus from the tonsils of five cases of acute rheumatism with angina. Bouillon cultures of this streptococcus produced characteristic polyarticular arthritis in rabbits. The exudates in the joints were usually serous, and sterile by ordinary culture methods. Meyer and Leyden both believe that this diplostreptococcus is the true specific agent of acute articular rheumatism.

Allaria, in Bozzolo's clinic, instituted control experiments, in the course of which he came across three cases of so remarkable a character as to merit publication. The first, a young woman, aged sixteen years, who had pre-

viously had several attacks of acute rheumatism, developed an acute tonsillitis, followed by a fresh attack of articular rheumatism lasting about four weeks. The sister of this patient, after nursing her, developed a similar attack, entering the hospital six days later. A friend of this patient, who had incipient pulmonary tuberculosis, and was in the hospital at the same time, developed also an acute tonsillitis after associating with the second case. Two days after the onset of the tonsillitis, acute rheumatism set in. A nurse, aged twenty-two years, who swabbed the throat of the first patient was seized with tonsillitis, followed by polyarticular rheumatism and high fever of a month's duration. Another nurse who took her place and attended the first two patients developed also tonsillitis, with polyarticular rheumatism; this was fortunately mild and without complications, though it kept her in bed for twenty-three days. Finally, a third nurse who followed this latter, although she took great precautions, developed severe tonsillitis, which lasted more than a week, and was associated with high fever. In all these cases the tonsillitis began violently with severe chills and rapid rise of temperature; the tonsils were much enlarged, although in two cases only were there slight traces of exudate. In the first three patients the bacteriological examinations of the throat were made. These showed streptococci, agreeing in all essential points and characteristics with those described by Meyer. A cubic centimetre of a forty-eight-hours' culture injected into guinea-pigs was followed by no clear reaction at the point of inoculation. The animals, however, gradually lost their vivacity, and four or five days after the injection painful swelling appeared in various joints, especially the tibiotarsal articulations of the posterior extremities. This swelling gradually disappeared, and the animals recovered. In those animals which were killed the fluid proved to be serous; there was subcutaneous oedema about the affected articulations, but no muscular infiltration. Under the microscope the exudate showed but few corpuscles and no micro-organisms, but on culture similar streptodiplococci were obtained.

In none of the animals used did exudates appear in the larger serous cavities. Cultures from the pleura, peritoneum, and heart's blood were negative.

A fourth case differed from the others in that the attacks were not preceded but followed by angina, and also in the greater virulence of the micro-organisms, which otherwise possessed characteristics similar to those of the preceding case. In this instance the control animals died rapidly with a general septicæmia. The author believes that while it is as yet impossible to assert positively with Meyer that this organism is the specific causal agent of acute rheumatism, yet if acute articular rheumatism is a true nosological entity and not a syndrome, as has been believed by some authors, there is much which would lead one to adopt this view.

Experimental Cholecystitis and Cholangitis of Auto-infectious Origin.

—EHRET and STOLZ (*Berliner klin. Wochenschrift*, 1902, vol. xxxix., p. 13) state that it has been shown that anything tending to diminish the motility of the gall-bladder and favor the accumulation of residual bile offers opportunity for the development of bacteria which otherwise do not flourish in the bile-passages. Especially is this true of the presence of foreign bodies

which hinder the entire expulsion of the bile from the gall-bladder. Ehret and Stolz have conducted a number of experiments to determine whether the injection of aseptic bodies into the gall-bladder or common duct might eventually be followed by the development of auto-infectious cholecystitis or cholangitis. In five cases the gall-bladders of dogs were filled with small hollow glass balls measuring about 10 to 13 millimetres in diameter. These had been carefully sterilized and broken at two points opposite one another. All the dogs recovered perfectly from the operation. One of the animals which was killed in two months showed a great abundance of bacteria in the gall-bladder. Three months after operation the dogs were placed under unfavorable conditions of diet. Severe diarrhoea followed in all four dogs. Two died of suppurative cholecystitis.

In six other instances small bits of absorbent cotton were introduced into the gall-bladder. Four of these dogs died after attacks of diarrhoea from eight to sixteen weeks after the operation. In all there was a suppurative cholangitis. In two in which laparotomy was done eight to ten weeks after the first operation the tampons were found in the common duct. The gall-bladders were greatly distended and showed an abundance of non-virulent bacteria. The bacteria varied in nature, being similar in the main to those which one finds in the normal bile of dogs when great quantities are used in the culture. They were essentially the same as those which appear after operations diminishing the motility of the gall-bladder, and also to those which usually entirely outgrow in bacteria which are introduced in attempts to develop an experimental cholecystitis. The spontaneous development of cholecystitis and cholangitis appeared to be favored by a temporary increase in virulence of the colon bacilli as a result of diarrhoea.

These observations are interesting in that they form an entire parallel experimentally with the conditions occurring clinically in cholelithiasis. The infection in these instances clearly occurred through the ducts.

A Case of Tuberculosis of the Skin following Accidental Inoculation with the Bovine Tubercle Bacillus.—RAVENEL (*University of Pennsylvania Medical Bulletin*, February, 1902, p. 453) reports a case of tuberculosis of the skin resulting from the infection of a wound during autopsies on two tuberculous cows. He has already reported three similar cases.

On July 27, 1901, Dr. G., while performing autopsies on two cows, the subjects of experimental tuberculosis, wounded the flexor surface of one of his wrists. By September 10th there was a nodule in the skin at the seat of the wound, measuring 15 by 8 mm. This was excised on September 14th. Portions were inoculated into two guinea-pigs, both of which developed generalized tuberculosis. Sections of the nodule showed the characteristic histological picture of the tubercle and also large numbers of typical tubercle bacilli.

Ravenel regards this case of great importance, considering Koch's opinion that human and bovine tuberculosis were different diseases and not intercommunicable. He says that such cases do not settle the entire question of the transmissibility of bovine tuberculosis to man, but that they prove most conclusively that the bovine germ finds soil and conditions in the tissues of man suitable for its multiplication.

SURGERY.

UNDER THE CHARGE OF

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Aneurism of the Mesenteric Arteries.—GALLIVARDIN (*Gaz. Hebdomadaire de Med. de Chir.*, 1901, No. 82) states that in studying the histories of cases of obliteration of the mesenteric arteries, which are composed nearly entirely of cases of embolus of the superior mesenteric artery, one is surprised not to encounter more frequently an infectious endocarditis as the origin of the embolic process. A recent monograph states that out of fifty-five cases of embolic obliteration of the vessels, the presence of an infectious endocarditis was only proven in four cases. The reason for this is that the septic emboli cause an alteration in the walls of the artery in which they may lodge, which tends to cause dilatation of that vessel, and so these cases are not reported as embolism but as aneurisms of the superior mesenteric artery. From the etiological point of view these aneurisms of the mesenteric artery should be considered in two groups: first, those of unknown origin, and second, those due to a septic embolus the result of an infectious endocarditis. Out of sixteen reported cases the author finds that seven should be considered in the first class, and of these in two cases there was a specific history, but in the other five cases the cause could not be determined. It is interesting to note that three of these five cases occurred in very young women, aged twenty, twenty-three, and twenty-four years, respectively. The remaining nine cases were the result of a septic embolus. The superior mesenteric artery was the site of the aneurism in nearly every case; a similar predominance is noted in the cases of embolism. Out of twenty-three reported cases, in only two cases was the inferior mesenteric artery affected. The aneurismal tumor is nearly always situated near the superior portion of the mesenteric artery, at the point where it penetrates the mesentery and gives off its first branches. The sac is rarely large, generally being about the size of a hen's egg. The sac may rupture in the folds of the mesentery or directly into the peritoneal cavity. The symptoms may be separated into two classes or periods: those of the evolution of the sac and the period of rupture. The symptoms of the first period, or that of evolution, vary much in different cases. The sac may occasion absolutely no symptoms, the patient being unaware of any abdominal lesion until the sac ruptures, as in the case reported by Lecadre. However, there usually exists some pain or other vague abdominal symptoms. In some cases the abdominal pains are so severe as to suggest the presence of an aneurism of the abdominal aorta. In only

four of the reported cases could the tumor be felt on palpation. In the last period the patient may die before the rupture of the sac from the extension of the causal endocarditis or as the result of some other intercurrent disease. However, rupture is a frequent termination of these cases. In all cases in which this happened death was not sudden, but soon followed after the appearance of abdominal symptoms, violent abdominal pain, and later nausea and vomiting. The patient has the facies abdominalis, is pale, and dies in a few hours. The same symptoms are caused by a rupture in the walls of the intestines in the region of the duodenum. The diagnosis of rupture of an aneurism of the mesenteric artery is nearly impossible, because of the difference in the symptoms which have been observed in particular cases, the absence of characteristic symptoms and the frequent presence of anomalous symptoms, and in the greater majority of cases the diagnosis has only been made at the autopsy.

Complete Atrophy of the Stomach.—VON ČAKOVIC (*Archiv f. klin. Chir.*, 1902, Band lxx., Heft 2) recommends jejunostomy in the following types of cases: (1) In marked stenosis of the oesophagus, when gastrectomy cannot be performed because of the atrophy of the stomach. (2) In those cases of carcinoma of the stomach when a radical operation is either contraindicated or it cannot be performed. (3) In cases of stenosis of the pylorus or cicatrix of the stomach where the patient is very weak or the cicatrices are apt to be multiple. (4) In cases of ulcer of the stomach with frequent hemorrhages which do not respond to treatment and there is danger of death from inanition. (5) In all cases of poisoning by corrosive liquids where there is danger of death either from rupture of the stomach, or inanition, or as the result of such corrosion, when no other operative measure is possible. (6) In all cases of insufficiency of the sutures after operations upon the stomach when any other method of relief is impracticable. (7) When a radical or palliative operation has proved to be a failure. The results of jejunostomy are not very encouraging, and this is not surprising when one considers that this is an operation which has been reserved as a last resort, either when other operative measures have been tried and proved to be failures, as the disease is so extensive that no other operation is possible. The author reports sixty-seven cases, with twenty-five deaths due to the operation, or a mortality of 37.3 per cent.

The Diagnosis of Tuberculous Peritonitis in Children.—KISSIL (*Archiv f. klin. Chir.*, 1902, Band lxx., Heft 2), after an interesting review of this subject, states in conclusion: (1) Tuberculous peritonitis is more common in children than is usually believed to be the case. (2) Many of the cases of so-called "spontaneous ascites" are in reality cases of ascites the result of a tuberculous peritonitis. (3) In many cases of tuberculous peritonitis the exudate will become absorbed under an appropriate tonic treatment, and eventually the patient will entirely regain his health. (4) In the majority of cases the onset of the disease is unnoticed. The first thing usually noted is that the child has become thin and pale without apparent reason. (5) The presence of a serous pleurisy at the same time as the tuberculous peritonitis markedly facilitates the diagnosis. (6) The most valuable diagnostic

symptoms are those caused by the presence of adhesions. (7) Examination of the fluid removed from a case of tuberculous peritonitis will show that it is rich in albumin and of high specific gravity. (8) Operation generally shows that the entire pericardium is involved in the tuberculous process. (9) The diagnosis is especially difficult in cases of chronic ascites complicated by tuberculous pericarditis. (10) It is very exceptional that the onset of tuberculous peritonitis is anything but insidious.

Carcinoma of the Lips.—JANOWSKY (*Archiv f. klin. Chir.*, 1902, Band lxx., Heft 2), as a result of an extensive review of the literature, states in conclusion: (1) The operation for carcinoma of the lips should not be considered as dangerous, and in the greater majority of cases union by first intention takes place in from nine to fifteen days. (2) All the lymphatic glands of the submental and submaxillary region must be removed, or recurrence will occur. (3) When necessary the inferior maxilla may be resected without danger. (4) The lymphatic glands are affected soon after the onset of the disease—in two or three months—but this should not prevent a radical operation from being successful. In many cases the glands are involved when not palpable, and recurrence will follow if they are not removed. (5) In 49 per cent. of the operated cases the end results were good. (6) In the cases which had recurrence the disease reappeared in the greater majority of cases within six months and nearly all the rest within the first year; in only a very few cases was the recurrence after a lapse of one or two years. (7) When recurrence took place it was generally found at the point where the disease first appeared or else in the submental region. (8) The operative results were influenced by the duration of the disease and the degree of malignancy in each particular case. Cases which were not severe were operated on successfully as late as several years after the onset of the disease. (9) Old age is not a contraindication to operation; the best operative results were obtained in patients between sixty and seventy years of age. (10) In cases of recurrence a second operation is only palliative and possibly may prolong life for several years. (11) Carcinoma of the upper lip is nineteen times less frequent than that of the lower lip, and carcinoma of the lip is ten times more frequent in males than females. In women carcinoma of the upper lip is five times more frequent than it is in men. (12) After a careful study of 178 cases, it is impossible to state that carcinoma of the lips is more frequent in any occupation or station in life than in any other. (13) The disease is most common in persons between the ages of sixty and seventy years. (14) Twenty-five per cent. of all the cases are rapid in growth and markedly malignant, 15 per cent. are less severe, while the remaining 60 per cent. are a compromise between these two.

What is the Prognosis in Tubercular Spondylitis?—MOSHER (*Brooklyn Medical Journal*, January, 1902) states that the consideration of the prognosis devolves the necessity of bearing in mind that tubercular spondylitis is nearly always a disease of childhood and that early diagnosis, with proper and rigid treatment, influences very strongly the ultimate results. The predisposing causes of Pott's disease are traumatism, hereditary syphilis, tuberculosis, anæmia, scrofulosis, scorbutus, and malnutrition from any cause. The exciting causes are fevers, sudden chillings of the body, overexertion of the

spine, and traumatism. There seems to exist among the profession as well as the laity the idea that traumatism is the only cause of tubercular spondylitis. The author believes that in the vast majority of cases, without the existence of a marked predisposing etiological condition, the slight injuries so often assigned as the cause of spondylitis would have no special significance. This disease begins insidiously with indefinite symptoms such as malaise, fever, night-cries, restlessness, lassitude, loss of appetite, indifference to surroundings, emaciation, irritability, and peevishness, all of which are more or less common in the severe illnesses of childhood. The pathognomonic sign is spinal rigidity, and it is the first marked symptom to attract attention, and is manifested by the child's attitude, which is characteristic. Later comes spinal deformity, progressing, abscesses opening, usually at some distance from the seat of the disease, and paralysis, more marked in the motor nerves, affecting the parts below the deformity. For a vast majority of cases it is possible to diagnose Pott's disease before there exists any apparent deformity. The diagnosis with the late symptoms present, such as deformity, abscesses or paralysis, is very easy, and is usually made by the parents, who will venture the information that the child has been ailing for some time and has been treated for various diseases. In the earlier stages, however, where the symptomatology is obscure, it becomes more difficult, but with proper attention to history, a thorough examination and careful arrangement of symptoms the number of self-diagnosed cases will be greatly reduced. This disease seems to lack to a marked degree the *vis medicatrix naturæ* existing in most diseases, and, therefore, instead of days and weeks, months and years are required for its cure, or even to stay its progress. To improve the patient's general health, to lessen and relieve pain, to prevent traumatism by, as far as practical, preventing the movements of the spine, to take the weight of upper part of body off the spine, to avoid, if possible, any deformity, or, if one already exists, to prevent its increase, are marked indications for the treatment of tubercular spondylitis. It is important that the patient's resistance be kept at the highest point by the judicious use of the proper medicinal agents indicated in each individual case. Iodoform (internally), creasote, syrup of the iodide of iron, cod-liver oil and stimulants have all proved of value in the author's experience. In the mechanical treatment recumbency seems to offer the best results in active spondylitis. By this is meant recumbency just so long as there is any tenderness or active disease.

Abscess and paralysis are the usual complications and indicate that the spine is not receiving proper support. Aspirations may be repeated in abscess, but where aspirations fail to obliterate the sac, or the pus is of such a character that aspiration is impossible, or when there is external inflammatory evidence, the abscess should be opened and drained, and, if psoas, counter-drained, vigorously swabbed out with gauze (not curetted), thoroughly irrigated, treated aseptically, and recumbency adopted. The paralysis due, supposedly, to inflammatory lesions more than pressure, should be treated by recumbency and counter-extension and by iodide of potash in increasing doses. This treatment usually takes many months, but if it fails Colot's forcible correction is in order. Laminectomy, owing to its seriousness, is not so encouraging. The author reports fifty-three cases of tubercular spondylitis, ranging in age from fifteen months to sixteen years, the average age being six years. In five

cases the cervical vertebræ were involved; in ten cases the cervicodorsal region was involved; in twenty-five cases the dorsal vertebræ were involved; in three cases the dorsolumbar region was involved, and in nine cases the lumbar vertebræ were involved. The time of treatment varied from one year to seven, average three and a half. Three cases were treated by recumbency alone; four cases were treated by brace alone; forty-six were treated by recumbency assisted by jackets, braces, etc. Time of recumbency one month to five years. Average two and a quarter years. Twelve cases suffered from abscesses, one from empyema, five from paralysis, one from hip-joint disease, and two from phthisis pulmonalis. Four cases had no deformity, twenty-five slight deformity, ten moderate deformity, and fourteen marked deformity when last seen. Seven died.

PEDIATRICS.

UNDER THE CHARGE OF

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Glandular Reaction in the Diseases of Children.—LABBÉ and BERTIN (*La Presse Médicale*, January 29, 1902, p. 99), in a thoughtful paper under this caption, point out that in the first years of life the lymphatic glands, like other lymphoid structures, have acquired their full anatomical development and are in their highest functional activity. They give origin to lymphocytes whose passage in great numbers into the circulation determines a leucocytosis distinct from that of the adult. In the course of divers acute and chronic affections they react with an intensity far beyond that manifested by the glands of the adult, the intensity of the reaction often being disproportionate to that of the excitation, so much so that the glandular reaction often appears to constitute the entire disease. This intensity of glandular reaction constitutes one of the peculiar characteristics of disease in childhood.

This peculiar physiological activity of the child's lymphatic system, which by its local functions arrests and destroys morbid germs, or at least attenuates their virulence, by producing leucocytosis, serves to explain why certain infections like pneumonia and typhoid fever are more benign in general in the child than in the adult.

The authors conclude by expressing the hope that artificial provocation of this activity of glandular reaction may be taken advantage of in the treatment of disease.

A Stigma of Degeneration Observed in Cretins and Idiots.—KOPLIK and LICHTENSTEIN (*Archives of Pediatrics*, February, 1902, p. 81) describe a hitherto unmarked anatomical sign observed in cretins and several types of

idiots. This is a prominence in the region of the antithenar eminence over the position of the pisiform bone. This prominence is immediately adjacent to the groove which separates the palm of the hand from the forearm. It is distinctly localized to this part of the antithenar eminence, and, viewed from the side, rises abruptly from the groove, giving a bayonet-like appearance. It is apparently an over-development or hypertrophy of the small muscles of the inner border of the hand attached to the os pisiformis, as well as, perhaps, an enlarged condition of the bone itself. Inasmuch as this prominence was observed in a cretin only three months old, it seems just to assume that it is a congenital anomaly and not a hypertrophy caused by the act of crawling on the hands and knees, as was first thought from its resemblance to a similar prominence seen on the forefoot of the domestic cat. This prominence has not been seen in a perfectly normal infant or child. It was observed in idiots, cretins, microcephalic idiots, and children congenitally deficient. So far it has not been seen in the Mongolian idiot, though a large number of cases of this affection have been examined. It has been found in all cases of cretinism, was absent in a case in which there were certain physical signs of cretinism, but in which the child was quite intelligent. It was also found in two markedly intelligent children with macroglossia and polydactylia.

The authors are not prepared to draw any definite conclusions, but consider this prominence a true anatomical stigma of degeneration, and, as far as cretinism is concerned, a highly interesting part of the symptomatology of this disease.

Syphilitic Enteritis under the Form of Melsena Neonatorum.—EßER (*Archiv f. Kinderheilkunde*, 1901, Bd. xxxii., S. 177) reports the observation of a newborn baby in which melsena appeared on the fifth day after birth, causing death six days later. The mother was a primipara of healthy appearance; the father was unknown.

At the autopsy the site of hemorrhage was found in the jejunum. Here there was a thickening of the mucosa due to a circular infiltration of small cells, with the formation of several ulcerations. This diffuse infiltration was particularly accentuated about the vessels, where in places it had produced obliteration. Analogous lesions were found in the liver and in the spleen, the latter being hardened and enlarged. There was also a certain degree of osteochondritis in the bones that were examined. This ensemble of lesions marked the melsena in this case as of syphilitic origin directly dependent, upon a specific ulcerative enteritis.

A Contraindication to Circumcision.—REY (*Jahrbuch f. Kinderheilkunde* June, 1901) cautions against indiscriminate circumcision. He calls attention to the fact that in infancy there is what may be called a physiological phimosis, so that retraction cannot be made without exerting undue force. The preputial orifice expands during childhood, so that by eight years of age it can usually be retracted readily. Unless obvious symptoms are produced by an exaggeration of this physiological condition of the prepuce in infancy operation is unnecessary. The period of life up to the fifth year is one in which cystitis and urethritis are common complaints, and urinary troubles during this period are very rarely due to phimosis. The existence of urethral

or cystic disease is the strongest contraindication against producing a wound at the end of the penis.

In the presence of cystitis, which is first indicated by an ammoniacal odor, of the napkins, the exposed skin surfaces become irritated and the prepuce becomes inflamed or ulcerated from evaporation of the urine and external pressure. If, however, there is no prepuce in front of the glans the urethral orifice is liable to ulceration, which may be followed by stricture, with its train of secondary sequelæ, or may leave scars that in later life may set up incontinence of urine of an intractable character.

The author, therefore, believes that the prepuce is really a great protection to the glans in the presence of a cystitis, and he advises the avoidance of circumcision until after the fifth year, unless it be required soon after birth for obstruction not amenable to cure by simple dilatation.

Treatment of Typhoid Myocarditis in Children.—CALAMET (*Traitément médicamenteux de la Myocardite typhoïdique*, Thèse de Paris, 1902) considers that the bath treatment of typhoid is not contraindicated by the appearance of myocardial changes if the possibility of syncope is guarded against by subcutaneous injection of sulphate of strychnine or sparteine, by the application of an ice-bag over the heart, or by the injection of saline solution (Guinon).

In regard to the medicinal treatment of myocarditis, he admits the value of digitalis in dose of 20 to 30 drops of the tincture. Under its use the pulse becomes slower and increases in force, fullness, and regularity, and vascular tension increases. Its cumulative effect, its power of provoking asystole, and its slowness of action are disadvantages. Caffeine does not have a cumulative action, and it acts rapidly in dose of 0.25 to 1 gramme a day, by hypodermic injection. It sometimes has the inconvenience of producing irritation and even delirium. In these cases it advantageously may be replaced by sparteine. One or two syringefuls of a solution containing 40 centigrammes of sulphate of sparteine to 10 grammes of distilled water may be used. Camphorated oil (1 to 10) or sulphate of strychnine (one-fourth of a milligramme, $\frac{1}{47}$ of a grain) may be employed; and ergotin, in virtue of its vasoconstrictor action, has been successfully used by Demange.

Syncope may be treated by the usual methods: lowering of the head, flagellations, frictions, tongue traction.

Erythema Scarlatiniforme and True Scarlatina in the Course of Diphtheria.—LOBLIGEOIS (*Des érythèmes scarlatiniformes et de la Scarlatine vraie au cours de la diphthérie*, Thèse de Paris, 1902) calls attention to the importance of examination of the blood and the urine in determining the diagnosis between a scarlatiniform erythema due to the use of antidiphtheritic serum and a true scarlatina occurring in the course of diphtheria treated by the serum. Such a differential diagnosis is sometimes almost impossible by the clinical features alone. In these cases an examination of the blood at the beginning of eruption offers the following distinguishing features: *a.* Polynucleosis is more marked in scarlatina than in the erythemas due to serum. *b.* Eosinophiles are more rare in scarlatina. *c.* In the scarlatiniform erythema abnormal forms are encountered which are absent in scarlatina. Examination of

the urine also furnishes valuable aid: the diazo-reaction, which is very frequent in scarlatina, is exceptional in diphtheria, and particularly in scarlatiniform erythema.

THERAPEUTICS.

UNDER THE CHARGE OF

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Adrenal Extract and Hæmatemesis.—DR. J. ADAM presents a new method in the use of this remedy. Fenwick recently called attention to the good effects which he had obtained in the treatment of hemorrhage from the stomach by the ingestion of capsules of adrenal extract. It is interesting to notice that another British physician called attention to the same fact in much the same manner, namely Grünbaum, early in 1900. These observers agree in stating, it is well to remember, that when administered by the mouth this drug may cause violent vomiting, which in its turn will aggravate the hemorrhage; the new method overcomes this. The instance is reported of a young girl with an intense hemorrhage from the stomach, who, after having taken several doses of a number of tablets of the pulverized extract of the adrenal capsules, continued to vomit more or less fresh blood. In the presence of this intolerance of the extract by the mucous membrane of the stomach he substituted the rectum as the avenue for giving the drug. After having washed out the bowel with twenty grains of calcium chloride he injected into the rectum, at first every two hours, then every four hours, ten grains of the pulverized adrenal extract suspended in water. The hæmatemesis ceased completely and promptly. This fact should be remembered, because it seems to prove that this extract acts as hæmostatic, not in bringing about coagulation of the blood, but in causing contraction of the vessels which are bleeding. The power of the mucous membrane of the rectum to absorb the drug was certainly increased to a considerable degree by the large quantity of blood which the patient had lost, and similarly the action of the drug was increased by the same cause. The author also uses this drug during the course of surgical operations on tampons impregnated with adrenal extract, with the purpose of clearing the operative field of blood, and thus of making it accessible to view. The drug may also be employed to increase and prolong the effect of anæsthesia, with the aid of ethyl chloride, in the operations of minor surgery and in removal of teeth. For this purpose it is necessary to apply a small dressing soaked in the extract upon the region where ethyl chloride is to be applied.—*La Semaine Médicale*, 1902, No. 4, p. 60.

Action of Tetramethylammonium Chloride on Circulation.—E. FORMAN states that fairly large doses of tetramethylammonium chloride injected into dogs first increase the blood pressure, then lower it, and finally again increase it. The pulse at the same time is retarded. The same changes were noted on division of both vagosympathetic nerves. Preliminary injections of atropine render the retardation of the pulse insignificant, and the high pulse waves disappear, and it may even happen that there will be an acceleration with low pulse waves. It may therefore be said that the retardation depends upon irritation of the peripheral vagus, and that the drug has an action analogous to muscarine. Destruction of the medulla and the spinal cord did not affect the changes in pressure, which appeared to be due to irritation of the peripheral vasoconstrictor nerves, especially in the splanchnic area. The subsequent fall of pressure is due to direct weakening of the heart or its ganglia. A comparison of the tetra- with the mono-, di-, and trimethylammonium chloride shows that with increased methylation the blood pressure is increased and the direct heart action diminished.—*Archives Internationales de Pharmacodynamie et de Therapie*, 1902, vol. ix., fasc. 4 and 6, p. 483.

Treatment of Cardiac Dilatation.—DR. I. BURNEY YEO describes the modern causes of cardiac dilatation, ascribing to athletics a very important rôle. Within general limits, of late years three manifest causes have been at work in giving rise to the prevailing tendency to cardiac dilatation; these are, he says, the influenza epidemic, the abuse of muscular exercise, and the excessive use of tobacco. With reference to the prophylaxis of this affection following influenza, prolonged periods of physical rest are very valuable. In those cases in which overexertion has led to cardiac strain, the avoidance of all kinds of muscular effort must be insisted upon; gentle exercise alone should be permitted. When the condition has been induced by overexcitement, emotional or mental, or by the addiction to unwise habits, these causes must be sought out and corrected. An open air life in the country or at the seaside, gentle exercise, and a nourishing but light diet are remedial measures of importance. Some cardiac tonic may be advisable, and the following prescriptions are offered by the author: Iron and ammonium citrate, 2; tincture of digitalis, 1; aromatic spirit of ammonia, 8; infusion of columbo, 24. Of this two tablespoonfuls twice a day, an hour after meals, are to be taken. He prefers, however, in the less serious forms, to employ strophanthus, or strychnine, or nux vomica, with coca, in combination with iron, quinine, or arsenic, as may seem desirable. The following has been serviceable: Quinine sulphate, 1; tincture of nux vomica, 4; fluid extract of coca, 4; spirit of chloroform, 5; water to 82. Of this two tablespoonfuls twice a day, an hour before food, are to be taken. In purely anæmic cases iron and nux vomica, together with some aperient to insure a regular action of the bowels, will be most appropriate, as: Dried iron sulphate, $1\frac{1}{2}$; soap, $\frac{1}{2}$; powdered nux vomica, 1; aloin, $\frac{1}{2}$ gr. as a pill. One or two (as necessary) twice a day, after lunch and dinner. In cases of somewhat acute dilatation, however induced, the hypodermic injection of strychnine in doses of $\frac{1}{16}$ to $\frac{1}{8}$ of a grain will often be attended with remarkably good results. In extreme cases of cardiac dilatation the ventricular muscle will often be found to be in a state of advanced degeneration, and digitalis

and other cardiac tonics will usually fail in producing any restorative or strengthening effect on it. Our chief resource in such a case must be the most absolute repose, with careful attention to the general nutrition. Light, easily digested, or predigested, highly nutritious food must be prescribed—such as pounded meat very little cooked, beaten-up eggs, chicken and game, a little whitefish when agreeable, milk, and a small quantity of good, sound wine, or a little weak brandy or whiskey and water, but the total amount of fluid taken must be strictly limited, so as not to augment the volume of blood. A regular action of the bowels must be maintained by suitable aperients. Free action of the bowels is very advantageous in nearly all cases of cardiac dilatation and feebleness. Aperients should, however, be so given as to clear away only the residue of digestion. For this purpose the best method is to give an aloetic pill after dinner or at bedtime and a saline dose early in the morning, about an hour before breakfast. Careful attention must, of course, be given to the individual sensitiveness to aperient medicines.—*Practitioner*, 1902, vol. lxviii. p. 281.

Dilatation of the Heart in Children.—DR. EUSTACE SMITH reports that cardiac dilatation, to a moderate extent, is far from uncommon in early life; indeed, in childhood the heart may be said to dilate with especial ease. The dilatation may be found out apart from any valvular affection; it is due to blood pressure in a flabby, ill-nourished or degenerated heart, and may occur without there being any resistance to the passage of blood from the heart. Acute infections, such as bronchopneumonia, diphtheria, and acute rheumatism are particularly liable to cause this. In any such case of acute disease where the dilatation is rapid, a recumbent position should be enforced, and on no pretence should the patient be allowed to lift even his head from the pillow. The diet should be regulated so that those foods that tend to ferment and fill the stomach with wind should be forbidden; baked apples, grapes, oranges, and acid fruits are to be avoided. The patient should be fed with milk, custards, strong soups, yolk of egg, and rusk. In the matter of drugs, if the case be a rheumatic one, and sodium salicylate being taken, it is well to combine with it five to ten grains of iron ammonio-citrate. Strychnine is of great value, and iron perchloride with solution of strychnine, given in full doses well diluted with aerated water, is held by the author to be very valuable. Alcohol should not be forgotten, and the author thinks well of the brandy-and-egg mixture of the *British Pharmacopœia*.—*Practitioner*, 1902, vol. lxviii. p. 56.

The Treatment of Gout.—DR. A. P. LUFF says that quite apart from the treatment of an attack of gout, which is a comparatively easy and simple affair, must be considered the treatment of the conditions which lead up to the attacks. Unstable metabolism is the keynote of this instability of the digestive, the nervous, and the circulatory systems, and the main point in treatment is to determine which set of organs is mainly at fault, and devote the major portion of the treatment to the remedying of such disturbance. He speaks specifically of several lines of treatment. Of the alkalies, the potassium salts of citric and carbonic acids are mostly employed. When combined with colchicum, excellent results seem to be assured. He prefers the combi-

nation of potassium citrate and colchicum. Sodium salts are not contraindicated, but are of less service unless they are used as cathartics. Lithium salts are even less useful than potassium or sodium. He maintains that lithium salts are more toxic than the others, and that the continued use of so-called lithia tablets is specious and wrong. If the gouty attack is combined with constipation and other indubitable signs of hepatic torpor, there is no better treatment at the outset than a dose of calomel or blue pill at night, with Epsom or Carlsbad salts in the morning. Subsequently a pill containing a small dose of calomel combined with euonymin and colocynth will be found most useful. A combination of sodium bicarbonate, gentian, and nux vomica taken a quarter of an hour before meals has proved very useful as a digestant in such cases. Baths, massage, galvanism, superheated, radiant heat are all advisable adjuncts to the drug treatment, but care must be exercised in the selection of the patient and the selection of the procedure as well. In the treatment of gouty eczema attention should be paid to two things: The bowels should be freely opened by blue pill or calomel and followed by a saline; and total abstention from alcohol in any form, red wines especially. As a local application lead subacetate, liquor carbonis detergens, one drachm of each to a pint of aqua sambuci, to be followed by a simple dusting-powder. Carbolic-acid lotions may be advisable to relieve pruritus. Sulphur baths are at times necessary. Dyspepsia should be avoided. Insomnia, which is a usual symptom, needs the free use of calomel. Moderate indulgence in tobacco is not contraindicated. From the side of prophylaxis of the attack, Luff believes in guaiacum resin, basing his hypothesis on the hepatic stimulating properties of this drug. He prescribes the powdered resin in cachets, five grains thrice after meals and gradually increasing the dose to ten or twelve grains. From a somewhat limited experience in the use of quinic acid, he is inclined to believe in its efficiency. The dietary of a gouty patient cannot be laid down in hard-and-fast lines; it is largely an individual question. Simplicity is desirable, and the consumption of large amounts of water are advised both at meals and between periods of eating. As far as climate is concerned, a fairly bracing air, with low, relative humidity is most suitable. High mountain air and low, wet valleys are not advisable resting places. Exposure to east and northeast winds is to be avoided, and residences by the sea are not to be chosen.—*Practitioner*, 1902, vol. lxviii. p. 297.

Blood and Urine in Syphilis.—DR. E. BUFFA, in his conclusions on the condition of the urine and blood in syphilis, says that (1) the urine of syphilitics, far from being hyperacid, is, on the contrary, hypoacid, and that this hypoacidity is but little diminished by a stay in the hospital; (2) the acidity of the blood also suffers a considerable diminution; (3) the mercurial cure, acting upon the body in general, and probably the liver in particular, increases the hypoacidity of the urine, and consequently also the blood; (4) finally, the mercurial cure, specifically for the syphilitic manifestations, is insufficient to bring on a complete cure, and it is necessary to combine with it a treatment acting upon nutrition in general and upon the nervous and hepatic functions in special—*Archives Internationales de Pharmacodynamie et de Therapie*, 1902, vol. ix., fasc. 5 and 6, p. 495.

GYNECOLOGY.

UNDER THE CHARGE OF

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Dysmenorrhœa.—THEILHABER (*Centralblatt für Gynäkologie*, 1902, No. 3) calls attention to the fact that a thorough revision of our views on this subject has become necessary in the light of recent experience. In his opinion more than 75 per cent. of the cases of painful menstruation are not dependent upon anatomical causes. The pain is really due to tetanic contraction of the circular muscle at the os internum, such as occurs in other sphincter muscles in neurotic subjects. He opposes Menge's theory that dysmenorrhœa is due simply to an exaggeration of the contractions of the longitudinal muscular fibres, which always accompany normal menstruation. If, he argues, the symptom is due purely to mechanical obstruction it should invariably disappear after childbirth, which is not the case in nervous and hysterical women. Uterine colic cannot be due only to the passage of clots, since in many typical cases of dysmenorrhœa there is a free escape of fluid blood. Moreover, the pains are often most severe from twelve to twenty-four hours before the flow appears, instead of on the second or third day, when it is most profuse and clots usually appear.

Apparent Abdominal Tumors.—EINHORN (*Berliner klin. Wochenschrift*, 1901, No. 48) found enlargements of the abdomen in 42 out of 6045 patients examined by him, which were not due to neoplasms. These were situated in the epigastric or hypochondriac regions, and are included by the writer under four classes—viz.: Prolapse of the left lobe of the liver, thickening and prominence of the abdominal aorta, localized hypertrophy of the muscles of the parietes, and probable adhesions around the lesser curvature of the stomach.

These are distinguished from true neoplasms by their smooth surface and more sharply defined outline. The site of the enlargement gives some clew as to its possible character. There is normally marked enteroptosis in the conditions before mentioned, while the history of each case shows that it is of long standing.

Uterine Castration.—PINCUS (*Centralblatt für Gynäkologie*, 1902, No. 8) under this term refers to the abolition of the functions of the uterus by means of atmocautis. He reports cases of women with phthisis and Bright's disease in whom obliteration of the uterine cavity by the introduction of steam not only eliminated the metrorrhagia, which threatened to shorten the life of the patient, but prevented future conception—a most undesirable event

under the circumstances. The writer concludes that the operation is only justifiable in the case of women with incurable diseases.

Radical Operations for Cancer of the Uterus.—WERTHEIM (*Centralblatt für Gynäkologie*, 1902, No. 8) reports 77 cases of abdominal extirpation of the cancerous uterus, glands, and broad ligaments. Of the last 27 operations only one terminated fatally—from pulmonary embolus on the fifth day—which fact would seem to show that the immediate results are not so bad as they have been represented. By clamping the vagina below the cervix previous to removing the uterus sepsis can be certainly avoided.

Necrosis of the wall of the ureter occurred only once in the last 17 cases, due to greater care in preserving the vascular supply of the duct. Cystitis is a common complication after the operation. The writer is not yet willing to give his conclusions with regard to the ultimate value of the radical operation, although his oldest cases have been under observation for three years.

With regard to the indications for removing the glands, the writer states that he does not disturb them unless they are distinctly enlarged, since careful microscopical studies have shown that cancerous foci are not present otherwise. He admits, however, that experience may prove that this rule is not always a safe one.

Treatment of Inoperable Cancer of the Uterus.—TORGGER (*Münchener med. Wochenschrift*, 1901, No. 30) has abandoned the use of dry powders, preferring peroxide of hydrogen. He reports 260 cases of inoperable cancer treated by tamponade of the vagina with iodoform gauze saturated in pure peroxide, which is left *in situ* for three or four days. After removal of the gauze as much of the diseased tissue as possible is removed with the sharp spoon, the raw surface is cauterized, and is then covered for a few minutes with cotton soaked in 40 per cent. solution of formaldehyde. Within from six to ten days a slough is thrown off, leaving a dry wound.

Vaginal Puncture and Incision.—FRANZ (*Münchener med. Wochenschrift*, 1901, No. 31) reports 81 cases of diagnostic puncture through the posterior vaginal fornix. This is especially valuable in cases in which it is impossible to make a positive diagnosis between retro-uterine hæmatocele and abscess of the tube or ovary.

The writer seems to be opposed to explorative vaginal incision, especially in cases of hæmatocele. He lays stress upon the importance of using a large, long needle and observing strict asepsis.

Antero-lateral Colpotomy.—DÜHRSEN (*Berliner klin. Wochenschrift*, 1901, No. 44) describes under this heading a new method of colpo-cœliotomy, the advantages of which are as follows: 1. The broad ligaments being first divided, before the peritoneal cavity is opened, it is possible to evacuate abscesses extraperitoneally. 2. The subsequent extirpation of the adnexa is easier. 3. The uterus can be drawn down to the vulva, rendering the tubes and ovaries more accessible. 4. Infected stumps can be brought down and sutured to the side of the vagina. 5. Drainage is more perfect than when the gauze is introduced through a posterior incision. 6. The antero-

lateral method permits more perfect hæmostasis, and thus renders total extirpation unnecessary. Partial incision of the broad ligament is recommended in the case of a large ovarian or tubal abscess situated laterally. Complete separation of the ligament is preferable in the presence of extensive exudates with fixation of the uterus, with infected stumps, and when thorough drainage is indicated. Bleeding from the posterior surface of the uterus after the separation of adhesions, or from deep tears, is best reached and controlled in this way.

Prolapse of the Urethra.—GLÆVECKE (*Münchener med. Wochenschrift*, 1901, No. 22) states that only 150 cases have been reported, to which he adds another. The principal cause of this condition is relaxation of the tissues in consequence of the poor general condition of the patient, senile involution, or frequent child-bearing. Circular incision of the prolapsed portion and suture of the wound is the only proper method of treatment. [We do not understand why the writer should regard this condition as so rare, having operated upon two well-marked cases in old women within the past fortnight.—ED.]

Parametritis Posterior.—MUELLER (*Centralblatt für Gynäkologie*, 1902, No. 9) concludes an article on this subject with the statement that the condition usually described as retroposition of the uterus, or pathological antelexion, resulting from inflammatory shortening of the sacro-uterine ligaments, is really due to inflammation of the rectum at the point where it is surrounded by these ligaments. He believes that the cases are rare in which parametritis posterior is secondary to uterine infection.

The treatment, therefore, should be addressed to the intestinal trouble, being practically the same as that of colitis. He uses injections of tannin, oak-bark, permanganate of potash, protargol, nitrate of silver, etc., with careful regulation of diet and laxatives. Vaginal massage is recommended to stretch the shortened ligaments, or rather perirectal exudates. The best results are obtained by making simultaneous pressure through the rectum by means of a thick sound or bougie. Electricity is also recommended.

Traumatic Granuloma of the Bladder.—KOLISCHER (*Centralblatt für Gynäkologie*, 1902, No. 10) reports four cases that were clearly due to prolonged trauma in a bladder which was the seat of inflammation. The symptoms were tenesmus and a feeling of fulness in the bladder. Hemorrhages were frequent and copious, and the urine was loaded with phosphates. Through the cystoscope one or more bright-red sessile growths were seen, always at the site of a cicatrix. These were quite vascular, and bled at the slightest touch, although they were not sensitive. The usual applications employed in cystitis do no affect granulomata, which must be excised, preferably through the cystoscope. Bleeding is usually sharp, but may be controlled by suprarenal extract. Recurrence does not occur after complete removal.

OBSTETRICS.

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Attempted Criminal Abortion in Ectopic Gestation.—In the *Boston Medical and Surgical Journal*, January 9, 1902, SWAN reports the case of a patient about seven weeks pregnant, who had instruments introduced within the womb to produce abortion. On the ninth day afterward she was attended by two physicians who examined and curetted the uterus. The patient died in the afternoon of the following day. Upon autopsy the peritoneal cavity contained three pints of dark fluid and clotted blood. In the right Fallopian tube was a ruptured swelling, from which protruded clotted blood and fine filaments. The right ovary contained a corpus luteum. The patient evidently perished from ruptured ectopic gestation.

The direct evidence of criminal abortion had been removed by dilatation and curetting; accordingly, the only assignable cause for death was ruptured ectopic gestation, and the abortionist escaped prosecution.

[This case is an illustration of the many circumstances which protect the criminal abortionist. A physician's first impulse when summoned to a case of pelvic septic infection following criminal abortion is to cleanse the uterus by curetting. By so doing, however, he destroys the evidence of the abortion, and the abortionist usually escapes prosecution. It is better in such cases, before interfering in any manner, to notify the authorities and to give them an opportunity to secure evidence. Then dilatation and curetting, if necessary, may be performed. A further reason for delay lies in the fact that many of these patients, after criminal abortion, suffer from exhaustion, through hemorrhage and infection. They are ill-prepared to endure an anæsthetic and an operation. A better result is usually obtained if sufficient delay is practised to build them up by stimulation.]

The Treatment of Placenta Prævia.—In the *Boston Medical and Surgical Journal*, January 2, 1902, HIGGINS reported the results of seventy-five cases of placenta prævia, of which fifty-six were treated in hospital wards and nineteen in out-patient service in their homes. Among the house cases there were six deaths, a mortality of 10.7 per cent. Of these six, five were complete placenta prævia. Of the nineteen out-patients there were two deaths, one of the complete and one of the incomplete variety of placenta prævia, a mortality of 10.5 per cent. Of the seventy-five cases there were eight deaths, a mortality of 10.6 per cent. These cases were treated by the use of the tampon and occasionally by the employment of forceps. Sixty-two per cent. of the children were born prematurely.

The method of treatment described and most usually employed consisted in the induction of labor so soon as the diagnosis is made by rupturing the membranes and packing the vagina tightly with dry sterile gauze. Under the influence of the gauze-packing hemorrhage is controlled and dilatation proceeds and, if necessary, the patient can be finally delivered by forceps or version.

The purpose of this paper is to criticise the performance of Cæsarean section for placenta prævia. The writer quotes the results of thirty-two Cæsarean operations in the Boston Lying-in Hospital during the last five years, with three maternal and three fetal deaths, a mortality of 9.3 per cent. He believes that the general mortality of the Cæsarean operation is about 10 per cent. He gives three quotations from American writers, but makes no reference to the foreign literature of the subject. He draws attention to three cases of placenta prævia recently operated upon in the vicinity of Boston, with a mortality of 66.6 per cent.

In discussion Worcester would not condemn Cæsarean section for placenta prævia. He drew attention to the fact that aseptic precautions are more easily exercised in abdominal section than in prolonged labor with repeated vaginal packing. In placenta prævia the cervix is easily lacerated, and while he was satisfied with the ordinary methods of treatment in cases where the placenta is not central, in central placenta prævia he believed that there existed a field for the modern Cæsarean operation.

Donoghue has performed Cæsarean section twice for placenta prævia, with the recovery of one mother and two children. He believed that when the placenta is central Cæsarean section is indicated. It is also indicated in incomplete placenta prævia when malpositions are present and in cases where the mother has been delivered previously by operations. Others spoke in favor of Cæsarean section in cases where the os and cervix are tightly closed, the placenta central, the patient in good condition and in the hands of an operator competent to perform abdominal section.

During the discussion of this paper attention was called to the fact that obstetric cases are usually in the hands of those incapable of performing abdominal section. While this may be true in many cases, it is not in all, and is a condition which should not be so. No one is competent to attend obstetric cases who cannot deal with ruptured ectopic gestation by abdominal section or labor in contracted pelvis, or who cannot operate in any manner to save the life of mother and child.

A Complicated and Fatal Case of Early Pregnancy.—In the *Centralblatt für Gynäkologie*, No. 1, 1902, HAEERLIN reports the case of a patient, aged thirty-nine years, who had borne seven children and had been for the last year out of health. She complained of pain in the lower portion of the abdomen and marked distress in the stomach. She had failure of appetite, and the filling of the stomach caused intense distress. When seen she was eight weeks advanced in pregnancy and was growing much worse. She could not sleep, was ejecting all food and in a highly melancholy frame of mind. She was very anæmic, the skin dry and yellow, the tongue also dry. The action of the heart was regular, the pulse 80, small and with little tension, and the uterus was of the size commonly seen at eight weeks' gestation.

Because of the patient's threatening general condition it was determined to empty the uterus, and at the request of the patient and her husband to make further pregnancy impossible. Accordingly, the uterus was curetted, cleansed and packed with iodoform gauze. Abdominal section was performed and the Fallopian tubes were excised. There was some oozing of blood when the left tube was removed. In general the operation proceeded well and the patient roused quickly from the anæsthetic. Iodoform gauze was removed from the uterus on the following morning. The patient's pulse became very rapid and irregular. She had great apprehension, and complained of strong and tumultuous beating of the heart. The bowels acted after injections, the wound in the abdomen healed by first intention, and the patient gradually grew better. Five days after the patient had become convalescent she had a similar attack of rapid heart action and general distress, from which she gradually recovered.

The author suggests no definite explanation for this case. The character of the pulse resembled that of iodoform poisoning. Ileus might produce such symptoms also.

Cæsarean Section by Incision of the Fundus.—In the *Centralblatt für Gynäkologie*, 1902, No. 5, JUROWSKI reports the case of a patient, aged thirty-nine years, who lost her first child after tedious labor, and in whom the second and third labors were terminated by craniotomy. In her fourth labor the amniotic liquid was early discharged, and the patient brought to the hospital in labor. Upon examination the pelvis was considerably contracted, the internal conjugate being $7\frac{1}{2}$ cm. The head was presenting, but not engaged. Cæsarean section was chosen and accepted by the patient, and the uterus opened by transverse incision across the fundus. The child was extracted by the feet, and the placenta easily removed. The bleeding was very slight, and ceased completely upon removal of the placenta. This was situated on the anterior wall of the uterus, its margin coming to the edge of the fundal incision. The patient made an uninterrupted recovery. Upon examination it was found that the uterus was adherent to the abdominal wall at the lower end of the abdominal incision.

The second case was a patient who had had hip-joint disease in childhood. She had been four times pregnant, and in the first labor had craniotomy, and in the second and third the child was lost in version and extraction. The external conjugate was 17 cm. The internal conjugate could not be measured because of the high position of the promontory of the sacrum. The head was high above the pelvic brim and the membranes had not yet ruptured. Cæsarean section by transverse incision was chosen, and at the operation the placenta presented and was rapidly torn through and delivered to avoid hemorrhage. The child was delivered by the feet, and the uterus closed in the usual manner. An excellent recovery followed.

The third case was a patient who had lost three children, one in spontaneous birth and two with the use of forceps. She had a contracted pelvis, with an external conjugate of $17\frac{1}{2}$ cm. The head had not engaged, and the membranes were unruptured. The operation proceeded smoothly, and the patient made an uninterrupted recovery.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Report of the Men Working under the Cancer Commission of Harvard University for the Year 1901-1902.—(*Journal of Medical Research*, 1902). It is claimed by the men who believe that cancer is due to parasites: 1. That a proliferation of the epithelial cells analogous to cancer can be produced by certain protozoa (nodules due to coccidium oviforme). 2. That certain skin lesions characterized by epithelial-cell proliferation are due to the action of a so-called protozoan, molluscum contagiosum. 3. That blastomycetes constantly are present in human cancers, and are the cause of the lesion. 4. That by experimental inoculations of animals with blastomycetes, true epithelial or cancerous nodules can be produced. 5. Finally, that the well-known endocellular bodies seen in the protoplasm of cancer cells have a definite morphology, are parasites, and cause cancer. It was the object of investigators whose work is here reported to study each of these questions.

COCCIDIUM INFECTION OF THE RABBIT'S LIVER. TYZZER describes the lesions produced by coccidium oviforme in the liver of rabbits. He finds that the lesions consist of small white or yellow nodules which always are connected with the bile-ducts and may occur as cyst-like or dense and firm structures. Histologically the nodules are composed of cysts into which project papillæ covered with epithelium, or there may be very little lining epithelium to the cysts, with a dense fibrous capsule and remote cirrhosis of the liver. The infection takes place through the stomach by the ingestion of a capsulated form, the oöcyst, in which develop eight actively moving sporozoites. The capsule is dissolved by the digestive juices, and the sporozoites are set free and invade the biliary epithelium. In the epithelial cell the sporozoite develops into a schizont, which divides and breaks up into falciform bodies, the merozoites, which in turn are set free and invade the biliary epithelium. This is the process of autoinfection.

There is also a process of sexual reproduction. Some of the organisms become microgametocytes, from which small male elements, microgametes, are developed, which fertilize the female form. Other forms may develop into macrogametes, the adult female form, which, becoming fertilized, develops a resistant envelope, the sporocyst; this adult organism set free passes out through the intestinal tract, and may infect other animals if ingested.

The histological changes are as follows: After invasion of the epithelium by the protozoan the epithelial cell degenerates and is destroyed. This produces a defect in the epithelial lining of the duct, and inflammatory exudation appears. Following the destruction of the epithelium there comes an increase in the underlying connective tissue which pushes into the bile-duct to form papillæ, and a proliferation of epithelium occurs in order to cover in the defect. Repair is brought about by walling off of the lesion by connec-

tive tissue. No metastases occur. In only one stage does the parasite in the least resemble the so-called "parasite" of cancer. The lesions produced by *coccidium oviforme* are essentially those of chronic inflammation.

MOLLUSCUM CONTAGIOSUM. WHITE gives a most complete and admirable review of the literature. The histological changes are described with accurate detail. The nodule is due to a hyperplasia of the rete cells, which push down and out and produce a globular tumor. The deeper layers consist of quasi-normal spinous cells with well-marked nucleoli, usually single or double, which stain deeply. Among these are cells with no nucleus and with fibrillar protoplasm. Nearer the surface are cells with distorted nuclei surrounded by a clear halo, or the cells may lose their nuclei altogether. In still more superficial layers the cells increase in size, the nucleus is pushed to one side, and the cytoplasm becomes honey-combed. Mitotic figures are common. In still higher layers the nuclei disappear and the cell looks like a multilocular cyst. Still higher, probably in the stratum granulosum, the cells become smaller, the trabeculae disappear, and the cells become homogeneous with keratin-like walls. There are no bodies which resemble the gregarines, nor any doubly-contoured bodies. Often there is well-marked inflammation about the nodule. The staining reaction shows that the end result of the cell changes is the production of keratin—i. e., the entire process is a peculiar metamorphosis of the rete cells into keratin.

The bacteriological work was done by ROBEX. He used ordinary bacteriological media and a special medium, a bouillon made of finely chopped human skin. Extensive cultural experiments showed the presence of no organism except the *staphylococcus epidermidis albus* of Welch. Inoculation of animals with this organism produced the usual *staphylococcus* inflammation; in others it produced no results. There was no other cultivatable parasite.

CULTURAL EXPERIMENTS OF MALIGNANT TUMORS. RICHARDSON made an extensive series of cultural experiments with fresh cancerous material obtained from operations at the Massachusetts General Hospital. He used a great number of different media. The cultures were observed over periods of time varying from fifteen days to months. The result of his investigations confirms his report of previous work, and he concludes that it is impossible to cultivate from new-growths anything which can be regarded as a specific infective agent.

FOUR PATHOGENIC TORULÆ. WEISS studied the morphology and cultural peculiarities of four blastomycetes (really torulæ), two of which have been isolated from cancer—one by Plimmer and one by Sanfelice. One came from fruit, and was said to produce cancer by experimental inoculation (Sanfelice's *neoformans*). One came from milk, and was pathogenic for rabbits (Klein). Weiss gives a systematic classification of the fungi, and says that the torulæ are micro-organisms similar to the *saccharomycetes*, except that they do not produce spores or develop mould-like vegetations (mycelia). As a rule, they have no power to produce fermentation, although a few can. Morphologically the torulæ cells are round masses of protoplasm, each contained in an envelope. It is doubtful if they have a nucleus. The protoplasm consists of a fine network of vacuoles and oil-drops. Sometimes in the vacuoles is a nucleus with Brownian movement. The membrane con-

sists of two layers, which sometimes can produce a gelatinous substance outside of the capsule, the nature of which is not evident. Weiss used special technical methods, as taught by the Copenhagen school. He found that the four organisms he examined did not ferment sugar, or form spores, or produce mycelia. He found after a certain length of a life of cultivation that each organism had constant morphological and cultural peculiarities which allow them to be differentiated. He considers all four organisms studied to be torulæ, and gives an extensive table of their characteristics.

CELL INCLUSIONS IN CANCER AND IN NON-CANCEROUS TISSUE. GREENOUGH examined various tissues to see if bodies similar to the well-known cancer bodies exist in tissues other than cancer. He took as his standard of cell inclusion the form described by Plimmer in the *Practitioner* of April, 1899. He examined a large number of different tissues with entirely negative results. He examined also thirty non-malignant tumors of the breast, including acute and subacute mastitis, adenoma, adenofibroma, and myxofibroma. In eleven cases of chronic mastitis, which were non-malignant—that is, breasts which had round-celled infiltration with an increase of connective tissue, atrophy of the gland tubules, and cyst formation—there was no penetration of the epithelium beneath the basement membrane, and no marked increase of mitotic figures; in each of these cases he found typical inclusions like those seen in cancer cells. The nucleus of the cell in which these inclusions occurred was always near the basement membrane, while the inclusions were on the side nearest the lumen. Exactly similar conditions were found in eight adenofibromata of the breast, in one myxofibroma, in three out of four adenomata, and in eight cancers. In the cancers the inclusions, as a rule, were larger than in the other tumors of the breast. Greenough believes that since the inclusions are seen entirely in alveolar glandular carcinomata and in no epitheliomata they have to do with the process of secretion, and that they do not arise from nuclear degeneration.

From a review of the literature it is evident that in secretion in glandular epithelium there is associated a definite differentiation of the cell protoplasm, with which a protoplasmic body, the centrosome, is involved. Moreover, the great variation in size of the inclusions in cancer suggests secretion and accumulation, which may attain great size if the gland duct is occluded, as happens in cancers where the epithelial cells are massed together and no lumen is left. The inclusions occur more frequently in slowly than in rapidly growing cancers. The cells have probably lost their power of secretion with greatly increased capacity for growth. Inclusions in mitosing cells are very uncommon. He concludes (1) that cell inclusions of a constant type occur in practically all cases of cancer of the mammary gland; (2) that they also occur in non-cancerous disease of the breast, but (3) not in epitheliomata or sarcomata; (4) that apparently the staining reaction and situation of these bodies justify the hypothesis that they are the results of secretory activity; (5) there is no reason for believing that they are of parasitic origin.

THE RELATION OF BLASTOMYCETES (TORULÆ) TO CANCER. NICHOLS reviews the literature at some length. The idea that cancer is due to a blastomyces practically dates from a case reported by Busse, clinically considered to be a "soft sarcoma" or "chronic inflammation." Blastomycetes were present in the primary tumor and in internal metastases. They could

be cultivated in pure culture, and in animals produced local and secondary granulomata.

Sanfelice considered that the well-known cancer bodies were identical morphologically with blastomycetes. He cultivated in fruit juices a blastomycetes which he obtained "from the air" the "*saccharomyces neoformans*," inoculated many animals, and produced local "tumors" with large lymph nodes and internal metastases. In but three cases did he claim to produce cancer, and in these cases there are no epithelial metastases, nor could he obtain his organism from the nodules.

Plimmer obtained a similar organism which he considered to be the cause of cancer, but by inoculation produced only "endotheliomata," not "cancer." Other men claimed that morphologically the cancer bodies were analogous to blastomycetes, but the evidence of this identity was very weak.

Many men inoculated animals with blastomycetes from animal or human tumors. They produced local and secondary lesions of inflammatory or granulation tissue, but never cancer, except Leopold, whose experiments were puerile. In human beings lesions, clinically sarcomata, but histologically granulomata, due to the action of blastomycetes, have been observed. A few cases of dermatitis, due to blastomycetes, have also been seen in human beings, but the lesion is essentially an intraepithelial inflammation, and is not analogous to cancer. In horses a disease clinically like glanders, due to the action of blastomycetes, has been observed.

Many men working with blastomycetes obtained in various ways have found many inert and some pathogenic; the pathogenic varieties produced only abscesses or nodules of granulation tissue. Some men working with Sanfelice's *neoformans* also produced granulation tissue in animals, but never cancer.

Moreover, yeasts are seldom found in cancers, and when present are found in ulcerating nodules and represent a secondary infection. The cancer bodies are not identical morphologically with blastomycetes; they are found in other lesions than cancer; they are not constantly present in cancers; when they are present they are not so situated, and do not occur in such numbers that they can be considered as the cause of the disease. The mere presence of blastomycetes in cancer, even if constant, is no proof that they are the cause of the disease.

Nichols inoculated thirty animals—eighteen with Sanfelice's *neoformans*, twelve with Plimmer's organism. There was always a local reaction, fever and abscess, or nodules of peculiar granulation tissue composed of connective tissue, many endothelioid cells, and relatively few bloodvessels. The blastomycetes were free or in phagocytic cells, and were always surrounded by a gelatinous capsule. Usually there was little acute inflammatory exudate, but there always were some plasma, lymphoid, and eosinophilic cells. In glandular organs the reaction was confined entirely to the interglandular connective tissue. There was no glandular proliferation. In the epidermis the epithelial cells were destroyed by contact with the blastomycetes. There never was proliferation. The lymph nodes always were enlarged, and were myxomatous or necrotic. Blastomycetes were free in the sinuses or were contained in the endothelial cells. Ultimately they destroyed the lymphoid tissue, which was replaced by a proliferation of connective tissue and endothelioid cells. In

the lungs the blastomycetes were in the alveolar wall, and caused a proliferation of connective tissue, which encroached upon and finally obliterated the alveoli. Often there were pneumonia and atelectasis. The liver practically always was free. In the spleen the follicles were primarily affected and were replaced by connective tissue and endothelioid cells. In the kidney the lesions were always in the cortex, beginning usually in the glomeruli, were composed of connective tissue and endothelioid cells, and destroyed adjacent tubules by pressure. The morphology of the blastomycetes was constant and not the same as that of cancer bodies.

Nichols' conclusions are the following: 1. Certain blastomycetes are pathogenic. 2. They produce in animals spontaneously infected acute inflammations, abscesses, or nodules of granulation tissue. 3. In human beings spontaneously infected they produce acute inflammation or proliferation of endothelium and connective tissue. The occasional proliferation of the epidermis is secondary to the chronic inflammation of the corium, and is not analogous to cancer. 4. Blastomycosis in human tissue is rare. 5. The lesions produced in animals experimentally are inflammation or nodules of granulation tissue. Sanfelice's "successful" cases are coincidents and not results. 6. The toxic power of blastomycetes is small. 7. Blastomycetes primarily extend along lymphatic clefts. 8. Rarely there may be a blood dissemination and general infection. 9. Secondary nodules are the same as the primary—i. e., composed of granulation tissue. 10. The morphology of the cancer body is not identical with the blastomycetes. 11. Blastomycetes are not constantly present in human cancers. 12. When they do occur they are not present in such numbers and in such a relation to the lesions as to justify the belief that they are the cause.

All of which leads to the ultimate conclusion that blastomycetes have nothing to do with the production of cancer.

GENERAL CONCLUSIONS. As a result of the work just reported it is concluded that: 1. The lesion produced by *coccidium oviforme* is essentially a chronic inflammation not analogous to cancer. 2. The lesions in molluscum contagiosum show certain changes in the epidermis not due to the action of protozoa and not analogous to cancer. 3. The so-called "blastomycetes" of Sanfelice and Plimmer are torulæ. 4. The lesions produced by these torulæ are essentially nodules of peculiar granulation tissue, not cancerous or in any sense "tumors." 5. Blastomycetes are not constantly present in human cancers. 6. The cancer bodies are not parasites or the cause of the lesions, but are probably atypical stages of the process of secretion by glandular epithelium.

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With the same formula prescribe 1 tablespoonful of Mellin's Food instead of 2, and the analysis then is

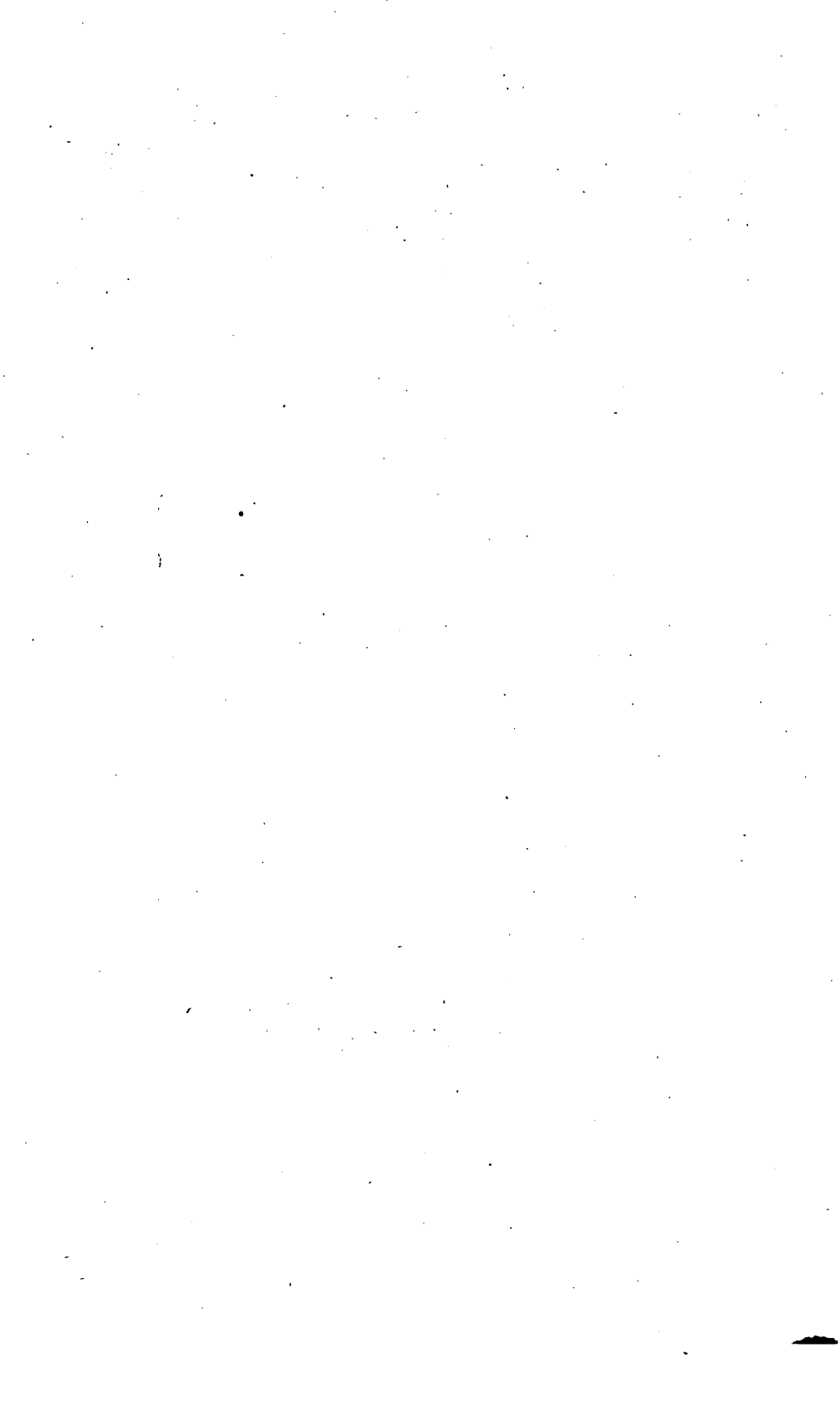
| Fat  | Proteids | Carbohydrates | Salts | Water |
|------|----------|---------------|-------|-------|
| 3.16 | 2.85     | 5.44          | .71   | 87.84 |

Many other formulæ and analyses are given in our Formula Booklet. It is free to physicians. We should like to send you one. You know the Food.

MELLIN'S FOOD COMPANY, BOSTON, MASSACHUSETTS.









R.P.L. Bindery  
SEP 27 1902